Jacalyn J. Robert-McComb
Reid L. Norman
Mimi Zumwalt
*Editors*

# The Active Female

## Health Issues Throughout the Lifespan

Second Edition

Springer

# The Active Female

Jacalyn J. Robert-McComb
Reid L. Norman • Mimi Zumwalt
Editors

# The Active Female

## Health Issues Throughout the Lifespan

Second Edition

Springer

*Editors*
Jacalyn J. Robert-McComb, Ph.D., F.A.C.S.M., A.C.S.M., P.D.
Professor of Exercise Physiology
Department of Health, Exercise and Sport Sciences
Texas Tech University Health Sciences Center School of Medicine
Lubbock, TX, USA

Mimi Zumwalt, M.D.
Associate Professor
Head of Sports Medicine
Department of Orthopaedic Surgery and Rehabilitation
Texas Tech University Health Sciences Center School of Medicine
Lubbock, TX, USA

Reid L. Norman, Ph.D.
Professor and Chair
Pharmacology and Neuroscience
Texas Tech University Health Sciences Center School of Medicine
Lubbock, TX, USA

ISBN 978-1-4614-8883-5 ISBN 978-1-4614-8884-2 (eBook)
DOI 10.1007/978-1-4614-8884-2
Springer New York Heidelberg Dordrecht London

Library of Congress Control Number: 2013953599



Printed on acid-free paper

Springer is part of Springer Science+Business Media (www.springer.com)

*I would like to dedicate this book to all the significant females in my life: my grandmother who helped to raise me but now has passed, my mother who fought to bring me here from Vietnam, and especially my daughter who provides me with more joy than words can express. I dearly love and appreciate them all!*

Mimi Zumwalt, M.D.

*We would like to dedicate this book to female athletes and active girls and women of all ages.*

Jacalyn J. Robert-McComb
Reid L. Norman

# Preface

Medical practitioners and health care providers/educators must continue to be vigilant of the growing and ever-changing health issues relating to females who lead an active lifestyle by participating in sports and exercise. There have been recent landmark legislations changing the social perception that girls and women not only can but should be physically active. With any change in the social milieu, there are always evolving health issues associated with the journey. Continuing medical education for physicians, nurses, allied health professionals, wellness educators, and certified professionals in sports medicine is vital to the economic and public health care system. Education has been recognized as the most important tool that we can utilize to prevent illness and disease throughout our lifetime.

In 1972, Congress passed Title IX of the Educational Amendments Act assuring that all females would have the equal opportunity to participate in interscholastic and intercollegiate sports. The effect has been a tremendous increase in the participation of girls and women in interscholastic sports from approximately 300,000 to greater than 2.2 million in the late 1990s [1].

Participation in recreational exercise for health and fitness, from young girls to elderly women, has substantially increased in the last four decades and has become a prominent part of public life more so than ever before [2]. Physical activity has been recognized as a therapeutic means to decrease illness and increase health/well-being for females of all ages and racial groups. In the US Public Health Services release "Healthy People 2000," one of the recommendations was to increase the physical fitness of all women in an effort to reduce the health disparities between males and females, and also among different ethnic and racial groups [3].

What makes female health issues unique? Girls and women are different from boys and men, not only physically and physiologically but also psychologically. Body image issues are more prominent in young girls than young boys and body dissatisfaction seems to start very early in life. Collins et al. [4] reported that 42 % of a sample of 6–7-year-old girls indicated a preference for different body figures which are thinner than theirs. Similarly, Thompson et al. [5] found that 49 % of fourth-graders indicated that their ideal body would be thinner than their current figure. Anatomically, young girls' bodies begin changing at puberty. This may become a hindrance to sport performance. Internal and external pressures placed on females to achieve or maintain unrealistically low body weight may affect their normal

life cycle. Monthly menstrual cycles, childbearing and menopause are experiences in the female life cycle that are unique to girls and women. The lack of menses caused by extreme energy deficiency may even seem desirable to young females, yet there are long-term health consequences that initially are not so obvious to the poorly informed.

In 1992, The Female Athlete Triad was the focus of a consensus conference called by the Task Force on Women's Issues of the American College of Sports Medicine [6]. The three components of the triad are: disordered eating, amenorrhea, and osteoporosis. However, these are not disorders confined to elite athletes, nor are they limited to females competing in sports. These disorders are also found in young girls and elderly women who have never participated in collegiate or intercollegiate teams. These disorders represent a growing health concern for females of all ages and physical skill levels.

Almost a decade ago, the US National Institutes of Health (NIH), recognizing the lack of inclusion of women in health research and realizing that many health issues are unique to and/or affect women differently from men, established the Office for Research in Women's Health with the goal to improve women's health status across the lifespan [7]. A couple of years earlier, the Female Athlete Triad Coalition was formed in 2002 as a group of national and international organizations dedicated to address unhealthy eating behaviors, hormonal irregularities, and bone health among female athletes and active women. The Female Athlete Triad Coalition represents key medical, nursing, athletic, health educators, and sports medicine groups, as well as concerned individuals who have come together to promote optimal health and well-being for active women and female athletes (www.femaleathletetriad.org).

The importance of physical activity to health and well-being continues to grow nationwide. The *Physical Activity Guidelines for Americans* released in 2008 is the first-ever publication of national guidelines for physical activity [8]. The position stand on the Female Athlete Triad was updated in 1997 [9], and now it reflects our current understanding of the issues unique to girls and women: the interrelationships among energy availability, menstrual function, and bone mineral density. Healthy People 2010 and 2020 [10, 11] reflect the strong state of the science supporting the health benefits of moderate and vigorous physical activities and muscle-strengthening activities for girls and women.

Lubbock, TX, USA

Jacalyn J. Robert-McComb
Reid L. Norman
Mimi Zumwalt

## References

1. Bunker LK. Psycho-physiological contributions of physical activity and sports for girls. Phys Activity Fitness Res Digest. 1998;3:1–8.
2. Garrett W, Lester G, McGowan J, Kirkendall D. Women's health in sports and exercise. Rosemont, IL: American Academy of Orthopaedic Surgeons; 2001.

3. Public Health Service. Healthy People 2000: National Health Promotion and Disease Prevention Objectives. Full Report, With Commentary. Washington, DC: US Department of Health and Human Services; 1990 (DHHS publication (PHS) 91-50212).
4. Collins LR, Lapp W, Helder L, Saltzberg J. Cognitive restraint and impulsive eating: insights from the Three-Factor Eating Questionnaire. Psychol Addict Behav 1992;6:47–53.
5. Thompson SH, Corwin S, Sargent RG. Ideal body size beliefs and weight concerns of fourth-grade children. Int J Eat Disord. 1992;21:279–284.
6. Yeager K, Agostini K, Natttiv A, Drinkwater B. The female athlete triad. Med Sci Sports Exerc. 1993;25:775–777.
7. Klimis-Zacas D, Wolinsky I. Nutritional concerns of women. Boca Raton, FL: CRC Press; 2004.
8. United States Department of Health and Human Services. Physical Activity Guidelines for Americans. 2008. http:// www.health.gov/paguidelines.
9. Nattiv A, Loucks AB, Manore M, Sanborn C, Sundgot-Borgen J, Warren M. The female athlete triad. Med Sci Sports Exerc. 1997;39:1867–1882. doi: 10.1249/mss.0b013e318149f111.
10. United States Department of Health and Human Services. Healthy People 2010. http://www.healthypeople.gov/2010/Document/tableofcontents.htm#under.
11. United States Department of Health and Human Services. Healthy People 2020. http://healthypeople.gov/2020/default.aspx.

# Role and Scope Statement

There are specified educational competencies in the field of medicine, as in every professional allied health discipline, yet not all facets of therapeutic medicine can be covered adequately through a formal educational setting. Physicians, allied health professionals, health educators, and sports medicine practitioners are confronted daily with female health and ever-changing fitness needs. Continuing education in very specific domains is needed to stay current and be an effective health educator and medical practitioner. Therefore, the role of this book is twofold: to increase the awareness of wellness and fitness issues specific to the active female by providing an effective means to disseminate knowledge and to enable the medical practitioner, allied health professional, health educator, and certified individuals in sports medicine an avenue not only to gain knowledge but also to earn the continuing medical education (CME) credits mandated by their respective governing bodies.

The American College of Sports Medicine (ACSM) endorses more than 180 conferences a year that extend Continuing Education Credits (CEC) and Continuing Medical Education (CME) credits to individuals seeking these types of credit hours. ACSM is also accredited by the Accreditation Council for Continuing Medical Education (ACCME) to sponsor continuing medical education for practicing physicians.

The content material in this book and accompanying supplemental teaching materials (multiple choice questions and answers for each chapter) are ideal for 1- or 2-day workshops/conferences focused on women's health issues, or college/university classes/courses. Given that most licensing and certifying organizations offer CEC and CME credits for collegiate classes and endorsed online courses, this textbook is perfect for the development of Web-based educational programs in women's health issues since multiple choice questions and answers accompany each chapter.

In addition to licensed and certified physicians who need continuing medical education credits to maintain their licensure and certification, there are licensed and certified allied health professionals such as physician assistants, nurses, physical therapists, and athletic trainers who, among others, must maintain their status through continuing education credits (CEC's).

Other recognized organizations which provide certifications in the field of sports medicine include: ACSM (www.acsm.org), the American Council on Exercise (www.acefitness.org), the National Strength and Conditioning

Association (www.nsca-lift.org), and the Aerobics and Fitness Association of America (www.afaa.com). Continuing education credits are needed to maintain current certification status of each organization.

Health Educators are also certified by the National Commission for Health Education Credentialing (NCHEC) and must earn CEC's to maintain their certification. NCHEC seeks to promote scientific, ethical, and state-of-the-art programs of professional preparation and continuing education (http://www.nchec.org/NCHEC).

Part I of the book serves as a foundation to help the reader understand the interrelationship between body image concerns, the female reproductive cycle, and the musculoskeletal anatomy/physiology of females that make their health risks and concerns unique to the female gender. An overview of the Female Athlete Triad is presented in Part II, with the authors discussing the individual components of the triad, which relate not only to the female athlete but also to the recreationally active woman throughout the lifespan. In Part III, reproductive health is discussed by a prominent researcher in reproduction/endocrinology. Part IV, written by a female orthopedic surgeon who sub-specializes in treating the female athlete, focuses on the prevention and management of common musculoskeletal injuries. Finally, appropriate exercise and nutritional guidelines for active females are discussed in Parts V and VI of the book by certified professionals in the field of sports medicine.

## Audience

Appropriate audiences for this book are physicians, allied health care practitioners, medical/other wellness educators, and students who are interested in advancing women's health issues in the field of therapeutic medicine. Sports medicine specialists, family practitioners, gynecologists, team physicians, residents doing fellowships in sports medicine, athletic trainers, health educators, nurses, physicians assistants, physical therapists, sport psychologists, counselors, athletic trainers, and other members of the sports medicine team who may be involved in the education and development of the active female throughout the lifespan should find this book of interest. It has a particular appeal for licensed and certified professionals in the field of sports medicine and Certified Health Education Specialists who must earn CEC and CME credits.

This book would be a useful textbook for a women's health issues class in Physical Therapy settings. Papanek discusses the emerging role for physical therapists in treating women who have experienced certain aspects of the female athletic triad: eating disorders, amenorrhea, and osteoporosis (Papanek PE. The female athlete triad: an emerging role for physical therapy. J Orthop Sports PhysTher. 2003;33(10):594–614. Review). Other disciplines that would find this textbook suitable for academic coursework are Athletic Training, Health, Exercise and Sport Sciences, and Women's Studies. Last but not least, invited guest authors include physical therapists, clinical researchers in biomechanics, and gynecologists.

The information in this book is also particularly valuable for the sports medicine health care team and wellness educators at the junior high or high school level, since adolescence is a particularly important time for bone remodeling and bodily changes. Lastly, the educated layman or woman who may be experiencing these gender-specific problems, or have daughters who may be experiencing this triad of disorders, would find this book interesting and informative. This book is by no means all-encompassing in terms of female fitness but provides a foundation upon which more information can be added, and new ideas built upon. It serves as a guide for any reader who is seeking further knowledge of girls and women who choose to stay physically active from birth till death.

# Acknowledgements

I would like to acknowledge the following people for their technical contribution in helping me to complete/edit my book chapters. Without their dedication and hard work, the job would have been much more difficult: Brittany Dowling and Parijat Fnu. I want to also recognize AND commend Ms. Beverly Reed for her patience and tolerance, not to mention the tremendous effort, work, and time she put into the challenging tasks especially near the end. I also want to thank my operating room crew: Adria Renegar, Daryl Anderson, and Genaro Marquez; Al, Berni, Rosa, and Katy–my closest friends; Mich, my brother; Francoise and Kevin, my parents; and most of all, Demi and Miko, my wonderful children, plus my puppies Lala, Stormi, and Molli for all their love and undying moral support for me always!

Mimi Zumwalt, M.D.

We would like to thank the following people for helping us with the task of writing this book. First and foremost, we would like to thank Beverly Reed. She has worked tirelessly in rewriting and reformatting chapter after chapter. We would also like to thank the graduate students at Texas Tech University who have helped with referencing the citations: Karlyn Meyers, Devesh Digwal, Hiten Kothari, Parijat Fnu, and Kembra Albracht.

Jacalyn J. Robert-McComb

# Contents

# Contributors

**Kembra D. Albracht, Bachelor of Science in Exercise Science** Department of Health, Exercise and Sport Science, Texas Tech University, Lubbock, TX, USA

**Natalia E. Bustamante-Ara, Ph.D.** Physical Activity and Sport, Department of Physical Activity and Health, Nengn Company, Maipú, Santiago, Chile

**Elvis Álvarez Carnero, Ph.D.** Biodynamic and Body Composition Laboratory, Sports and Physical Activity Sciences, University of Malaga, Málaga, Málaga, Spain

**Lesley Carraway, M.S.** Department of Health, Exercise, and Sports Sciences, Texas Tech University, Lubbock, TX, USA

**Andrew Cisneros, Bachelor of Science in Exercise Science, Masters of Science in Exercise Physiology, Student Physical Therapy (S.P.T.), Masters of Science (M.S.), Certified Strength and Conditioning Specialist (C.S.C.S.)** Physical Therapy, School of Allied Health Sciences, Texas Tech University Health Sciences Center, Lubbock, TX, USA

**Brittany Dowling, Master of Science** Department of Health, Exercise, and Sports Sciences, Texas Tech University, Colorado Springs, CO, USA

**Ascensión Blanco Fernández, Ph.D. Physiology** Especialidades Médicas, Psicología y Pedagogía, Universidad Europea de Madrid (UEM), Madrid, Spain

**Kellie F. Flood-Shaffer, M.D., F.A.C.O.G.** Department of Obstetrics and Gynecology, University of Cincinnati Medical Center, University of Cincinnati, Cincinnati, OH, USA

**Annette Gary, Ph.D., M.S.N., R.N.** Starcare Specialty Health System, Lubbock, TX, USA

**Lauren Gates, M.S., A.C.S.M., C.E.S., Bachelor of Science, Master of Science** Department of Health, Exercise and Sport Sciences, Texas Tech University, The Woodlands, TX, USA

**Claudia Cardona Gonzalez, Ph.D. in Physical Activity for Health, (M.Sc. equivalent), Bachelor in Physical Activity and Sports Sciences** Department of Sports Fundamentals, Universidad Europea de Madrid, Madrid, Spain

Department Fundamentos de la moticidad y el Entrenamiento Deportivo, Universidad Europea de Madrid, Madrid, Spain

**Ángela García González, M.D., Ph.D.** Pharmaceutical and Food Sciences, University San Pablo-CEU, Madrid, Spain

**Eduardo Iglesias-Gutiérrez, B.Sc., Ph.D., M.Sc.** Department of Functional Biology (Physiology), University of Oviedo, Oviedo, Asturias, Spain

**Maesy Indigo, M.S.** Department of Health, Exercise & Sport Sciences, Texas Tech University, HESS, Lubbock, TX, USA

**Melissa R. Iñigo, M.S. in Exercise and Sports Sciences** Department of Health, Exercise, and Sports Sciences, Texas Tech University, Lubbock, TX, USA

Doctoral student University of Maryland, College Park, MD, USA

**C. Roger James, Ph.D., F.A.C.S.M.** Department of Rehabilitation Sciences, Center for Rehabilitation Research, Texas Tech University Health Sciences Center, Lubbock, TX, USA

**Marta Montil Jiménez, Ph.D. in Physical Activity for Health, Bachelor in Physical Activity and Sports Sciences** Department of Kinesiology and Athletic Training, Universidad Europea de Madrid, Madrid, Spain

**Shannon L. Jordan, Ph.D.** Department of Kinesiology, Texas Wesleyan University, Ft. Worth, TX, USA

**Shelby D. Kloiber, Masters of Science** Department of Health, Exercise, and Sport Sciences, Texas Tech University, Lubbock, TX, USA

**José E. Almaraz Marroquin, M.D.** Department of Internal Medicine, Hospital Universitario de Gran Canaria, Dr. Negrín, Spain, Las Palmas, Gran Canaria, Spain

**Marilyn Massey-Stokes, Ed.D., C.H.E.S., F.A.S.H.A., I.C.** Department of Health Studies, Texas Women's University, Denton, TX, USA

**Jacalyn J. Robert-McComb, Ph.D., F.A.C.S.M., A.C.S.M., P.D.** Department of Health, Exercise, and Sports Sciences, Texas Tech University, Lubbock, TX, USA

**Tyrel S. McCravens, Master of Science in Exercise Physiology** Department of Health, Exercise, and Sport Sciences, Texas Tech University, Lubbock, TX, USA

**Jennifer J. Mitchell, M.D., F.A.A.F.P.** Department of Family and Community Medicine, Texas Tech University Health Sciences Center School of Medicine, Lubbock, TX, USA

**Fernando Naclerio, Ph.D.** Sports Sciences, Centre of Sport Science and Human Performance, School of Science, University of Greenwich, Kent, UK

**Reid L. Norman, Ph.D.** Department of Pharmacology and Neuroscience, Texas Tech University Health Sciences Center School of Medicine, Lubbock, TX, USA

**Phillip S. Sizer Jr., P.T., Ph.D., O.C.S., F.A.A.O.M.T., Ph.D. in Sensory Motor Control, M.Ed. in Ex Science, B.S. in Physical Therapy** SCD Program in Physical Therapy, Department of Rehabilitation Sciences, Texas Tech University Health Sciences Center, Lubbock, TX, USA

**Anna M. Tacón, Ph.D.** Texas Tech University, Lubbock, TX, USA

**Maria Fernandez-del-Valle, Ph.D. in Physical Activity for Health, (M.Sc. equivalent), Bachelor in Physical Activity and Sports Sciences** Department of Health, Exercise, and Sport Sciences, Texas Tech University, Lubbock, TX, USA

**Carolina Chamorro-Viña, Ph.D.** Faculty of Kinesiology, Biomedicine and Health's Sciences, University of Calgary, Calgary, AB, Canada

**Mimi Zumwalt, M.D.** Department of Orthopedic Surgery and Rehabilitation, Texas Tech University Health Sciences Center, Lubbock, TX, USA

# Part I

# Focusing on Active Female's Health Issues: Unique Gender Related Psychological and Physiological Characteristics of Females

# Body Image Concerns Throughout the Lifespan

1

Jacalyn J. Robert-McComb
and Marilyn Massey-Stokes

**Abstract**

Due to the rising rates of eating disorders and obesity, increasingly more attention is being paid to body image and body image difficulties. Body dissatisfaction, which is ubiquitous among girls and women, can be defined by the difference between one's perceived body size and ideal body, particularly with regard to the desire to be thin. Body dissatisfaction has become so commonplace that it has been described as "normative discontent." It is also considered one of the most robust risk and maintenance factors for clinical eating disorders. A wide range of risk factors contribute to the development of body dissatisfaction, including biological and physical factors, sociocultural influences, and individual characteristics. In addition, females who place a strong emphasis on thinness and physical appearance and routinely engage in body comparison are especially vulnerable to experiencing body dissatisfaction. Body image problems and disordered eating behaviors know no boundaries; they impact females across age groups, ethnicities, cultures, and socioeconomic levels. Therefore, it is essential for health professionals to understand the development of body image difficulties and be knowledgeable about body image assessment techniques and effective prevention and intervention programs. Armed with this insight, health professionals will be in position to foster healthy body image and enhance quality of life among females across the lifespan.

**Keywords**

Body image • Body dissatisfaction • Risk factors • Assessment • Theoretical foundations • Prevention

J.J. Robert-McComb, Ph.D., FACSM, ACSM P.D. (✉)
Department of Health, Exercise, and Sports Sciences, Texas Tech University, Lubbock, TX, USA
e-mail: jacalyn.mccomb@ttu.edu

M. Massey-Stokes, Ed.D., C.H.E.S., F.A.S.H.A., I.C.®
Department of Health Studies, Texas Woman's University, Denton, TX, USA
e-mail: mmasseystokes@twu.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_1, © Springer Science+Business Media New York 2014

## 1.1 Learning Objectives

After completing this chapter, readers should be able to:

1. Explain the difference between normal body image concerns, body dissatisfaction, and body image distortion.
2. Analyze risk factors that contribute to body dissatisfaction in females.
3. Compare and contrast how body image concerns affect females throughout the life course.
4. Discuss body image among various ethnic and cultural groups.
5. Examine different assessment tools for the evaluation of body image.
6. Explain the different categories of prevention.
7. Examine theoretical foundations that can be applied to body image interventions.
8. Examine various strategies that can be used in body image interventions.
9. Discuss future directions for body image and eating disorder research.

## 1.2 Introduction

Positive body image is integral to healthy development and overall well-being throughout the lifespan. Body image is a multidimensional and highly complex construct [1] that can be defined in general terms as "the subjective evaluation of one's appearance" [2] (p. 4). Body image encompasses cognitive, perceptual, affective, and behavioral dimensions [3] and is a dynamic representation that the person constructs over time from daily experiences and within a certain sociocultural milieu [4]. Due to the rising rates of eating disorders and obesity, increasingly more attention is being paid to body image and body image difficulties [5]. Various terms have been used to depict body image concerns, including negative body image, body dissatisfaction, body dysphoria, body image distortion, and body image disturbance. Due to the complexity of the body image construct, researchers have typically focused on examining disturbances within the cognitive-affective (evaluative) and perceptual components of body image. A disruption within the evaluative component is usually represented as body dissatisfaction [6], which refers to a person's negative self-evaluation of his or her body weight, size, and shape that can lead to cognitive, psychological, affective, and behavioral disturbances [7, 8]. Body dissatisfaction can also be defined by the difference between one's perceived body size and ideal body size. In contrast, a disturbance within the perceptual component is usually referred to as body image distortion, which involves a person's inability to accurately perceive body size and shape [9] and is a symptom of eating disorders such as anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified [10].

It is widely recognized that body dissatisfaction is ubiquitous among girls and women, particularly with regard to the desire to be thin. In fact, body dissatisfaction has become so commonplace that it has been described as "normative discontent." [11] Moreover, body dissatisfaction is considered one of the most robust risk and maintenance factors for clinical eating disorders [12]. Body image and eating disturbances have also been linked to other psychological problems, including depression, low self-esteem [13–16] as well as anxiety [14], body dysmorphic disorder [17], self-harm and childhood sexual abuse [18], and social phobia [14, 19]. In sum, body image and eating problems hinder healthy development and negatively impact overall well-being and quality of life.

Negative body image, body image disturbances, and disordered eating behaviors know no boundaries; they impact females across age groups, ethnicities, cultures, and socioeconomic levels. It is essential for health educators and health care clinicians to understand the etiology and development of body image problems and be knowledgeable about body image assessment techniques and effective prevention and intervention programs. It is also important for health professionals to reflect about their own perceptions of body image and eating concerns and how these perceptions may influence their own attitudes and behaviors as well as their interpersonal interactions with clients and patients.

## 1.3 Research Findings

### 1.3.1 Risk Factors for the Development of Body Dissatisfaction

A wide range of risk factors contribute to the development of body dissatisfaction, including biological and physical factors, sociocultural influences, and individual characteristics [16, 20]. Females with a higher body mass index (BMI) that is not in line with societal expectations of a thin ideal body type—the thin ideal—can experience body dissatisfaction. Sociocultural factors often exert a powerful influence on body image by promoting the thin ideal, which can be particularly problematic for females who tend to internalize such messages [16, 21–25]. Even females from ethnic groups who are generally more accepting of larger body types can experience an internal tug-of-war regarding their body image and sense of self-worth, particularly when they are acculturated or exposed to Western or Western-influenced cultures that promote the thin ideal [26, 27]. In addition, females who place a strong emphasis on thinness and physical appearance and routinely engage in body comparison are especially vulnerable to experiencing body dissatisfaction [28–30].

Sociocultural influences on body image have been the focus of numerous studies, and various sociocultural models have been developed to depict how these factors promote the thin ideal as the societal standard for beauty [28, 31]. More specifically, the tripartite influence model (see Fig. 1.1) posits that three major sociocultural influences—peers, parents, and media—play a role in the development of body dissatisfaction, eating disorders, and negative affect.

The model also proposes that internalization of the thin ideal and appearance comparison mediate the relationships between these influences and body image and eating concerns [25]. This model has been used with diverse cultural samples and appears to be a viable model for studying risk factors leading to body dissatisfaction and eating disorders [14, 25, 32].

Within the sphere of major sociocultural influences, mass media transmit powerful messages to both adults and youth, particularly with regard to body image. This media influence conveys another level of meaning in light of the fact that US children and adolescents aged 8–18 use media roughly 7½ h/day [33]. Furthermore, because most young people use two or more media concurrently, they actually engage with media for more than 10½ h/day. This time does *not* include doing schoolwork on the computer, texting, or talking on a cell phone [33]. Lower-income,

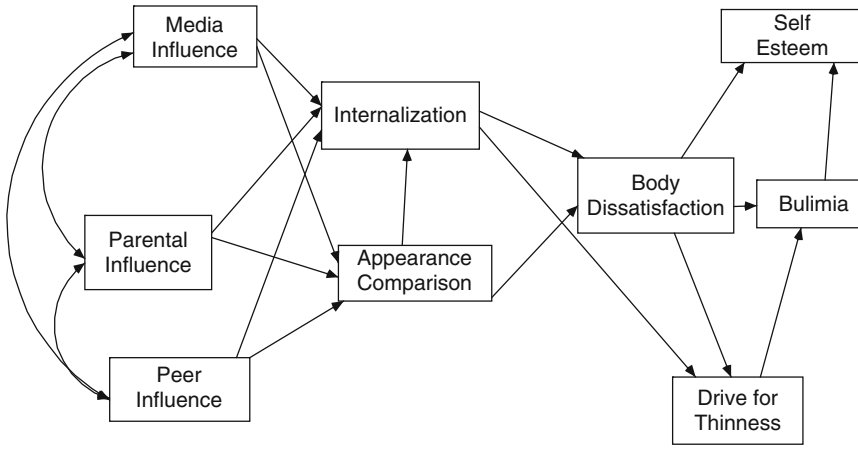



**Fig. 1.1** The theoretical tripartite model. Source: Rodgers R, Chabrol H, Paxton SJ. An exploration of the tripartite influence model of body dissatisfaction and disordered eating among Australian and French college women. Body Image. 2011:208–215. doi:10.1016/j.bodyim.2011.04.009. p. 210

Hispanic, and African-American children consume much more media than their middle-class and Caucasian peers. Young children are also mass media consumers; although computer and Internet use are on the rise, television is still the medium of choice for this age group [34].

There is also evidence to suggest that children watch adult television after the age of 9; and in watching these programs, they learn about social norms that might influence their attitudes and behaviors [35]. For example, many adult programs contain content and cues that uphold the thin ideal, which can influence the way young girls view their own bodies. In fact, young girls have mentioned that they learn about dieting through the media [36]. Furthermore, research has shown that perceived pressure from the media predicts body image and eating difficulties among girls aged 10–18 [37].

Parents and other caregivers make many decisions regarding the media use of young people; however, they often do not set or consistently enforce rules regarding their children's media exposure [33]. Therefore, the subtle and overt messages that young people receive about body image via various media outlets often go unchecked. Children and youth do not yet possess completely developed higher-order cognitive skills (e.g., critical thinking and problem solving), which makes them even more vulnerable to the constant barrage of media messages endorsing the thin ideal. Studies have shown that a relationship exists between media influences and body image development in girls. For example, preadolescent and adolescent girls who watch television shows with an emphasis on appearance experience less satisfaction with their own appearance [24, 35, 38]. Moreover, meta-analyses have clearly demonstrated that exposure to the thin ideal in the media is strongly related to negative body image in girls and women, with adolescent girls displaying particular vulnerability [39, 40]. According to Bell and Dittmar [41] (p. 489), "it is not the *type* of media exposure that is important in understanding girls' vulnerability to negative body image, but rather the extent of girls' identification with media models."

### 1.3.2 Body Image Concerns Across Age Groups

#### 1.3.2.1 Preadolescent and Adolescent Females

Most studies concerning body image in preadolescent and adolescent females have focused on girls' desire to be thinner [5, 16, 42]. Researchers have shown that psychological processes related to body dissatisfaction are already well established by the age of 9 [23, 35, 43], and the "normative discontent" with body shape and size that is so common among adolescent females and young adult women is applicable to young girls as well [38, 44]. Many girls and women who internalize these "thin is in" messages are unable to meet the high standard and therefore often experience body dissatisfaction [38, 41, 45]. Girls as young as 5 and 6 experience a greater awareness and internalization of the thin ideal in ways that negatively impact their body image development and self-esteem [29, 46]. Therefore, it appears that the development of body dissatisfaction emerges around the time that girls start school [16].

The peer group is another key sociocultural influence in the development of body image and body image concerns, especially among adolescent females. Schools, which are major socialization environments for young people [28] and give rise to various peer groups, contribute to the formation of "appearance cultures" [24] in which adolescent girls integrate sociocultural standards for female attractiveness (e.g., the thin ideal) into their peer group cultures [23, 42, 47]. Adolescent girls who frequently engage in appearance conversations and appearance comparisons with their peers are more likely to internalize the thin ideal and experience body dissatisfaction [22–24]. In addition, watching or reading appearance-based media is significantly related to appearance conversations with peers, which, in turn, are related to internalization of appearance ideals and body dissatisfaction [23]. A related peer group element involves heterosocial involvement, which has also been associated with peer pressure to be thin; and this peer pressure has been associated with increased body dissatisfaction

[42]. Data from Project EAT-II, a longitudinal study of large, ethnically, and socioeconomically diverse group of adolescents, revealed that adolescent females are strongly influenced by their peers' dieting behaviors. More specifically, the more the teens perceived that their friends were dieting, the more likely they were to report engaging in chronic dieting, extreme weight control behaviors, and binge eating 5 years later [48]. Therefore, peer groups exert a strong influence on adolescent females' attitudes and behaviors related to body image and weight control.

#### 1.3.2.2 Young Adult Women

A sizeable number of young adult females are concerned about their body weight and shape as well [49]. According to the American College Health Association [50], an estimated 44 % of college women are dieting to lose weight; and approximately 61 % of these young women are exercising to lose weight. Other studies have also shown that college-aged females struggle with body image issues and report high levels of body dissatisfaction and disordered eating [14, 51–53]. In addition, college females who report more fear about receiving negative appearance evaluation from others experience a greater drive for thinness and higher body dissatisfaction [54].

There is support for the sociocultural model as it relates to body image and eating disorders among college-aged women [55]. More specifically, sociocultural pressures aimed at the thin ideal exert the most negative impact on young women when they are internalized [56, 57]. To shed light concerning *how* internalization of the thin ideal may lead to body dissatisfaction, Fitzsimmons-Craft et al. [57] (p. 48) found that social comparison and body surveillance mediated the relationship between thin ideal internalization and body dissatisfaction in a sample of college-aged women. Nevertheless, "only body surveillance emerged as a significant specific mediator of this relation." In a different study [14], the combination of media influence and social phobia emerged as a significant predictor of body dissatisfaction in college-aged females.

A related aspect of body image centers on "fat talk," which is a term for how females talk with each other about the size and shape of their bodies, generally in negative terms [58]. Studies have revealed that fat talk occurs so frequently among college-aged women that it is considered a "normative phenomenon." [59] Young women have indicated that engaging in fat talk acts as a coping mechanism of sorts because it helps them express distress about *feeling* fat as opposed to *being* fat. However, engaging in fat talk may actually be maladaptive because this type of talk is linked with thin-ideal internalization, body dissatisfaction, body image disturbance, and eating pathology [59, 60].

#### 1.3.2.3 Older Women

Similar to younger females, body image in older women is multifaceted and varies among women [61]. However, there are some distinct differences. For example, as women age, they experience physical, social, and environmental changes that impact how they perceive their bodies [62, 63]. Although more attention has been devoted to studying body image in middle-aged and older women in recent years, there have been mixed findings regarding the relationship between aging and body image [61, 62]. For example, Webster and Tiggemann [64] found no difference with age, while other researchers [65, 66] have supported the thought that women who experience body dissatisfaction may struggle with this concern throughout their lifetime.

Many correlates and risk factors associated with body dissatisfaction and disordered eating that are found in younger females have also been found in older women, such as BMI, sociocultural influences, and internalization of the thin ideal. Nevertheless, other factors present unique body image challenges to older women, including menopause and anxiety related to the aging process [6]. Women in midlife and beyond may face similar eating- and weight-related obstacles as younger girls and women, including body dissatisfaction and body image distortions; and these issues are often intensified by the aging process. For example, life events that often take place in midlife, such as career changes, marital problems, divorce, "empty-nest" syndrome, and chronic illness, can create even more distress for

women who are already struggling with body image and eating difficulties. Among the indicators that body image and disordered eating among older women appear to be on the rise is that fact that there has been a 42 % increase in the number of women over the age of 35 who sought treatment within the past 10 years [67].

In a large Internet survey study of women aged 50 and older, 13 % of the sample reported current eating disorder symptomology. In addition, over 70 % of the participants reported experiencing body dissatisfaction, with a large percentage (83.9 %) expressing dissatisfaction with the stomach. Researchers also found that higher BMI was associated with higher rates of diet pill/diuretic use, both of which are considered maladaptive weight management behaviors and have been reported in other studies. However, regardless of BMI, excessive concern with body shape and weight and body dissatisfaction can negatively impact women's self-esteem and overall quality of life, and even lead to full-blown clinical eating disorders. Therefore, health professionals need to be aware of and appropriately screen for eating and body image difficulties among older women [68].

In spite of these challenges and risk factors, Liechty's [61] (p. 84) qualitative study of older women revealed "complex cognitive and behavioral means by which older women were able to feel satisfaction with their bodies despite desire for physical change." Participants conveyed that health and functionality were more important to their body image than their physical appearance. Those with positive body image focused on controllable elements of their physical appearance (e.g., clothing) and had developed at least some degree of acceptance of their physical imperfections. Conversely, participants who highly valued youthfulness and conforming to society's thin ideal experienced increasing body dissatisfaction as they aged. According to McLean et al. [69], appearance acceptance and placing less emphasis on the appearance aspect of self-concept may serve as protective factors that buffer aging women from the negative impact of body dissatisfaction.

In addition, Liechty and Yarnal [62] (p. 1213, 1215) found that women's body image exhibited both stable and fluctuating patterns throughout the lifespan. The participants' thoughts about body image went beyond "level of satisfaction" and "included evaluations of health and ability, beliefs about the importance of appearance, and feelings about their overall lives." These findings supported Hatch's [70]"life course perspective" about body image, which can help researchers design more robust studies, including how the body image construct might change as women age [61–63].

#### 1.3.2.4 Females with Type 1 Diabetes

Females with type 1 diabetes are an important subgroup who are at heightened risk for experiencing body dissatisfaction and eating problems. According to Jones et al. [71], eating disorders occur approximately twice as often among adolescent females with type 1 diabetes as in their peers without diabetes [72–74]. Insulin omission or restriction along with maladaptive eating and exercise attitudes and behaviors can lead to a number of negative health and medical consequences for females with type 1 diabetes [71–74]. Health professionals working with these individuals should be alert to warning signs of a possible eating disorder, including symptoms of depressed or anxious mood, over-concern about body weight and shape, atypical exercise habits (sometimes accompanied or followed by frequent hypoglycemia), and very low-calorie meal plans. In addition, unexplained increases in hemoglobin A1c (HbA1c) values and recurring problems with diabetic ketoacidosis (DKA) should raise a raise flag that insulin restriction may be occurring [71, 72].

### 1.3.3 Body Image Among Different Ethnic and Cultural Groups

#### 1.3.3.1 Body Dissatisfaction Across the Globe

Although females residing in westernized nations (e.g., USA, Australia, UK) have been the focus of much body image and eating disorder research, studies have shown that these issues are a global concern [22, 75–83]. It is important to examine body dissatisfaction across various cultures in

order to obtain richer understanding regarding how the cultural environment influences body image concerns [79].

Numerous studies point to the influence of "Westernization" in advancing the thin ideal and contributing to the development of body dissatisfaction globally, but other factors appear to play a role as well [75, 79–81]. For example, results from the International Body Project I [81] indicated that within Malaysia and South Africa, heavier bodies are preferred in low-SES areas compared to high-SES areas; however, this is not the case in Austria. Also, some studies show mixed results regarding the impact of Western influences on body image concerns, such as those that have been conducted in Latin-American countries [79].

In addition to the degree of Westernization, other factors that appear to contribute to different body weight ideals across different cultures include the role and level of equality of women in society, the acceptance of the overweight stigma [81], and BMI [79].

The thin ideal for female attractiveness is also esteemed in China, and some studies have shown that the relation between appearance pressure and comparison to body dissatisfaction in Chinese adolescents is somewhat similar to that of adolescents in Western countries. Other findings have revealed some cultural differences in select developmental and interpersonal influences on body dissatisfaction, with Chinese adolescents showing lower levels of body dissatisfaction [22]. However, according to Holmqvist and Frisén [79], when Asian research participants are recruited from parts of East Asia that are affluent and more westernized, they appear to demonstrate more body dissatisfaction than their US counterparts. Chen and Jackson's [22] study also highlighted middle adolescent Chinese girls' susceptibility to body dissatisfaction and perceived appearance pressure from media and interpersonal influences compared to their early adolescent peers.

Japan is another country that has witnessed an alarming increase in body image and eating problems [32]. According to studies cited by Yamamiya and colleagues [32], eating disorders have increased approximately sixfold over the past 25 years; and Japanese women's perception of the "ideal" body type appears to be thinner than that of American women. In addition, a study involving Japanese female undergraduate students revealed that sociocultural factors (family, peers, media) appear to influence body image and eating problems similar to what is found with US samples [32]. For more thorough discussions of body image and eating disorders across various cultures, see Anderson-Fye [75], Holmqvist and Frisén [79], Levine and Smolak [80], and Swami et al. [81].

#### 1.3.3.2 Body Dissatisfaction Among US Ethnic Groups

According to Levine and Smolak [80] (p. 224), "ethnicity is a *culturally constructed* definition of a group of people who are assumed to be related in terms of values and beliefs and, often, in terms of race." In the USA, the relationships among ethnicity, acculturation, and body satisfaction are not clear [80, 84–88]. One of the hindrances associated with body image research is that many of the studies have been conducted in samples of predominantly Caucasian female samples, with less focus on ethnic differences [85], particularly among preadolescents [5]. When body image among different ethnic groups has been studied, much of the focus has focused on differences between Caucasians and African Americans, with surprisingly few studies investigating body image among Hispanics/Latinos, Asian Americans, and Native Americans [89]. Furthermore, body image research conducted with ethnically diverse populations may not account for the heterogeneity that exists within each group.

In studies examining body image and problematic eating among ethnically and culturally diverse samples, results have been mixed. For example, many studies have shown that African American females tend to be more accepting of larger body types and display higher levels of body satisfaction than other ethnic groups, whereas study findings are less clear-cut concerning Hispanic/Latino and Asian American groups. Some studies indicate no significant differences between these groups and Caucasians,

while other studies reveal differences [80]. These mixed findings have led researchers to posit that some of the differences in body dissatisfaction among various ethnicities may depend on the assessment instruments that are used [79, 80]. Even if females from two different ethnic groups display similar levels of body dissatisfaction, the cultural messages about body image as well as the impact of these messages are likely to be different for each group [5]. For more detailed discussions concerning the relationships of body dissatisfaction and eating disorders across different ethnic and cultural groups, see Anderson-Fye [75], Franko and George [89], Holmqvist and Frisén [79], and Levine and Smolak [80].

### 1.3.4 Body Image Assessment

It is important to emphasize that assessment is a process, and there is no particular instrument or battery of tests that is appropriate to use in all situations [90]. Moreover, due to the multidimensionality of the body image construct and the wide availability of various instruments, it is particularly important to carefully consider the selection of body image assessment tools [91].

Thompson [91] outlined ten tips for enhancing body image assessment in clinical and research settings, including the importance of selecting instruments with established reliability and validity and using selected instruments with appropriate target populations. Banasiak et al. [92] pointed out that many instruments used to assess body image concerns in adolescents have been validated using adult samples. Nevertheless, many of the measures can still be used with adolescent females when care is taken to ensure that girls understand the terms used in the particular instrument. For example, the Physical Appearance State and Trait Anxiety Scale [93] was validated with an adult sample, yet exhibited excellent internal reliability (0.93) in measuring weight-related body dissatisfaction in adolescent girls aged 14–16 [41]. In the same study [41], researchers measured appearance dissatisfaction with an adapted version of the Body Image State Scale, a six-item scale that has good construct validity [94] and demonstrated high internal reliability with an adolescent sample in a previous study [95]. As illustrated in these examples, it is important for researchers to establish new reliability and validity scores whenever they use an instrument with a target population that differs from the standardized sample [91]. In addition, researchers have acknowledged the need to develop valid and reliable instrumentation for studying body image in diverse female populations, including younger girls [75], older women [61, 65], and females from diverse ethnic and cultural backgrounds [55, 61, 80, 96].

#### 1.3.4.1 Body Image Assessment Scales and Questionnaires

There are a number of well-validated instruments that have been developed to assess body image and eating concerns in children, adolescents, and adults; many of these measures are discussed in more detail elsewhere [3, 90, 96–100]. Table 1.1 lists a few of the body image measures that have been reported in the research literature with an internal consistency rating and test-retest reliability rating of at least 0.70 [93, 94, 101–107].

In addition, examples of body image questionnaires that have been validated for college-aged women and have internal consistency and test-retest reliability scores above 0.70 are located in Appendices 1–3: Body Image Quality of Life Inventory [101], Body Image Concern Inventory [105], and Physical Appearance State and Trait Anxiety Scale [93]. In addition, Cash and Grasso [108] reported the normative data and acceptable internal reliability measures of four body image instruments—Body Image Disturbance Questionnaire [109], Appearance Schemas Inventory-Revised [110], Body Image Coping Strategies Inventory [111], and Body Image Quality of Life Inventory [101, 112]. These instruments measure various facets of the body image construct and were used across seven studies with female and male college students. Other valid and reliable scales that can be used in body image research include Body Image Assessment Scale-Body Dimensions [113]; Body Shape Questionnaire [114], which has a shortened version [115] and is available in different language

**Table 1.1** Instruments for assessing body image with high internal consistency and test-retest coefficients (>0.70)

| Author | Test name | Description of test | Reliability<br>IC: Internal consistency<br>TR: Test-retest | Standardization sample |
|---|---|---|---|---|
| Cash and Fleming [101] | Body Image Quality of Life Inventory | A 19-item instrument designed to quantify the impact of body image on aspects of one's life<br>Participants rate the impact of their own body image on each of the 19 areas using a 7-point bipolar scale from −3 to +3 | IC: 0. 95<br>TR: 0.79 | 116 college-aged women ($M = 21.3 \pm 5.1$) |
| Cash et al. [94] | Body Image States Scale | A multi-item measure of momentary evaluative/ affective experiences of one's physical appearance | IC: 0.77 (women)<br>IC: 0.72 (men)<br>TR-state: 0.69 (women)[a]<br>TR-state: 0.68 (men)[a] | 174 college students—116 women, 58 men (median age = 20) |
| Cash and Szymanski [102] | Body-Image Ideals Questionnaire | A measurement of self-perceived discrepancies from and importance of internalized ideals for multiple physical characteristics | IC-BIQ discrepancy: 0.75<br>IC-BIQ importance: 0.82<br>IC-weighted discrepancy: 0.77<br>TR: none given | 284 college undergraduate women at a mid-Atlantic urban university |
| Garner [103]; Garner and Olmstead [104] | Eating Disorder Inventory (EDI and EDI-2). Body Dissatisfaction Scale | 9-item subscale assesses feelings about satisfaction with body size; items are 6-point, forced choice; reading level is 5th grade | IC: Adolescents (11–18)<br>Females = 0.91<br>Males = 0.86<br>Children (8–10)<br>Females = 0.84<br>TR: None given | 610 males and females ages 11–18 (Shore & Porter, 1990) 109 males and females ages 8–10 (Wood et al., 1996) |
| Littleton et al. [105] | Body Image Concern Inventory | A *brief* instrument for assessing dysmorphic concern; only takes a few minutes to answer. Despite its brevity, the BICI provides an assessment of body dissatisfaction, checking and camouflaging behavior, and interference due to symptoms—such as discomfort with and avoidance of social activities (see Appendix 2) | IC: 0.93<br>TR: None given | 184 undergraduates at a medium-sized Southeastern University; approximately 89 % were women |
| Reed et al. [93] | Physical Appearance State and Trait Anxiety Scale | Participants rate the anxiety associated with 16 body sites (8 weight relevant, 8 nonrelevant); trait and state versions available | IC: Trait: 0.88–0.82, state: 0.82–0.92 TR: 2 weeks, 0.87 | 205 female undergraduate students |
| Shisslack et al. [106] | McKnight Risk Factor Survey III (MFRS-III) | Participants use 5-item subscale that assesses concern with body weight and shape | IC: Elementary = 0.82<br>Middle school = 0.86<br>High school = 0.87<br>TR: Elementary = 0.79<br>Middle school = 0.84<br>High school = 0.90 | 103 females, 4–5th grade;<br>420 females, 6–8th grade;<br>66 females, 9–12th grade |
| Wooley & Roll [107] | Color-A-Person Body Dissatisfaction Test | Participants use five colors to indicate level of satisfaction with body sites by masking on a schematic figure | IC: 0.74–0.85<br>TR: 2 weeks (0.72–0.84)<br>4 weeks (0.75–0.89) | 102 male and female college students, 103 bulimic individuals |

[a]Acceptable for a *state* assessment

[116]; Eating Disorder Inventory-3 [117], which contains the Drive for Thinness and the Body Dissatisfaction subscales; the Sociocultural Attitudes Towards Appearance Scale-3 for measuring multiple societal influences on body image and eating disturbances [118]; and Children's Body Image Scale [119, 120]. Researchers have also explored using realistic 3-dimensional body-scan images for body image research and found that the scanned images are a viable alternative to contour line drawings [121]. Innovative tools for investigating changes in adolescent body perception have also been developed, such as the Adolescent Body-Shape Database and Adolescent Body Morphing Tool [122].

## 1.4 Contemporary Understanding of the Issues

### 1.4.1 Body Image Interventions

#### 1.4.1.1 Prevention Categories

Body image and eating disorder prevention programs focus on preventing or delaying the onset of subclinical and full-blown clinical eating disorders by reducing risk factors and increasing protective factors that promote resilience and overall health and well-being [123–126]. According to a report from the National Academies [127], there are different categories of prevention activities that can drive health promotion interventions—universal, selective, and indicated. *Universal* prevention programs are aimed at educating an entire group (population). These programs are often implemented in schools and focus on educating children and youth about particular health issues such as body image and eating disorders. Many researchers have also called for universal and *integrated* prevention programs that address the spectrum of body image and weight-related issues that encompass both disordered eating and childhood obesity [128–131]. While these programs are aimed at preventing risk factors such as body dissatisfaction, dieting, and unhealthy weight-control practices [129, 132], they can also foster important protective factors such as self-esteem, self-efficacy, media literacy, social-emotional learning, stress management, creative problem solving, and overall resilience [133].

*Selective* prevention programs are designed to reach a population subgroup who is considered at higher-than-average risk for a particular health concern (e.g., eating disorders). *Indicated* (or *targeted*) prevention programs target individuals considered to be at very high risk or actually exhibiting early signs of the disorder. A meta-analysis [134] highlighted that both universal and targeted prevention programs produced favorable and significant effects at follow-up on the measures of risk and eating pathology; this finding held steady for younger and older participants. In addition, the analysis demonstrated that the most effective programs are those that target high-risk individuals and use interactive strategies (as opposed to didactic ones) to teach life skills for healthy eating and physical activity and/or countering sociocultural pressure promoting the thin ideal. It is also important to note that evidence from meta-analyses has not supported iatrogenesis; therefore, "concerns about inducing body image or eating problems should be carefully considered but should not deter the development of prevention programs" [135] (p. 219).

#### 1.4.1.2 Theoretical Foundations

Health education and health behavior theories and models can guide the development and evaluation of health promotion and education programs and interventions [136]. For example, the ecological model can be effective for prevention programs aimed at improving body image and decreasing eating disorder risk [81, 125, 135]. The underlying premise of the ecological model is that there are multiple levels of influences on health attitudes and behaviors, including intrapersonal (biological, psychological), interpersonal (social, cultural), organizational, community, physical environmental, and policy. Ecological models serve as "comprehensive frameworks for understanding the multiple and interacting determinants of health behaviors. . . . and can be used to develop comprehensive intervention approaches that systematically target mechanisms of change at each level of influence" [137] (p. 466). For example, healthy body image can be

promoted through targeting individual attitudes, knowledge, beliefs, and behaviors (intrapersonal); involving the family and peers in reinforcing positive body image in concert with healthy and balanced nutrition and physical activity (interpersonal, environmental); integrating healthy body image, eating, and physical activity interventions into school health programs (organizational, environmental); and advocating for communication, education, and motivational campaigns with messages that promote healthy body image (community).

Additional theoretical frameworks that can be used to guide body image interventions include Social Cognitive Theory (SCT) [138] and Social Marketing [139]. The hallmark of SCT is reciprocal determinism, which is the dynamic interaction of individuals and groups, their behavior, and the environment. Other SCT concepts relevant to the promotion of healthy body image include outcome expectations (beliefs about the likelihood and value of the consequences associated with behavioral choices), self-efficacy (beliefs about one's ability to successfully engage in positive behavior change), observational learning (learning to perform new behaviors by observing others, e.g., via peer modeling), facilitation (providing tools and resources or modifying the environment to facilitate behavior change), and self-regulation (controlling oneself through self-monitoring, goal setting, self-reward, and social support) [140]. School- and community-based body image interventions can reinforce key SCT concepts by emphasizing personal and group goal setting, teaching media-literacy skills, encouraging healthy peer group interaction, providing regular opportunities to practice decision-making and problem-solving skills linked to real-life body image issues, and incorporating meaningful family involvement activities.

Healthy body image can also be fostered through social marketing, which is designed to influence voluntary behavior change that can positively impact health and quality of life at the intrapersonal, interpersonal, organizational, community, and public policy levels, thereby contributing to an ecological approach to mitigate body image and eating problems. Social marketing includes communication that focuses on the four "Ps" of the marketing mix: product, price, place, and promotion [141]. For example, with positive body image as the *product*, health professionals can develop messages and slogans to emphasize the benefits of healthy body image and identify the negative costs associated with body dissatisfaction. These messages can influence females' perceptions of the cost-benefit ratio associated with adopting and maintaining attitudes and behaviors reflecting positive body image, sound nutrition, and healthy physical activity (*price*). Health professionals can also increase access to quality information about body image through various *places*, such as schools and other education centers, medical offices, health clinics, community centers, churches and other places of worship, hair and nail salons, and social media outlets. *Promotion* of healthy body image can occur by making incentives accessible to the target audience, including popular items such as T-shirts and water bottles, sling bags, posters, magazine and Internet ads, social media contests, and games.

With a consumer focus in mind, targeting social norms has proven to be another successful social marketing strategy. Social norms have been defined as "expectations about how different people will evaluate our behavior and our willingness to be guided by their evaluation" [140] (p. 172). Therefore, because "perception usually trumps reality" [141] (p. 447), social norms marketing can be used to inform females about the actual frequency of certain attitudes and behaviors linked with body image dissatisfaction (e.g., dieting among peers, unrealistic expectations for body type, internalizing the thin ideal), with the intent to create social pressure for change. Health professionals can also implement programs to promote a new social norm [142] emphasizing healthy body image, followed by positive reinforcement at different levels of influence within the ecological framework (e.g., social support from family and peers, "body image friendly" messages and environments that facilitate the development of positive body image). For additional discussions involving theoretical approaches and prevention programs aimed at

promoting healthy body image and preventing disordered eating, see Bauer et al. [143], Levine and Smolak [135], Massey-Stokes et al. [125], and Sinton and Taylor [124].

#### 1.4.1.3 Strategies Aimed at Sociocultural Influences

Developing interventions aimed at preventing or reducing body image and eating problems must include skill development for recognizing and effectively managing sociocultural pressures espousing the thin ideal. Within this scope, it is very important to emphasize the key mediational variables of appearance comparison and internalization of the thin ideal [25, 32, 55], including strategies for dealing with these tendencies. Body image interventions should also accurately reflect the central role of popular culture and traditional values across diverse cultural, ethnic, and socioeconomic populations [22, 144, 145]. The heterogeneity within each group should be taken into account when planning and implementing body image intervention programs so as not to miss other manifestations of disordered eating and related health issues [87]. It can also be beneficial to tailor body image interventions to address a range of treatment barriers that have been identified across ethnic groups [5, 27].

Interactive programs that can be effective in addressing sociocultural influences on females' body image include those emphasizing media literacy [23, 41, 129, 146–148], self-awareness and self-esteem [129, 146, 148], healthy weight management [147–150], peer support groups [23, 151, 152], and healthy emotions and coping skills [126, 148]. Studies have also supported the use of a dissonance intervention to increase girls' resistance to internalizing the thin ideal that often resonates from peer appearance conversations [23] and other appearance-related messages that are so prevalent in the lives of adolescent females [150]. Dissonance education involves having young women with body image difficulties who have internalized the thin ideal participate in verbal, written, and behavioral exercises in which they critique this ideal. The intended result of such activities is that they "will result in psychological discomfort that motivates [the young women] to reduce internalization of the thin-ideal, which decreases body dissatisfaction, dieting, negative affect, and eating disorder symptoms [150]" (p. 2).

Related research has shown that high general self-determination (a strong sense of self that is "integrated, unified, and noncontingent") can buffer females from having an adverse response to the thin ideal portrayed through the media. Therefore, prevention efforts aimed at enhancing self-determination can help foster the development of healthy body image [153] (p. 490). Additionally, given that females often perceive that males prefer a thinner body type than they typically do [154], health professionals can educate females about actual male preferences [84] via a social norms approach. Health professionals can also tap into the power of social networks by incorporating the influence of family and friends on attitudes and behaviors related to body image and eating concerns. Lastly, training young adult women to serve as positive role models and mentors for younger girls is an intervention strategy that appears to hold promise for decreasing body dissatisfaction and drive for thinness among younger girls [149].

## 1.5 Future Directions

According to Glanz, Rimer, and Viswanath [136] (p. 25), the "task of health behavior and health education is both to understand health behavior and to transform knowledge about behavior into effective strategies for health enhancement." It is important for researchers to continue developing and refining theories and models to guide research and practice in the areas of body image and eating disorder prevention. In sync with this focus, practitioners must be diligent in staying abreast of the literature so they can implement programs that have a sound evidence base.

Because there are various social, cultural, and economic risk factors that contribute to the development of body dissatisfaction and eating disturbances among girls and women from different ethnic groups [32, 55, 84, 87, 89, 155], it is important to continue to study how the impact of

sociocultural and socioeconomic factors on body image and eating may vary across different ethnic and cultural groups [55, 79]. These differences can be studied between as well as within groups to provide a richer knowledge base to inform body image assessment and development of culturally relevant prevention programs that reflect distinctive ethnic and cultural factors [32, 79, 89]. Similarly, there is a need for researchers to examine how age interacts with culture [79] in order to provide a framework for planning and implementing age-appropriate and culturally relevant body image interventions.

It is important to tailor prevention programs to align with various female populations who deal with body image and eating issues, which increases the relevance for program participants and has the potential to promote real, positive behavior change. For instance, there is a need to implement and evaluate prevention interventions that address the negative impact of engaging in body comparison [30]. Future studies also need to examine whether there are ways to enhance the effects of dissonance and healthy weight interventions, such as by using web-based booster sessions and web-based support groups, increasing the number and duration of program sessions, or adding an intervention component targeting parents [150]. Studying how self-determination at the intrapersonal and interpersonal levels of influence can buffer females from the thin ideal and improve body image and eating-related outcomes is a worthwhile line of research as well [153]. Then, too, researchers can contribute to the prevention knowledge base by further examining the use of yoga as a creative intervention strategy to enhance body awareness and prevent disordered eating among women [156]. Virtual reality is another emerging strategy for enhancing body image in patients with eating disorders as well as in high-risk, subclinical samples; therefore, this is an area that warrants further investigation [9]. Regardless of what particular type of body image intervention is implemented, it is important for researchers and practitioners to conduct formative and summative evaluation, including follow-up procedures to determine whether changes in attitudes, knowledge, and behaviors are sustainable over time.

Future directions should also include targeted health communication interventions to promote healthy body image among females. Schiavo [157] (p. 10) comprehensively defined health communication as "a multifaceted and multidisciplinary approach to reach different audiences and share health-related information with the goal of influencing, engaging, and supporting individuals, communities, health professionals, special groups, policymakers and the public to champion, introduce, adopt, or sustain a behavior, practice, or policy that will ultimately improve health outcomes." Although health communication interventions can be multifaceted, it is important to remember that health communication efforts are part of an overall prevention initiative. Therefore, health communication is an effective adjunct to other body image programming. For example, because most teens and young adults are prolific users of technology, health communication campaigns and health promotion interventions aimed at promoting healthy body image among these groups should incorporate social networking, blogging, emailing, and texting. Health communication can be enhanced by developing multimedia campaigns that integrate words, music, and images to produce messaging that resonates with the target group [158]. Furthermore, it is important to pilot health communication campaigns to ensure that the intended audience will both understand the materials and act on their message [159]. This is particularly relevant when targeting individuals who may experience communication and health literacy barriers, such as those who speak English as a second language and those who are d/Deaf and hard-of-hearing. For more information and resources pertaining to health communication, see Schiavo [157].

In addition, robust assessment for body image problems and eating disorders is necessary in order to more accurately detect body image difficulties and disturbances in diverse female populations. Instruments that are valid and reliable to use with a certain group of females must be reevaluated when used with other groups. For example, many body image and eating disorder instruments that have been validated with adult Caucasian females may not be appropriate or

relevant to use with adult females within other ethnic groups. Practitioners must be careful when administering assessments to make sure they are following recommended assessment protocol. There also continues to be an increased use of online instruments for assessing body image disturbances and eating disorders, which will require researchers to validate these assessment tools [96] for use with different female populations.

## 1.6 Concluding Remarks

The complexities inherent in body image are well documented, and body image concerns can range from a desire to look attractive to body dissatisfaction and a pathological concern with thinness or perfection. There are numerous risk factors associated with body image and eating concerns, including biological and physical factors, sociocultural influences, and individual characteristics. Body image concerns affect females of all ages across different ethnic, cultural, and socioeconomic groups; therefore, it is important to examine and address body image difficulties from various angles and at different levels of influence. As a result of these efforts, health professionals will be in better position to foster healthy body image and enhance quality of life among females across the lifespan.

## 1.7 Appendix 1: Body Image Quality of Life Inventory

Different people have different feelings about their physical appearances. These feelings are called "body image." Some people are generally satisfied with their looks, whereas others are dissatisfied. At the same time, people differ in terms of how their body image experiences affect other aspects of their lives. Body image may have positive effects, negative effects, or no effects at all. Listed below are various ways that your own body image may or may not influence your life. For each item, circle how and how much our feelings about you experience affect that aspect of your life. Before answering each item, think carefully about the answer that is most accurate about how your body image usually affects you.

| BIQLI items | −3 Very negative effect | −2 Moderate negative effect | −1 Slight negative effect | 0 No effect | +1 Slight positive effect | +2 Moderate positive effect | +3 Very positive effect |
|---|---|---|---|---|---|---|---|
| 1. My basic feelings about myself—feelings of personal adequacy and self-worth | | | | | | | |
| 2. My feelings about my adequacy as a man or women—feelings of masculinity or femininity | | | | | | | |
| 3. My interactions with people of my own sex | | | | | | | |
| 4. My interactions with people of the other sex | | | | | | | |
| 5. My experiences when I meet new people | | | | | | | |
| 6. My experiences at work or at school | | | | | | | |
| 7. My relationships with friends | | | | | | | |
| 8. My relationships with family members | | | | | | | |
| 9. My day-to-day emotions | | | | | | | |
| 10. My satisfaction with my life in general | | | | | | | |
| 11. My feelings of acceptability as a sexual partner | | | | | | | |
| 12. My enjoyment of my sex life | | | | | | | |
| 13. My ability to control what and how much I eat | | | | | | | |

(continued)

| BIQLI items | −3 Very negative effect | −2 Moderate negative effect | −1 Slight negative effect | 0 No effect | +1 Slight positive effect | +2 Moderate positive effect | +3 Very positive effect |
|---|---|---|---|---|---|---|---|
| 14. My ability to control my weight | | | | | | | |
| 15. My activities for physical exercise | | | | | | | |
| 16. My willingness to do things that might call attention to my appearance | | | | | | | |
| 17. My daily "grooming" activities (i.e., getting dressed and physically ready for the day) | | | | | | | |
| 18. How confident I feel in my everyday life | | | | | | | |
| 19. How happy I feel in my everyday life | | | | | | | |

Reprinted from Int J Eat Disord, 31(4), Cash TF, Fleming EC, Impact of Body Image Experiences: Development of the Body Image Quality of Life Inventory, 455–460, 2002, with permission from John Wiley and Sons

## 1.8 Appendix 2: Body Image Concern Inventory

Please rate how often you have had the described feeling or performed the described behavior on a Likert scale anchored by 1 = "never" and 5 = "always"

| | 1 Never | 2 Seldom | 3 Sometimes | 4 Often | 5 Always |
|---|---|---|---|---|---|
| 1. I am dissatisfied with some aspect of my appearance | | | | | |
| 2. I spend a significant amount of time checking my appearance in the mirror | | | | | |
| 3. I feel others are speaking negatively of my appearance | | | | | |
| 4. I am reluctant to engage in social activities when my appearance does not meet my satisfaction | | | | | |
| 5. I feel there are certain aspects of my appearance that are extremely unattractive | | | | | |
| 6. I buy cosmetic products to try to improve my appearance | | | | | |
| 7. I seek reassurance from others about my appearance | | | | | |
| 8. I feel there are certain aspects of my appearance that I would like to change | | | | | |
| 9. I am ashamed of some part of my body | | | | | |
| 10. I compare my appearance to that of fashion models or others | | | | | |
| 11. I try to camouflage certain flaws in my appearance | | | | | |
| 12. I examine flaws in my appearance | | | | | |
| 13. I have bought clothing to hide a certain aspect of my appearance | | | | | |
| 14. I feel others are more physically attractive than me | | | | | |
| 15. I have considered consulting/consulted some sort of medical expert regarding flaws in my appearance | | | | | |
| 16. I have missed social activities because of my appearance | | | | | |
| 17. I have been embarrassed to leave the house because of my appearance | | | | | |
| 18. I fear that others will discover my flaws in appearance | | | | | |
| 19. I have avoided looking at my appearance in the mirror | | | | | |

Reprinted from Behav Res Ther, 43(2), Littleton H, Axsom D, Pury CLS, Development of the Body Image Concern Inventory, 229–241, 2005, with permission from Elsevier

## 1.9 Appendix 3: Physical Appearance State and Trait Anxiety Scale: Trait

The statements listed below are to be used to describe how anxious, tense, or nervous you feel in general (i.e., usually) about your body or specific parts of your body.

Please read each statement and circle the number that best indicates the extent to which each statement holds true in general. Remember, there are no right or wrong answers.

| Never | Seldom | Sometimes | Often | Always |
|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 |

In general I feel *anxious*, *tense*, *or nervous* about

| | | | | | |
|---|---|---|---|---|---|
| 1. The extent to which I look overweight | 1 | 2 | 3 | 4 | 5 |
| 2. My thighs | 1 | 2 | 3 | 4 | 5 |
| 3. My buttocks | 1 | 2 | 3 | 4 | 5 |
| 4. My hips | 1 | 2 | 3 | 4 | 5 |
| 5. My stomach | 1 | 2 | 3 | 4 | 5 |
| 6. My legs | 1 | 2 | 3 | 4 | 5 |
| 7. My waist | 1 | 2 | 3 | 4 | 5 |
| 8. My muscle tone | 1 | 2 | 3 | 4 | 5 |
| 9. My ears | 1 | 2 | 3 | 4 | 5 |
| 10. My lips | 1 | 2 | 3 | 4 | 5 |
| 11. My wrists | 1 | 2 | 3 | 4 | 5 |
| 12. My hands | 1 | 2 | 3 | 4 | 5 |
| 13. My forehead | 1 | 2 | 3 | 4 | 5 |
| 14. My neck | 1 | 2 | 3 | 4 | 5 |
| 15. My chin | 1 | 2 | 3 | 4 | 5 |
| 16. My feet | 1 | 2 | 3 | 4 | 5 |

Reprinted from J Anxiety Disord, 5(4), Reed, DL, Thompson, JK, Brannick, MT., Sacco WP, Development and Validation of the Physical Appearance State and Trait Anxiety Scale (PASTAS), 323–332, 1991, with permission from Elsevier

## References


1. Cash TF. Body image: past, present, and future. Body Image. 2004;1(1):1–5.
2. Smolak L, Thompson JK. Body image, eating disorders, and obesity in children and adolescents: an introduction to the second edition. In: Smolak L, Thompson JK, editors. Body image, eating disorders, and obesity in youth: assessment, prevention, and treatment. 2nd ed. Washington, DC: American Psychological Association; 2009. p. 3–14.
3. Thompson J, Heinberg L, Altabe M, Tantleff-Dunn S. Exacting beauty: theory, assessment, and treatment of body image disturbance. Washington, DC: American Psychological Association; 1999.
4. Bruchon-Schweitzer M. Psicología del cuerpo. Barcelona: Herder; 1992.
5. Erickson SJ, Gerstle M. Investigation of ethnic differences in body image between Hispanic/biethnic Hispanic and non-Hispanic White preadolescent girls. Body Image. 2007;4:69–78. doi:10.1016/j.bodyim.2006.11.003.
6. Slevec JH, Tiggemann M. Predictors of body dissatisfaction and disordered eating in middle-aged women. Clin Psychol Rev. 2011;31:515–24. doi:10.1016/j.cpr.2010.12.002.
7. Bearman SK, Presnell K, Martinez E, Stice E. The skinny on body dissatisfaction: a longitudinal study of adolescent girls and boys. J Youth Adolesc. 2006;35(2):217–29. doi:10.1007/s10964-005-9010-9.
8. Wade T, George WM, Atkinson M. A randomized controlled trial of brief interventions for body dissatisfaction. J Consult Clin Psychol. 2009;77:845–54. doi:10.1037/a0016879.
9. Ferrer-García M, Gutiérrez-Maldonado J. The use of virtual reality in the study, assessment, and treatment of body image and eating disorders and nonclinical samples: a review of the literature. Body Image. 2012;1:1–11. doi:10.1016/j.bodyim.2011.10.001.
10. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed. Arlington, VA: Author; 2000. text revision.
11. Rodin J, Silberstein L, Streigel-Moore R. Women and weight: a normative discontent. In: Sondregger TB, editor. Psychol and gender: nebraska symposium on motivation. Lincoln, NE: University of Nebraska Press; 1985. p. 206–37.
12. Stice E, Schupak-Neuberg E, Shaw HE, Stein R. Risk and maintenance factors for eating pathology: a meta-analytic review. Psychol Bull. 2002;128:825–48.
13. Johnson F, Wardle J. Dietary restraint, body dissatisfaction, and psychological distress: a prospective analysis. J Abnorm Psychol. 2005;114:119–25.
14. Rodgers RF, Salés P, Chabrol H. Psychological functioning, media pressure and body dissatisfaction among college women. Eur Rev Appl Psychol. 2010;60(2):89–95. doi:10.1016/j.erap.2009.10.001.
15. Stice E, Hayward C, Cameron RP, et al. Body-image and eating disturbances predict onset of depression among female adolescents: a longitudinal study. J Abnorm Psychol. 2000;3:438–44.
16. Wertheim EH, Paxton SJ, Blaney S. Body image in girls. In: Smolak L, Thompson JK, editors. Body image, eating disorders, and obesity in youth: assessment, prevention, and treatment. 2nd ed. Washington, DC: American Psychological Association; 2009. p. 47–76.
17. Phillips KA, Menard W, Fay C. Gender similarities and differences in 200 individuals with body dysmorphic disorder. Compr Psychiatry. 2006;47:77–87.

18. Murray CD, Macdonald S, Fox J. Body dissatisfaction, eating disorders and suicide ideation in an Internet sample of self-harmers reporting and not reporting childhood sexual abuse. Psychol Health Med. 2008;13(1):29–42. doi:10.1080/13548500701235757.
19. Izgic F, Akyuz G, Dogan O, Kugu N. Social phobia among university students and its relation to self-esteem and body image. Can J Psychiatry. 2004;49(9):630–4.
20. Field AE, Kitos N. Eating and weight concerns in eating disorders. In: Agras WS, editor. The oxford handbook of eating disorders. New York, NY: Oxford University Press; 2010. p. 206–22.
21. Abraczinskas M, FisakJr B, Barnes RD. The relation between parental influence, body image, and eating behaviors in a nonclinical female sample. Body Image. 2012;9:93–100. doi:10.1016/j.bodyim.2011.10.005.
22. Chen H, Jackson T. Gender and age group differences in mass media and interpersonal influences on body dissatisfaction among Chinese adolescents. Sex Roles. 2012;66:3–20. doi:10.1007/s11199-011-0056-8.
23. Clark L, Tiggemann M. Appearance culture in nine- to 12-year-old girls: media and peer influences on body dissatisfaction. Soc Dev. 2006;15:628–43.
24. Jones DC, Vigfusdottir TH, Lee Y. Body image and the appearance culture among adolescent girls and boys: an examination of friend conversations, peer criticism, appearance magazines, and the internalization of appearance ideals. J Adol Res. 2004;19:323–39.
25. van den Berg P, Thompson JK, Obremski-Brandon K, Coovert M. The tripartite influence model of body image and eating disturbance—a covariance structure modelling investigation testing the mediational role of appearance comparison. J Psychosom Res. 2002;53:1007–20.
26. Ayala GX, Mickens L, Galindo P, Elder JP. Acculturation and body image perception among Latino youth. Ethn Health. 2007;12:21–41. doi:10.1080/13557850600824294.
27. Cheney AM. "Most girls want to be skinny": body (dis)satisfaction among ethnically diverse women. Qual Health Res. 2011;21(10):1347–59. doi:10.1177/1049732310392592.
28. Carey RN, Donaghue N, Broderick P. 'What you look like is such a big factor': girls' own reflections about the appearance culture in an all-girls' school. Feminism Psychol. 2011;21(3):299–316. doi:10.1177/0959353510369893.
29. Tiggemann H, Dohnt M. The contribution of peer and media influences to the development of body dissatisfaction and self-esteem in young girls: a prospective study. Dev Psychol. 2006;42(5):929–36.
30. Durkin SJ, Paxton SJ, Sorbello M. An integrative model of the impact of exposure to idealized female images on adolescent girls' body satisfaction. J Appl Soc Psychol. 2007;35(5):1092–117.
31. Ricciardelli LA, McCabe MP, Holt KE, Finemore J. A biopsychosocial model for understanding body image and body change strategies among children. J Appl Dev Psychol. 2003;24:475–95.
32. Yamamiya Y, Shroff H, Thompson JK. The Tripartite influence model of body image and eating disturbance: a replication with a Japanese sample. Int J Eat Disord. 2008;41(1):88–91. doi:10.1002/eat.20444.
33. Rideout VJ, Foehr UG, Roberts DF. Generation M2: media in the lives of 8- to 18-year-olds. The Henry J. Kaiser Family Foundation. 2010. http://www.kff.org/entmedia/upload/8010.pdf. Accessed 22 Jun, 2012.
34. Gutnick AL, Robb M, Takeuchi L, Kotler J. Always connected: the new digital media habits of young children. 2010. New York: The Joan Ganz Cooney Center at Sesame Workshop. http://www.ictliteracy.info/rf.pdf/jgcc_alwaysconnected.pdf. Accessed 22 Jun, 2012.
35. Anschutz DJ, Spruijt-Metz D, Van Strien T, Engels RCME. The direct effect of thin ideal focused adult television on young girls' ideal body. Body Image. 2011;8:26–33.
36. Lawrie Z, Sullivan EA, Davies PS, Hill RJ. Body change strategies in children: relationship to age and gender. Eat Disord. 2007;8:357–63.
37. Warren C, Schoen A, Shafer K. Media internalization and social comparison as predictors of eating pathology among Latino adolescents: the moderating effect of gender and generational status. Sex Roles. 2010;63:712–24. doi:10.1007/s1199-010-9876-1.
38. Dohnt HK, Tiggeman M. Body image concerns in young girls: the role of peers and the media prior to adolescence. J Youth Adol. 2006;35:135–45.
39. Grabe S, Ward ML, Hyde JS. The role of the media in body image concerns among women: a meta-analysis of experimental and correlational studies. Psychol Bull. 2008;134:460–76. doi:10.1037/0033-2909.134.3.460.
40. Groesz LM, Levine MP, Murnen SK. The effect of experimental presentation of thin media images on body satisfaction: a meta-analytic review. Int J Eat Disord. 2002;31:1–16. doi:10.1002/eat.10005.
41. Bell BT, Dittmar H. Does media type matter? The role of identification in adolescent girls' media consumption and the impact of different thin-ideal media on body image. Sex Roles. 2011;65:478–90. doi:10.1007/s11199-011-9964-x.
42. Gondoli DM, Corning AF, Blodgett Salafia EH, Bucchianeri MM, Fitzsimmons EE. Heterosocial involvement, peer pressure for thinness, and body dissatisfaction among young adolescent girls. Body Image. 2011;8(2):143–8. doi:10.1016/j.bodyim.2010.12.005.
43. Sands ER, Wardle J. Internalization of ideal body shapes in 9–12-year old girls. Int J Eat Disord. 2003;33:193–204.
44. Dittmar H, Halliwell E, Ive S. Does Barbie make girls want to be thin? The effect of experimental exposure to images of dolls on the body image of 5- to 8-year-old girls. Dev Psychol. 2006;42(2):283–92.

45. Thompson JK, Stice E. Thin-ideal internalization: mounting evidence for a new risk factor for body-image disturbance and eating pathology. Curr Dir Psychol Sci. 2001;10:181–3.
46. Murnen SK, Smolak L, Mills JA, Good L. Thin, sexy women, and strong, muscular men: grade-school children's responses to objectified images of men and women. Sex Roles. 2003;49:427–37.
47. Jones DC, Crawford JK. The peer appearance culture during adolescence: gender and body mass variations. J Youth Adol. 2006;35:257–69.
48. Eisenberg ME, Neumark-Sztainer D. Friends' dieting and disordered eating behaviors among adolescents five years later: findings from Project EAT. J Adol Health. 2010;47:67–73. doi:10.1016/j.jadohealth.2009.12.030.
49. Luce KH, Crowther JH, Pole M. Eating disorder examination questionnaire (EDE-Q): norms for undergraduate women. Int J Eat Disord. 2008; 41:273–6.
50. American College Health Association. National college health assessment: reference group data report. Linthicum, MD: Author; 2010.
51. Berg KC, Frazier P, Sherr L. Change in eating disorder attitudes and behavior in college women: prevalence and predictors. Eat Behav. 2009;10:137–42. doi:10.1016/j.eatbeh.2009.03.003.
52. Neighbors LA, Sobal J. Prevalence and magnitude of body weight and shape dissatisfaction among university students. Eat Behav. 2007;8:429–39.
53. Vohs KD, Heatherton TF, Herrin M. Disordered eating and the transition to college: a prospective study. Int J Eat Disord. 2001;29:280–8.
54. Gilbert N, Meyer C. Fear of negative evaluation and eating attitudes: a replication and extension study. Int J Eat Disord. 2005;37:360–3.
55. Rodgers R, Chabrol H, Paxton SJ. An exploration of the tripartite influence model of body dissatisfaction and disordered eating among Australian and French college women. Body Image. 2011;8:208–15. doi:10.1016/j.bodyim.2011.04.009.
56. Dittmar H, Howard S. Thin-ideal internalization and social comparison tendency as moderators of media models' impact on women's body-focused anxiety. J Soc Clin Psychol. 2004;23(6):768–91. doi:10.1521/jscp.23.6.768.54799.
57. Fitzsimmons-Craft EE, Harney MB, Koehler LG, Danzi LE, Riddell MK, Bardone-Cone AM. Explaining the relation between thin ideal internalization and body dissatisfaction among college women: the roles of social comparison and body surveillance. Body Image. 2012;9:43–9. doi:10.1016/j.bodyim.2011.09.002.
58. Nichter M, Vuckovic N. Fat talk. In: Sault N, editor. Many mirrors: body image and social relations. New Brunswick, NJ: Rutgers University Press; 1994. p. 109–31.
59. Salk RH, Engeln-Maddox R. "If you're fat, then I'm humongous!": frequency, content, and impact of fat talk among college women. Psychol Womens Q. 2011;35(1):18–28. doi:10.1177/0361684310384107.
60. Ousley L, Cordero ED, White S. Fat talk among college students: how undergraduates communicate regarding food and body weight, shape, and appearance. Eat Disord. 2008;16:73–84.
61. Liechty T. "Yes, I worry about my weight…but for the most part I'm content with my body": older women's body dissatisfaction alongside contentment. J Women Aging. 2012;24:70–88. doi:10.1080/08952841.2012.638873.
62. Liechty T, Yarnal CM. Older women's body image: a lifecourse perspective. Ageing Soc. 2010;30:1197–218. doi:10.1017/S0144686X10000346.
63. Tiggemann M. Body image across the adult life span: stability and change. Body Image. 2004;1(1): 29–41.
64. Webster J, Tiggemann M. The relationship between women's body satisfaction and self-image across the life span: the role of cognitive control. J Genet Psychol. 2003;164(2):241–52.
65. Baker L, Gringart E. Body image and self-esteem in older adulthood. Ageing Soc. 2009;29:977–95. doi:10.1017/S0144686X09008721.
66. Pruis TA, Janowsky JS. Assessment of body image in younger and older women. J Gen Psychol. 2010;137(3):225–38.
67. The Renfrew Center. Midlife. http://renfrewcenter.com/services/treatment-tracks/midlife. Accessed 10 Aug, 2012.
68. Gagne DA, Von Holle A, Brownley KA, Runfola CD, Hofmeier S, Branch KE, Bulik CM. Eating disorder symptoms and weight and shape concerns in a large web-based convenience sample of women ages 50 and above: results of the gender and body image (GABI) study. Int J Eat Disord. 2012;45:832–44. doi:10.1002/eat.22030.
69. McLean SA, Paxton SJ, Wertheim EH. A body image and disordered eating intervention for women in midlife: a randomized controlled trial. J Consult Clin Psychol. 2011;79(6):751–8. doi:10.1037/a0026094.
70. Hatch LR. Beyond gender difference: adaptation to ageing in the life-course perspective. Amityville, NY: Baywood; 2000.
71. Jones JM, Lawson ML, Daneman D, Olmsted MP, Rodin G. Eating disorders in adolescent females with and without type 1 diabetes: cross sectional study. *BMJ*. 2000;320:1563–1566. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC27398/pdf/1563.pdf. Accessed 8 Feb, 2013.
72. Goebel-Fabbri AE. Diabetes and eating disorders. J Diabetes Sci Technol. 2008;2(3):530–2.
73. Howe CJ, Jawad AJ, Kelly SD, Lipman TH. Weight-related concerns and behaviors in children and adolescents with type 1 diabetes. J Am Psychiatr Nurses Assoc. 2008;13:376–85.
74. Kichler JC, Foster C, Opipari-Arrigan L. The relationship between negative communication and body image

dissatisfaction in adolescent females with Type 1 Diabetes Mellitus. J Health Psychol. 2008;13(3): 336–47. doi:10.1177/1359105307088138.
75. Anderson-Fye E. Cross-cultural issues in body image among children and adolescents. In: Smolak L, Thompson JK, editors. Body image, eating disorders, and obesity in youth: assessment, prevention, and treatment. 2nd ed. Washington, DC: American Psychological Association; 2009. p. 113–33.
76. Chen H, Jackson T. Predictors of changes in weight esteem among mainland Chinese adolescents: a longitudinal analysis. Dev Psychol. 2009;45(6):1618–29.
77. Crawford J, Lee I-C, Portnoy G, Gurung A, Khati D, Jha P, Regmi AC. Objectified body consciousness in a developing country: a comparison of mothers and daughters in the US and Nepal. Sex Roles. 2009;60:174–85. doi:10.1007/s11199-008-9521-4.
78. Frederick DA, Forbes GB, Berezovskaya A. Female body dissatisfaction and perceptions of the attractive female body in Ghana, the Ukraine, and the United States. Psychol Top. 2008;17(2):203–19.
79. Holmqvist K, Frisén A. Body dissatisfaction across cultures: findings and research problems. Eur Eat Disord Rev. 2010;18:133–46. doi:10.1002/erv.965.
80. Levine MP, Smolak L. Cultural influences on body image and the eating disorders. In: Agras WS, editor. The oxford handbook of eating disorders. New York, NY: Oxford University Press; 2010. p. 223–46.
81. Swami V, et al. The attractive female body weight and female body dissatisfaction in 26 countries across 10 world regions: results of the International Body Project I. Pers Soc Psychol Bull. 2010;36(3):309–25. doi:10.1177/ 0146167209359702.
82. McHugh TLF, Kowalski KC. 'A new view of body image': a school-based participatory action research project with young Aboriginal women. Action Res. 2011;9(3):220–41.doi:10.1177/1476750310388052.
83. The Levin Institute – The State University of New York. Globalization and eating disorders. http://www.globalization101.org/globalization-and-eating-disorders-2/. Accessed 8 Jul, 2012.
84. Gillen MM, Lefkowitz ES. Body size perceptions in racially/ethnically diverse men and women: implications for body image and self-esteem. N Am J Psychol. 2011;13(3):447–68.
85. Grabe S, Hyde JS. Ethnicity and body dissatisfaction among women in the United States: a meta-analysis. Psychol Bull. 2006;132(4):622–40. doi:10.1037/ 0033-2909.132.4.622.
86. Hesse-Biber S, Livingstone S, Ramirez D, Barko EB, Johnson AL. Racial identity and body image among Black female college students attending predominately White colleges. Sex Roles. 2010;63:697–711. doi:10.1007/s11199-010-9862-7.
87. Sabik NJ, Cole ER, Ward LM. Are all minority women equally buffered from negative body image? intra-ethnic moderators of the buffering hypothesis. Psychol Womens Q. 2010;34:139–51.
88. Sussman NM, Truong N, Lim J. Who experiences "American the beautiful"?: ethnicity moderating the effect of acculturation on body image and risks for eating disorders among immigrant women. Int J Intercult Relat. 2007;31:29–49.
89. Franko DL, Edwards George JB. Overweight, eating behaviors, and body image in ethnically diverse youth. In: Smolak L, Thompson JK, editors. Body image, eating disorders, and obesity in youth: assessment, prevention, and treatment. 2nd ed. Washington, DC: American Psychological Association; 2009. p. 97–112.
90. Anderson DA, Murray AD. Psychological assessment of the eating disorders. In: Agras WS, editor. The oxford handbook of eating disorders. New York, NY: Oxford University Press; 2010. p. 249–58.
91. Thompson JK. The (mis)measurement of body image: ten strategies to improve assessment for applied and research purposes. Body Image. 2004;1:7–14. doi:10.1016/S1740-1445(03)00004-4.
92. Banasiak SJ, Wertheim EH, Koerner J, Voudouris NJ. Test–retest reliability and internal consistency of a variety of measures of dietary restraint and body concerns in a sample of adolescent girls. Int J Eat Disord. 2001;29:85–9.
93. Reed DL, Thompson KJ, Brannick MT, Sacco WP. Development and validation of the Physical Appearance State and Trait Anxiety Scale (PASTAS). J Anxiety Disord. 1991;54:323–32. doi:10.1016/0887-6185(91)90032-O.
94. Cash TF, Fleming EC, Alindogan LS, Whitehead A. Beyond body image as a trait: the development and validation of the Body Image States Scale. Eat Disord. 2002;10:103–13. doi:10.1080/1064026029 0081678.
95. Bell BT, Lawton R, Dittmar H. The impact of thin models in music videos on adolescent girls' body dissatisfaction. Body Image. 2007;4:137–45. doi:10.1016/j.bodyim.2007.02.003.
96. Túry F, Güleç H, Kohls E. Assessment methods for eating disorders and body image disorders. J Psychosom Res. 2010;60:601–11. doi:10.1016/j.jpsychores.2009.05.012.
97. Allison DB. Handbook of assessment methods for eating behaviors and weight-related problems. 2nd ed. Newbury Park, CA: Sage; 2009.
98. Cash TF. The body image workbook: an eight-step program for learning to life your looks. 2nd ed. Oakland, CA: New Harbinger Publications; 2008.
99. Thompson JK, van den Berg P. Measuring body image attitudes among adolescents and adults. In: Cash TG, Pruzinsky T, editors. Body image: a handbook of theory, research and clinical practice. New York, NY: Guilford; 2002. p. 142–53.
100. Yanover T, Thompson JK. Assessment of body image in children and adolescents. In: Smolak L, Thompson JK, editors. Body image, eating disorders, and obesity in youth: assessment, prevention, and

treatment. 2nd ed. Washington, DC: American Psychological Association; 2009. p. 177–92.
101. Cash TF, Fleming EC. The impact of body-image experiences: development of the Body Image Quality of Life Inventory. Int J Eat Disord. 2002; 31:455–60.
102. Cash TF, Szymanski ML. The development and validation of the Body-Image Ideals Questionnaire. J Pers Assess. 1995;64:466–77.
103. Garner D. Manual for the eating disorder inventory-2 (EDI-2). Odessa, FL: Psychological Assessment Resources; 1991.
104. Garner D, Olmsted M. Manual for the eating disorder inventory-2 (EDI-2). Odessa, FL: Psychological Assessment Resources; 1984.
105. Littleton HL, Axsom D, Pury CLS. Development of the body image concern inventory. Behav Res Ther. 2005;43(2):229–41. doi:10.1016/j.brat.2003.12.006.
106. Shisslack CM, Renger R, Sharpe T, et al. Development and evaluation of the McKnight Risk Factor Survey for assessing potential risk and protective factors for disordered eating in preadolescent and adolescent girls. Int J Eat Disord. 1999;25:195–214.
107. Wooley OW, Roll S. The color-a-person body dissatisfaction test: stability, internal consistency, validity, and factor structure. J Pers Assess. 1991;56:395–413.
108. Cash TF, Grasso K. The norms and stability of new measures of the multidimensional body image construct. Body Image. 2005;2:199–203. doi:10.1016/j.bodyim.2005.03.007.
109. Cash TF, Phillips KA, Santos MT, Hrabosky JI. Measuring "negative body image": validation of the body image disturbance questionnaire in a nonclinical population. Body Image. 2004;1:363–72.
110. Cash TF, Melnyk S, Hrabosky JI. The assessment of body-image investment: An extensive revision of the Appearance Schemas Inventory. Int J Eat Disord. 2004;35:305–16.
111. Cash TF, Santos MT, Williams EF. Coping with body-image threats and challenges: validation of the Body Image Coping Strategies Inventory. J Psychosom Res. 2005;58:191–9.
112. Cash TF, Jakatdar T, Williams EF. The body image quality of life inventory: further validation with college men and women. Body Image. 2004;1:279–87.
113. Gardner RM, Jappe LM, Gardner L. Development and validation of a new figural drawing scale for body-image assessment: the BIAS-BD. J Clin Psychol. 2009;65(1):113–22.
114. Cooper PJ, Taylor MJ, Cooper Z, Fairburn CG. The development and validation of the Body Shape Questionnaire. Int J Eat Disord. 1987;6:485–94.
115. Dowson J, Henderson L. The validity of a short version of the Body Shape Questionnaire. Psychiatry Res. 2001;102:263–71.
116. Warren CS, Cepeda-Benito A, Gleaves DH, et al. English and Spanish versions of the Body Shape Questionnaire: measurement equivalence across ethnicity and clinical status. Int J Eat Disord. 2008;41:265–72.
117. Gardner DM. Eating disorder inventory™ (EDI™-3). Lutz, FL: PAR; 2004.
118. Thompson JK, van den Berg P, Roehrig M, et al. The Sociocultural attitudes towards appearance scale-3 (SATAQ-3): development and validation. Int J Eat Disord. 2004;35(3):293–304. doi:10.1002/eat.10257.
119. Truby H, Paxton SJ. Development of the children's body image scale. Br J Clin Psychol. 2002;41: 184–203.
120. Truby H, Paxton SJ. The children's body image scale: reliability and use with international standards for body mass index. Br J Clin Psychol. 2008;47: 119–24. doi:10.1348/014466507X251261.
121. Domina T, Heuberger R, Macgillivray M. Use of 3-dimensional body scans for body-image research. Percept Mot Skills. 2008;106:653–8.
122. Aleong R, Duchesne S, Paus T. Assessment of adolescent body perception: development and characterization of a novel tool for morphing images of adolescent bodies. Behav Res Meth. 2007;39(3):651–66.
123. Piran N. Prevention of eating disorders: a review of outcome evaluation research. Isr J Psychiatry Relat Sci. 2005;42(3):172–7.
124. Sinton MM, Taylor CB. Prevention: current status and underlying theory. In: Agras WS, editor. The oxford handbook of eating disorders. New York, NY: Oxford University Press; 2010. p. 307–30.
125. Massey-Stokes M, Barton BA, Golman M, Holland DJ. An ecological approach to the prevention of eating disorders in children and adolescents. In: Goodheart K, Clopton J, Robert-Mccomb J, editors. Eating disorders in women and children: prevention, stress management, and treatment. 2nd ed. Boca Raton, FL: CRC Press; 2012. p. 225–67.
126. Massey-Stokes M, Stokes SB. Social-emotional learning, interpersonal skills, and resilience. In: Goodheart K, Clopton J, Robert-Mccomb J, editors. Eating disorders in women and children: prevention, stress management, and treatment. 2nd ed. Boca Raton, FL: CRC Press; 2012. p. 283–311.
127. O'Connell ME, Boat T, Warner KE, eds. *Preventing mental*, *emotional*, *and behavioral disorders among young people*: *progress and possibilities*. Washington, DC: The National Academies Press; 2009. http://www.ncbi.nlm.nih.gov/books/NBK32775/ pdf/TOC.pdf. Accessed 19 Jul, 2012.
128. Neumark-Sztainer D. Addressing the spectrum of adolescent weight-related problems: engaging parents and communities. Prev Res. 2007;14:11–4.
129. O'Dea J. Everybody's different: a positive approach to teaching about health, puberty, body image, nutrition, self-esteem and obesity prevention. Camberwell, VIC: ACER Press; 2007.
130. Shaw H, Ng J, Stice E. Integrating eating disorder and obesity prevention programs for adolescents. Prev Res. 2007;14:18–20.
131. Stock SC, Evans MS, Plessis S, et al. Healthy buddies: a novel, peer-led health promotion program for the prevention of obesity and eating disorders in children in elementary school. Pediatrics. 2007;120: e1059–68.

132. Neumark-Sztainer D, Levine MP, Paxton SJ, et al. Prevention of body dissatisfaction and disordered eating: what next? Eat Disord. 2006;14:265–85.
133. Massey-Stokes M. Body image and eating disturbances in children and adolescents. In: Robert-McComb JJ, Norman R, Zumwalt M, editors. The active female. Totowa, NJ: Humana Press; 2008. p. 57–79.
134. Stice E, Shaw H, Marti CN. A meta-analytic review of eating disorder prevention programs: encouraging findings. Annu Rev Clin Psychol. 2007;3:207–31.
135. Levine M, Smolak L. Recent developments and promising directions in the prevention of negative body image and disordered eating children and adolescents. In: Smolak L, Thompson JK, editors. Body image, eating disorders, and obesity in youth: assessment, prevention, and treatment. 2nd ed. Washington, DC: American Psychological Association; 2009. p. 215–39.
136. Glanz K, Rimer BK, Viswanath K, editors. Health education and health behavior: theory, research, and practice. 4th ed. San Francisco, CA: Jossey-Bass; 2008.
137. Sallis JF, Owen N, Fisher EB. Ecological models of health behavior. In: Glanz K, Rimer BK, Viswanath K, editors. Health education and health behavior: theory, research, and practice. 4th ed. San Francisco, CA: Jossey-Bass; 2008. p. 465–85.
138. Bandura A. Social foundations of thought and action: a social cognitive theory. Englewood Cliffs, NJ: Prentice Hall; 1986.
139. Andreasen A. Social marketing: definition and domain. J Publ Pol Market. 1993;13:108–14.
140. McAlister AL, Perry CL, Parcel GS. How individuals, environments, and health behaviors interact: social cognitive theory. In: Glanz K, Rimer BK, Viswanath K, editors. Health education and health behavior: theory, research, and practice. 4th ed. San Francisco, CA: Jossey-Bass; 2008. p. 169–88.
141. Storey JD, Saffitz GB, Rimón JG. Social marketing. In: Glanz K, Rimer BK, Viswanath K, editors. Health education and health behavior: theory, research, and practice. 4th ed. San Francisco, CA: Jossey-Bass; 2008. p. 435–64.
142. Kincaid DL. From innovation to social norm: bounded normative influence. J Health Com. 2004;91(1):37–57.
143. Bauer KW, Haines J, Neumark-Sztainer N. Obesity prevention: strategies to improve effectiveness and reduce harm. In: Smolak L, Thompson JK, editors. Body image, eating disorders, and obesity in youth: assessment, prevention, and treatment. 2nd ed. Washington, DC: American Psychological Association; 2009. p. 241–60.
144. Black MM, Young-Hyman D. Introduction to the special issue: pediatric overweight. J Pediatr Psychol. 2007;32(1):1–5. doi:10.1093/jpepsy/jsl042.
145. Neumark-Sztainer D, Croll J, Story M, Hannan PJ, French SA, Perry C. Ethnic/racial differences in weight-related concerns and behaviors among adolescent girls and boys: findings from project EAT. J Psychosom Res. 2002;53:963–74.
146. Feinberg-Walker HS, Barrett S, Shure J. Inside/outside self-discovery for teens: strategies to promote resilience, relationships, and positive body image. Santa Cruz, CA: ToucanEd; 2009.
147. Kater K. Healthy body image: teaching kids to eat and love their bodies too! 2nd ed. Seattle, WA: National Eating Disorders Association; 2005.
148. Escoto Ponce de León MC, Mancilla Díaz JM, Camacho Ruiz EJ. A pilot study of the clinical and statistical significance of a program to reduce eating disorder risk factors in children. Eat Weight Disord. 2008;13(3):111–8.
149. Golman M. A study to evaluate the effectiveness of the Girls in Motion program in improving body satisfaction in preadolescent girls, Ph.D. dissertation, Texas Woman's University, 2009.
150. Stice E, Shaw H, Becker CB, Rohde P. Dissonance-based interventions for the prevention of eating disorders: using persuasion principles to promote health. Prevent Sci. 2008;9:114–28.
151. McVey GL, Lieberman M, Voorberg N, Wardrope D, Blackmore E. School-based peer support groups: a new approach to the prevention of disordered eating. Eat Disord. 2003;11:169–85. doi:10.1080/10640260390218297.
152. Steese S, Dollette M, Phillips W, Hossfeld E, Matthews G, Taormina G. Understanding Girls' Circle as an intervention on perceived social support, body image, self-efficacy, locus of control, and self-esteem. Adolescence. 2006;41:55–74.
153. Mask L, Blanchard CM. The protective role of general self-determination against 'thin ideal' media exposure on women's body image and eating-related concerns. J Health Psychol. 2010;16(3):489–99. doi:10.1177/1359105310385367.
154. Forbes G, Adams-Curtis LE, Rade B, Jaberg P. Body dissatisfaction in women and men: the role of gender-typing and self-esteem. Sex Roles. 2001;44:461–84.
155. Viladrich A, Yeh M-C, Bruning N, Weiss R. "Do real women have curves?" paradoxical body images among Latinas in New York City. J Immigrant Minority Health. 2009;11:20–28. doi:10/1007/s10903-008-9176-9.
156. Daubenmeier JJ. The relationship of yoga, body awareness, and body responsiveness to self-objectification and disordered eating. Psychol Womens Q. 2005;29:207–19.
157. Schiavo R. Health communication: from theory to practice. San Francisco, CA: John Wiley & Sons; 2007.
158. Centers for Disease Control and Prevention. Audience insights: communicating to teens (aged 12-17). http://www.cdc.gov/healthcommunication/Audience/AudienceInsight_teens.pdf. Accessed 1 Feb, 2013.
159. Ammary-Risch NJ, Zambon A, McCormack BK. Communicating health information effectively. In: Fertman CI, Allensworth DD, editors. Health promotion programs: from theory to practice. San Francisco, CA: Jossey-Bass; 2010. p. 203–31.

# Reproductive Changes in the Female Lifespan

2

Reid L. Norman

**Abstract**

During the lifespan of women, there are dramatic and life-changing transitions associated with the beginning and cessation of reproductive function. These transitions, puberty and menopause, result in dramatic changes in the anatomy, physiology, and cognitive function of females and are caused by fluctuating levels of estrogen and progesterone. Regular menstruation is the result of cyclic release of reproductive hormones and is a sign that all is going well. When a woman approaches menopause, menstruation usually becomes more irregular and eventually ceases because hormones from the ovary are too low to stimulate the lining of the uterus. Because the life expectancy for women is now approaching 80 years and menopause occurs at about 50 years of age, the average female will live approximately 30 years after her ovaries have ceased to produce estrogen. This has serious physical and mental health implications in the aging population, which will increase dramatically in the next few decades. Hormone replacement therapy relieves some of the uncomfortable aspects of menopause, such as hot flashes and sleep disturbances, and protects against osteoporosis. However, recent evidence suggests there may be an increased risk of certain cancers and stroke with hormone therapy. Additional research is needed to unravel the complex actions of hormone replacement formulations so that women can have a healthier life after menopause.

**Keywords**

Puberty • Menarche • Menstruation • Osteoporosis • Menopause • Hormone replacement therapy • Alzheimer's

R.L. Norman, Ph.D. (✉)
Department of Pharmacology and Neuroscience,
Texas Tech University Health Sciences Center School
of Medicine, Lubbock, TX, USA
e-mail: reid.normna@ttuhsc.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_2, 

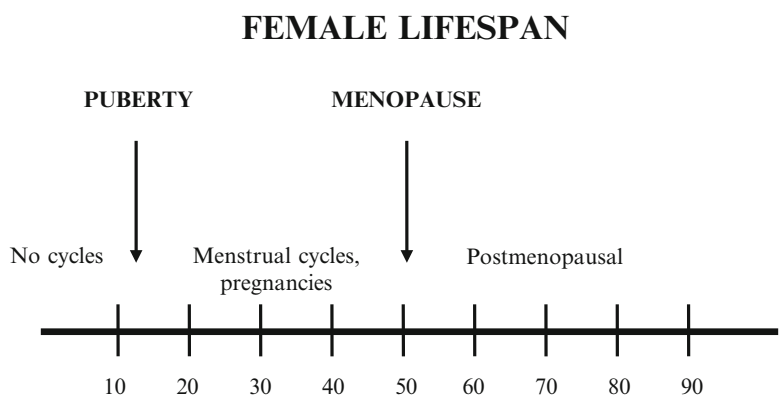



**Fig. 2.1** Graphic depiction of the female reproductive capabilities across the lifespan of the average woman

## 2.1 Learning Objectives

After completing this chapter, you should have an understanding of:

- The major transitions during a woman's life
- How reproductive hormones change throughout the lifespan of a woman
- The terminology used to describe changes in reproductive capabilities
- The impact of menopause on health measures

## 2.2 Introduction

When considering reproductive competence in the human female, the average lifespan can be divided into three phases. The first of these is childhood where, except in rare instances of precocious puberty, the reproductive system is quiescent and secondary sexual characteristics are absent. Puberty is the transition between childhood and sexual maturity, and menarche or first menstruation is an important sign of this transition. During this second stage of sexual maturity, which in the United States lasts from about 13 years to 51 years of age, menstrual cycles occur about once a month except when interrupted by pregnancy or by synthetic steroids used as birth control. Even when hormonal birth control is used, there is usually an attempt to maintain monthly cyclic menstruation. In modern societies, menstrual cycles can be suppressed by stressful life events or by lack of nutritional resources, the latter usually due to dieting or exercise. Most women stop having menstrual cycles in their late forties or early fifties and enter the postmenopausal stage. This transition between the fertile period and postmenopausal or infertile phase is called menopause (Fig. 2.1). In the past century, the time spent in the postmenopausal state has become extended because the average lifespan has increased from 50 to over 80 years. The health concerns of postmenopausal women have become a real concern in the past 50 years. The transitions between these two stages, puberty and menopause, are accompanied by dramatic changes in levels of the most important female hormones, estrogen and progesterone.

## 2.3 Research Findings

### 2.3.1 Hormonal Changes During Childhood

From shortly after birth until the beginning of sexual maturation, reproductive hormone levels are low, and available energy is largely committed to growth and development. Before puberty, boys and girls have similar lean body mass and the same amount of body fat. Body mass index in girls is generally between 15 and 18 and does not change much during the childhood years. Growth rates are similar for boys and girls before the pubertal growth spurt and, in the absence of

serious illness or genetic abnormality, 12 year-old girls and boys are the same height and weight on average.

Physicians routinely monitor height, weight, chronological age, bone age, and growth during the previous year to evaluate how pubertal development is progressing. Since Title IX legislation, which prohibits sex discrimination in any educational program or activity in institutions from elementary school through college that received federal funding, was passed in 1972, female participation in school athletic programs has increased dramatically. Of the total participants in high school athletics in 1971, less than 10 % were female. Today, the proportion of male and female athletes is nearly equal. While exercise is largely beneficial, excessive exercise can utilize energy needed for growth and development and can significantly impact these processes if energy intake is limited by dietary restrictions. This is important because in some competitive sports, and particularly in elite athletes, rigorous training begins before puberty and can potentially influence sexual development.

### 2.3.2 What Happens at Puberty?

Although puberty in human females is generally defined as the process of sexual maturation, and it is certainly that because the changes are primarily driven by the increased activity of the ovaries, a more inclusive definition would also encompass the accompanying physiological and behavioral changes that occur during this transition. This process of sexual maturation requires several years, and the ages of 8–14 years are considered the average range of when this process normally occurs. The defining event, menarche (first menstrual period), occurs at an average of 12.5 years in the United States, but there are differences among ethnic groups and between the United States and other countries. Menarche is a sign that the ovaries are sufficiently functional to support growth and development of the uterine lining. The diagnosis of *primary amenorrhea* is made when menarche does not occur by about age 16. Several years before sexual maturation, increased secretion of steroid hormones from the adrenal gland and ovaries initiates widespread physiologic changes in the body. One of these changes is the adolescent growth spurt. This acceleration in growth lasts for a year or so then slows, and growth is eventually terminated by fusion of the epiphyseal regions of the long bones where growth occurs. Estrogen is responsible for epiphyseal fusion in both males and females. During this pubertal transition in females, there is an increase in percentage of body fat which does not occur in males. Secondary sexual characteristics, such and breast and genital development, are directed by ovarian estrogens, but axillary (underarm) and pubic hair are controlled by androgens from both the ovary and adrenal. The appearance of the female body becomes very different from that of males, and it is primary the hormones, estrogen and progesterone, from the ovary that drive this change. A major question that has not been answered is the following: Why does the secretion of these ovarian steroid hormones increase at this time in life? In other words, what event initiates the process of sexual maturation?

What we do know is that the process of sexual maturation is driven by increased release of a small peptide (a small molecule composed of ten amino acids) called gonadotropin-releasing hormone or GnRH; what we do not know is why it occurs at this specific time. Recent evidence implicates an obligatory role for a peptide called kisspeptin which directly stimulates GnRH release from the hypothalamus [1]. The prevailing hypothesis is that puberty is initiated at some point when the brain and/or body is sufficiently mature to support successful reproduction. When this occurs, pulses of GnRH are released at 1–2 h intervals into the pituitary portal system and travel a short distance from the base of the brain (hypothalamus) to the pituitary and stimulate the release of two protein hormones (large hormones also composed of amino acids) called luteinizing hormone (LH) and follicle-stimulating hormone (FSH). The LH and FSH travel through the circulation to the ovary where they cause growth and development of follicles containing ova (eggs) and, at the same time, stimulate the secretion of the ovarian hormones (Fig. 2.2).

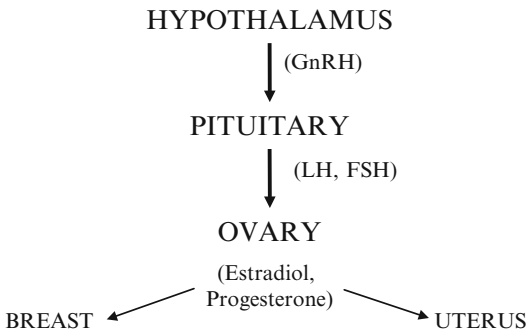



**Fig. 2.2** This figure shows the primary components of the female reproductive system and the hormones that communicate between the various organs. The hormones produced by each gland are shown in parenthesis

This process begins slowly at first, with LH pulses released only at night. Because the LH (and FSH) levels are not maintained at adult levels throughout the day and night, stimulation of the ovarian follicles is not sufficient to result in ovulation. As puberty progresses, the time when LH pulses are released gradually expands to the daytime, and in the adult, these pulses are observed throughout the day as well as at night. In response to FSH and LH, estrogen release from the stimulated follicles results in changes in the body including growth of the breasts and hips primarily due to the deposition of fat. Late in the pubertal process, ovulation occurs when the LH and FSH levels are maintained at a level to provide consistent support for the developing follicle. Menarche usually occurs late in the sequence of events defining puberty.

### 2.3.3 Sexual Maturity: The Reproductive Years

The ability to reproduce is one of the hallmarks of sexual maturity. Sexually mature women who are not taking birth control pills have regular menstrual cycles that average 26–35 days in length and that are (can be) occasionally interrupted by pregnancy and lactation. Menarche signals the beginning of the ability to reproduce although it is possible to become pregnant before the first menstrual period because ovulation can occur during this time. Menopause marks the end of the reproductive lifespan which lasts nearly 40 years from about 13 years (menarche) to 51 years (menopause). Menstruation occurs at the end of an ovarian cycle as the lining of the uterus dies and sloughs off when implantation of a fertilized ovum has not occurred. Menstruation is also the beginning of a new cycle, and by convention, the first day of menstrual flow is day 1 of the cycle. This sequence of follicular development, ovulation, and menstruation is repeated at regular intervals until menopause, unless interrupted by pregnancy and lactation. Reproduction can be postponed with birth control pills or other contraceptive methods for an indefinite period of time when women decide to pursue educational or career goals. Even though hormonal birth control pills inhibit follicular development and ovulation, they do not extend the fertile lifespan which peaks in the twenties and declines thereafter.

### 2.3.4 Menopause: The Climacteric

The average age of menopause (last menstrual period) in the United States is 51, but much like the pubertal transition, menopause is a process that occurs over a period of years. Menopause is recognized when a woman has not had a menstrual period for 12 months. As a woman ages, there is a steady decline in the number of ova (eggs) in her ovaries that can be mustered to develop into follicles with the potential to ovulate. As the number of developing follicles declines, so does the level of estradiol in the circulation. The brain and pituitary, sensing this gradual decline in estrogen, increases the signal (levels of LH and FSH) to the ovary to encourage more follicular development and estrogen production. Thus, as a women approaches menopause, there is a gradual increase in circulating LH and FSH levels, eventually reaching postmenopausal levels that remain high because there is no feedback signal (estrogen) from the ovary to suppress their release. This feedback relationship will be discussed in more detail in Chap. 4. During this menopausal transition, there is also an adjustment to this new hormonal environment

with many psychological and physiological changes, some of which can be unpleasant and disturbing. Symptoms of menopause that most women complain about are vasomotor changes (hot flashes), sleep disruption, mood changes, and urogenital problems. Hot flashes are experienced by about 75 % of menopausal women and typically last for about 3.8 years [2]. Intense heat, sweating, flushing, chills, and clamminess are all symptoms experienced during a hot flash. Once thought to be a figment of the menopausal imagination, hot flashes reflect a real increase in core body temperature and in skin temperature in the digits, cheek, forehead, upper arm, chest, abdomen, back, calf, and thigh [3]. It is interesting that menopausal hot flashes occur at the same time pulses of LH are released from the pituitary [4, 5]. This suggests that the abrupt increases in body temperature are linked to the same central nervous system event that causes the intermittent release of GnRH that stimulates LH release. The current opinion is that since estrogen regulates norepinephrine activity in the brain, and norepinephrine release influences both LH release and body temperature, it is changes in norepinephrine activity due to estrogen withdrawal that causes the hot flashes [3]. Long-term effects of decreased estrogen levels including increased cardiovascular disease, osteoporosis, and decreased mental function are far more debilitating than the transitional changes that occur at menopause.

### 2.3.5 Postmenopause: Life Without Estrogen

At the beginning of the twentieth century, the average age at menopause was 50 years, and this age was also the approximate life expectancy for women at that time (1900). Because the life expectancy at the present time for women has increased to over 80 years, most women will live more than a third of their life after menopause and without estrogen from their ovaries. This extended postmenopausal is a relatively recent phenomenon, and therefore, some of the health-related issues caused by aging are poorly understood and not well documented. Although the increase of 30 years in life expectancy in past 100 years is substantial, the gain in healthy, functional years is less impressive. Many women experience physical and mental impairment in these later years that restricts their social function and isolates them from their friends and family. What is even more disturbing is that because of a variety of environmental influences, many young women have menstrual cycle disturbances that result in the hormonal levels that approximate those seen in menopause. If menstrual cycle disruption is prolonged and particularly if there is amenorrhea during the reproductive years, this can result in some of the same consequences at age 30 or 40 that are usually experienced by women in their seventies and eighties.

#### 2.3.5.1 Osteoporosis

Of all the consequences of aging in women, osteoporosis is the most debilitating and affects the most women. The risk of a lumbar or hip fracture, particularly after the ages of 65, approaches 50 % in white women. There are effective treatments for this condition including hormone replacement therapy (HRT) and bisphosphonates. The HRT, specifically estrogen therapy, reduces bone turnover and improves calcium homeostasis. However, there are drawbacks to HRT and the risks of breast and uterine cancer in individuals with a family history of these diseases must be considered when decisions regarding the treatment of osteoporosis are made.

#### 2.3.5.2 Alzheimer's Disease

A significant percentage of older women have some form of dementia (deterioration of cognitive function), and estrogen may protect against this deterioration [6]. Although, compared with previous studies, the recent results of the Women's Health Initiative (WHI) suggest that there is an increased risk of ischemic stroke with estrogen (Premarin) either with or without progesterone (Prempro), this trial used HRT on older menopausal women with obesity as a complicating factor [7–10]. Well-controlled studies with the native estrogen, estradiol-17β, are needed before a rational, effective treatment regimen for menopausal/postmenopausal women can be

safely proscribed. There are studies suggesting that estrogen replacement therapy, if begun at menopause and continued for a few years, is effective in reducing both the risk for osteoporosis and dementia.

#### 2.3.5.3 Coronary Artery Disease and Stroke

The overwhelming evidence from observational studies indicates that estrogen has a protective effect against coronary artery disease [11]. This effect of estrogen appears to be limited to prevention of cardiovascular disease and does not ameliorate the progression of coronary disease that is established [12]. However, two recent trials, HERS (Heart and Estrogen/progestin Replacement Study) and WHI (Women's Health Initiative), have brought these observational data into question. These two large trials with 2,763 women (HERS) and 16,608 women (WHI) found no net benefit (HERS) or an increased risk (WHI) of coronary artery disease with HRT [13].

#### 2.3.5.4 Breast and Endometrial Cancer

One of the main concerns in women who take HRT for menopausal symptoms is the increased risk of breast and endometrial cancer. The analysis of some 50 studies clearly indicates an increased risk for breast cancer in women taking estrogen alone [14]. The risk is increased substantially when women are on combined treatment of estrogen and progesterone [15]. Conversely, progesterone has a protective effect against the increased incidence of endometrial cancer in postmenopausal women taking estrogen therapy alone [16].

## 2.4 Current Understanding of the Issues

Because of the dramatic increase in life expectancy in the past century, many women are living 30 or 40 years or more after menopause. Several studies have compared various health outcomes between groups of women who have and have not taken hormone replacement therapy (HRT) after menopause. Because the widely publicized results of these studies suggested increased risk of certain diseases with HRT, physicians and their patients are reluctant to use HRT, which may actually increase postmenopausal health problems unnecessarily. However, the studies have used different hormone formulations and different routes of administration, concentrations, and hormone sequence, given to women of different ages, and therefore are not directly comparable [18]. At the present time, there are data suggesting that brain and bone health benefit from HRT, but the effects on cardiovascular disease and breast and uterine cancer are not clear. What has emerged in the past two decades is that there are at least two forms of the estrogen receptor mediating the actions of this hormone on estrogen-sensitive tissues (bone, brain, breast, uterus, and heart) as well as adipose tissue and the immune system [18]. Furthermore, many factors including the age, health status, genetic background, previous hormone environment of the patient, and route and sequence of administration of HR may all influence the outcome of HRT [17, 18].

## 2.5 Future Directions

The HRT has primarily been used to treat the menopausal symptoms that cause the most discomfort: hot flashes, vaginal dryness, and disturbed sleep. However, evidence from large clinical trials has revealed that our knowledge of the constellation of effects of HRT on cardiovascular health, cognition, and reproductive organs is very limited. Additional studies examining both basic and clinical aspects of the effects of HRT on hormone responsive systems are needed before rational replacement therapies can be effectively used to improve health outcomes in postmenopausal women.

## 2.6 Conclusion

During the lifespan of women, there are dramatic and life-changing transitions associated with the beginning and cessation of reproductive functions.

These transitions, puberty and menopause, result in dramatic changes in the anatomy, physiology, and cognitive function of females and are caused by fluctuating levels of estrogen and progesterone. Because the life expectancy for women is now approaching 80 years and menopause occurs at about 50 years of age, the average female will live approximately 30 years after her ovaries have ceased to produce estrogen. This has serious physical and mental health implications in the aging population, which will increase dramatically in the next few decades. Additional research is needed to unravel the complex actions of hormone replacement formulations so that women can have a healthier life after menopause.

## References


1. Messager S, Chatzidaki EE, Ma D, et al. Kisspeptin directly stimulates gonadotropin-releasing hormone release via G protein-coupled receptor 54. Proc Natl Acad Sci. 2005;102:1761–6.
2. Avis NE, Crawford SL, McKinlay SM. Psychosocial, behavioral, and health factors related to menopause symptomatology. Womens Health. 1997;3:103–20.
3. Freedman RR. Pathophysiology and treatment of menopausal hot flashes. Semin Repro Med. 2005;23: 117–25.
4. Caspar RF, Yen SSC, Wilkes MM. Menopausal flushes: a neuroendocrine link with pulsatile luteinizing hormone secretions. Science. 1979;205:823–5.
5. Tataryn IV, Meldrum DR, Lu KH, et al. LH, FSH, and skin temperature during menopausal hot flush. J Clin Endocrinol Metab. 1979;49:152–4.
6. Wise PM. Estrogens and cerebrovascular stroke: what do animal models teach us? Ann N Y Acad Sci. 2005;1052:225–32.
7. Rossouw JE, Anderson GL, Prentice RL, et al. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women's Health Initiative randomized controlled trial. JAMA. 2002;288:321–33.
8. Rapp SR, Espeland MA, Shumaker SA, et al. Effect of estrogen plus progesterone on global cognitive function in postmenopausal women. JAMA. 2003;289:2663–72.
9. Shumaker SA, Legault C, Rapport SR, et al. Estrogen plus progestin and the incidence of dementia and mild cognitive impairment in postmenopausal women. JAMA. 2003;29:2651–62.
10. Anderson GL, Limacher M, Assaf AR, et al. Effects of conjugated equine estrogen in postmenopausal women with hysterectomy: the Women's Health Initiative randomized controlled trial. JAMA. 2004;291:1701–12.
11. Barrett-Conner E. Hormone replacement therapy. BMJ. 1998;317:457–61.
12. Herrington DM, Reboussin DM, Brosnihan KB, et al. Effects of estrogen replacement on the progression of coronary-artery atherosclerosis. New Eng J Med. 2000;343:522–9.
13. Hulley SB, Grady D. The WHI estrogen-alone trial—do things look better? JAMA. 2004;291:1769–71.
14. Collaborative Group on Hormonal Factors in Breast Cancer. Breast cancer and hormone replacement therapy: collaborative reanalysis of data from 51 epidemiological studies of 52,705 women with breast cancer and 108,411 women without breast cancer. Lancet. 1997;350:1047–59.
15. Willet WC, Colditz G, Stampfer M. Postmenopausal estrogen-opposed, unopposed, or none of the above. JAMA. 2000;283:534–5.
16. Grady D, Rubin SM, Petitti DE. Hormone therapy to prevent disease and prolong life in postmenopausal women. Ann Intern Med. 1992;117:1016–37.
17. Turgeon JL, McDonnell DP, Martin KA, et al. Hormone therapy: physiological complexity belies therapeutic simplicity. Science. 2004;304:1269–74.
18. Turgeon JL, Carr MC, Maki PM, et al. Complex actions of sex steroids in adipose tissue, the cardiovascular system, and brain: insights from basic science and clinical studies. Endoc Rev. 2006;27:575–605.

# Considerations of Sex Differences in Musculoskeletal Anatomy

3

Phillip S. Sizer and C. Roger James

**Abstract**

The musculoskeletal anatomy of women and men is grossly similar yet individually distinctive. Sexual dimorphism in the human musculoskeletal system is apparent, but more subtle than in other species. Some musculoskeletal sex differences in humans are present at an early age, while others tend to appear later in life. Sex differences in gross skeletal geometry and specific tissue characteristics are common. Women tend to have different characteristics of specific bones and bony features than men which have been explained by both genetic and environmental factors. Women and men appear to have several differences in collagenous, cartilage, and bone tissues, which may predispose women to certain pathologies such as osteoarthritis and osteoporosis later in life. Sexual dimorphism can manifest itself in specific differences in each joint throughout the body, possibly resulting in sex differences in clinical pathology and symptomology such as differences in shoulder impingement; laxity and idiopathic capsulitis; elbow tendinosis; carpal tunnel syndrome; hip fracture and labral tears; anterior cruciate ligament injuries; ankle sprains and Achilles tendinopathy; cervical spine macrotrauma; thoracolumbar postural changes including kyphosis, lordosis, and/or scoliosis; and sacroiliac joint conditions. Consideration of the sex differences in musculoskeletal anatomy is important for both the general public and health care professionals in order

P.S. Sizer, P.T., Ph.D., O.C.S., F.A.A.O.M.T. (✉)
SCD Program in Physical Therapy, Department of Rehabilitation Sciences, Texas Tech University Health Sciences Center, Lubbock, TX, USA
e-mail: phil.sizer@ttuhsc.edu

C.R. James, Ph.D., F.A.C.S.M.
Department of Rehabilitation Sciences, Texas Tech University Health Sciences Center, 3601 4th Street STOP 6223, Lubbock, TX 79430-6223, USA
e-mail: roger.james@ttuhsc.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_3, © Springer Science+Business Media New York 2014

to provide a basis for understanding normal and abnormal conditions that may exist. Moreover, a thorough appreciation that men and women have differences in musculoskeletal anatomy may help in the understanding that they have distinctive health care needs.

**Keywords**
Sexual dimorphism • Female • Pathoanatomy • Anatomy • Sex differences • Structure

## 3.1 Learning Objectives

After completing this chapter, you should understand:

1. Sexual dimorphism and how it applies to humans
2. Sex differences in general morphology
3. Sex differences in skeletal geometry
4. Sex differences in collagenous, cartilage, and bone tissue
5. Sex differences in the upper extremity anatomy and mechanics
6. Sex differences in the lower extremity anatomy and mechanics
7. Sex differences in the spine anatomy and mechanics

## 3.2 Introduction

The musculoskeletal anatomy of women and men is grossly similar yet individually distinctive. Structural differences exist between the sexes and these differences are due to both environmental and genetic factors. Sex differences in musculoskeletal anatomy can be described in terms of sexual dimorphism, which refers to physical differences in secondary sexual characteristics, such as size or color, between male and female individuals of the same species [1, 2]. Sexual dimorphism is present in many species of birds, spiders, insects, reptiles, fish, and mammals. Examples include male pheasants which are larger and more brightly colored than female pheasants, female spiders which are usually larger than their male counterparts, male deer which grow antlers, and the males of most species of mammals which are larger than the females [2]. However, with a few well-recognized exceptions, such as body hair, muscle mass, and breast differentiation, sexual dimorphism in humans is more subtle as compared to other species [2]. Yet, most people recognize that men and women exhibit different physical characteristics that include differences in body height, weight, shape, size, and alignment of the extremities (e.g., pelvic width, body mass distribution, and ligament/tendon laxity) [2–4]. Some of these differences in body structure are widely recognized and ingrained in cultural beliefs and stereotypes. For example, an artist's rendition of a typical man and woman was used to depict the sexes of the human species on the plaque of the Pioneer 10 spacecraft (Fig. 3.1) where the differences in gross structure are evident.

The typical differences in physical characteristics of the sexes are further exemplified by population data. Data from standard growth charts [5] demonstrate typical sexual dimorphic differences, but the division between men and women is usually less than one standard deviation and is age-dependent [6]. For example, according to the clinical growth charts provided by the Centers for Disease Control (CDC), girls and boys at the 50th percentile are approximately the same height (usually within 1–2 cm) until puberty (Fig. 3.2). However, beginning at about the age of 14 years, the heights of girls and boys diverge at an increasing rate until growth slows in both sexes in the late teen years. At the age of 20 years, men are an average of approximately 14 cm taller than women (Fig. 3.2) [5]. Similar relationships are documented for body weight, with a relatively

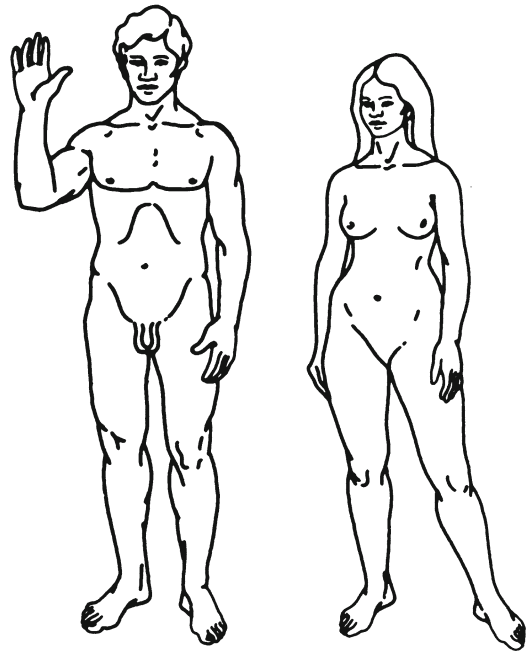


**Fig. 3.1** Symbolic representation of men and women as depicted on the plaque of the Pioneer 10 spacecraft in 1972. *Source*: NASA (www.nasa.gov/centers/ames/images/content/72418main_plaque.jpg). Adapted with permission

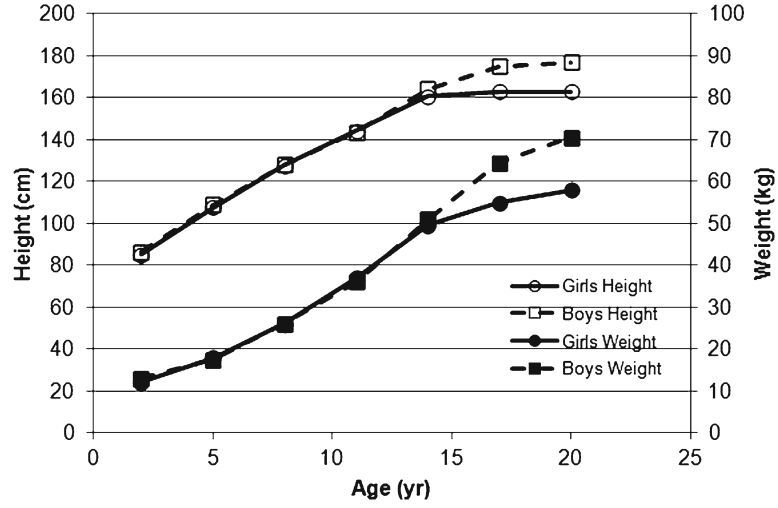



**Fig. 3.2** 50th-percentile height and weight of girls and boys from 2 to 20 years. Sexual dimorphic differences in height and weight begin to emerge at about the age of 14 years. Values are rounded to the nearest 0.5 cm and 0.5 kg. Adapted from www.cdc.gov/growthcharts (Anonymous 2006)

small sex difference observed before the age of 14 years and an approximately 12.5 kg difference (men greater than women) at the age of 20 years (Fig. 3.2) [5].

While sexual dimorphism is apparent in general body characteristics, other sexual dimorphic traits are less obvious. Reports of sex differences in skeletal and soft tissue components are prevalent in the literature, and these differences explain the differences in general appearance but also may influence movement patterns, injury risk, and the development and progression of musculoskeletal pathology. Consideration of the sex differences in musculoskeletal anatomy is important for both the general public and health care professionals in order to provide a basis for understanding normal and abnormal conditions that may exist. Moreover, a thorough appreciation that men and women have differences in musculoskeletal anatomy may indicate that they have distinctive health care needs. Therefore, the purposes of this chapter are to (1) examine sex differences in the anatomy of selected musculoskeletal components and (2) explore selected regional considerations in female functional pathoanatomy that are pertinent to women's health issues.

## 3.3 Research Findings

### 3.3.1 Sex Differences in the Anatomy of Selected Musculoskeletal Components

#### 3.3.1.1 Sex Differences in Skeletal Geometry

There are several differences in skeletal geometry between men and women. In the sports medicine literature, sex differences in musculoskeletal anatomy, including skeletal geometry, have been reported in context with common injuries that occur in active women and have mostly focused on lower extremity characteristics. For example, differences in size, shape, structure, or alignment of the pelvis, femur, tibia, tarsals, and toes all have been reported [3, 7–11]. Sex differences in the pelvis include a larger inlet and outlet [3], greater interacetabular distance [8], and a greater hip width normalized to femur length [4, 7] in women as compared to men. Sex differences in the femur include increased femoral anteversion [4] and a narrower femoral intercondylar notch width [9, 12] in women. However, other authors have reported no differences in notch width between the sexes [13, 14]. Greater genu recurvatum [3, 11], more lateral patellar alignment [3], increased internal tibial torsion [11], greater slopes of the medial and lateral tibial plateaus [15], and more bunions and deformities of the lesser toes [3] in women have been reported, as well. Because of the mechanical linkage and interaction among structures of the lower extremity, skeletal differences in one or more interacting structures may result in differences in overall lower extremity alignment. For example, quadriceps angle (Q angle) is the angle formed by the intersection of a line connecting the anterior-superior iliac spine and the midpoint of the patella with a line connecting the midpoint of the patella and the tibial tuberosity. In the literature, it is generally reported that Q angle is greater in women than in men [7, 10] and is a function of the structural and alignment characteristics of the involved bones (e.g., pelvic width, patella position, tibial torsion). Additionally, large deviations in Q angle have been suggested to contribute to selected knee and foot pathologies [3, 16], although reports are equivocal.

Further evidence supporting the existence of sex differences in skeletal geometry comes from the areas of forensic anthropology and archeology. Scientists from these areas have used knowledge of skeletal sexual dimorphism to determine the sex of deceased individuals from their skeletal remains. A large amount of literature exists that discusses the skeletal geometric characteristics commonly used and the ability of these characteristics to predict sex. The humerus [17], pelvis [18, 19], femur [17, 20–25], tibia [26–28], talus [29], and calcaneus [29–31] all have been used for this purpose. The ability to discriminate sex based on one or more skeletal geometric characteristics varies somewhat by which bone, as indicated by the percentage of individuals accurately categorized as male or female in the

respective studies cited: tibia (96 %) [27], femur (95 %) [21], calcaneus (92 %) [30], pelvis (88 %) [19], and talus (81 %) [29]. Furthermore, different parameters from the same bone appear to be better discriminators than other parameters [18, 19, 21, 24–26, 31]. For example, Mall et al. [24] reported that a differing percentage of individuals could be grouped correctly using a discriminant analysis when evaluating different characteristics of the femur. In this study, the femoral head transverse diameter (89.6 % of cases correctly categorized) was the best discriminator, followed by the head circumference (87.7 %), vertical head diameter (86.8 %), condylar width (81.4 %), maximum midshaft diameter (72.4 %), and maximum length (67.7 %) [24]. Using a combination of variables (midshaft diameter and head circumference), 91.7 % of the cases were classified correctly [24].

The presence and magnitude of sex differences in skeletal geometry appears to be dependent on a number of factors including skeletal maturity [18, 32], environmental stresses (i.e., loading) [17, 21, 23, 28], and genetics (i.e., race) [19–23, 25, 33]. Consideration of these factors is important in establishing sexually dimorphic traits in skeletal geometry. Nevertheless, ample evidence exists to support the presence of sex differences in skeletal geometry.

### 3.3.2 Sex Differences in Musculoskeletal Tissues

In addition to sex differences in skeletal geometry, there are several reported sex differences in musculoskeletal constituents, including collagenous (e.g., tendon, ligament, skin), cartilage, and bone tissues [34–37]. Collagen is a primary protein of connective tissues in mammals and constitutes more than 30 % of the protein in the human body [38]. It provides much of the strength of tendon, ligament, skin, and cartilage [38, 39]. Additionally, it is the main protein component of bone [38, 39].

In collagenous tissues, the collagen molecules align based on stress patterns and provide strength against tensile loads [38, 39]. Some of the reported sex differences in collagen include differences in thickness [34], orientation [34], content [40], diameter [35], volume [36], and metabolism [37] for the specific tissues examined. Some of these sex differences are associated with fiber strength. Moreover, sex differences in collagen degradation have been observed in subjects as young as 2 years of age [37], while other sex differences may not appear until after several decades of life [36]. Many disorders of collagenous tissues (e.g., lupus erythematosus, scleroderma, rheumatoid arthritis, dermatomyositis, Sjögren's syndrome) have been associated with sex differences. Etiological factors associated with collagenous tissue diseases are thought to exist at many levels (e.g., genetic, cellular, organ, age, behavioral, environmental), but sex hormones are thought to influence the onset and course of these disorders [41].

Sex differences in articular cartilage are reported in the literature [42–45] and have been associated with differences between men and women in the onset of osteoarthritis [43, 45, 46]. Epidemiological evidence suggests that women are more likely (1.5–4 times at greater risk) to develop osteoarthritis than men [43]. Sex differences in articular cartilage morphology have been reported in children (age 9–18 years), which persist throughout adulthood, and increase during the postmenopausal years [42–44, 47]. Reported sex differences in articular cartilage morphology include greater cartilage volume, thickness, and surface area in male compared to female subjects [42–45]. These differences appear to be partially related to other characteristics such as age, body mass index, bone region, physical activity, and the specific articulation involved [43, 47]. However, research suggests that sex differences remain at some cartilage sites even after adjustment has been made for these other factors [42, 43]. Furthermore, the sex differences in cartilage morphology have been associated with a faster cartilage tissue accrual rate in boys compared to girls (i.e., more cartilage tissue early in life) [43] and greater cartilage tissue degradation in older women compared to older men (i.e., greater loss of cartilage later in life) [48].

The presence of sex hormone receptors in cartilage tissue is thought to be an indicator that sex hormones influence these accrual and degradation processes [45, 46, 48, 49]. Evidence for the role of sex hormones in cartilage morphology and metabolism has been demonstrated in both animal [45, 49] and human [48] models. In mice, sex hormones have been shown to influence the inflammatory induction of cartilage degradation via modulation of cytokine production and release in granulomatous tissue [49]. Additionally, male rats have demonstrated higher levels of proteoglycan and collagen, less glycosaminoglycan loss, and greater proteoglycan synthesis than female rats in vitro [45]. Furthermore, cartilage from female rats was shown to have greater susceptibility to degradation when implanted into female rats compared to male cartilage implanted in female rats or both male and female cartilage implanted into male rats [45]. In humans, urinary markers of cartilage degradation have provided evidence that cartilage loss is greater in women than in men [48]. Additionally, cartilage degradation was shown to be greater in postmenopausal women compared to age-matched premenopausal women and less in postmenopausal women undergoing hormone replacement therapy compared to postmenopausal women not undergoing the therapy [48]. Therefore, sex differences in cartilage morphology and metabolism that exist early in life appear to increase with advancing age and may be explained by the difference in sex hormones.

Sex differences in bone tissue also are reported in the literature [50–53] and have been associated with differences between men and women in terms of fracture risk [54–56]. Sex differences in bone tissue vary by skeletal site [32, 57], but are reflected by differences in both morphological [32, 51, 54, 56–60] and remodeling [50, 52, 53, 57, 60, 61] characteristics, particularly in osteoporotic individuals [39, 57, 62, 63]. Sex differences in bone tissue are present at an early age [32, 50, 51, 58], persist throughout adulthood [39, 52–54, 60, 61], and diverge even more in older age [39, 52, 53, 55–57, 59, 61]. Bone mass accrues during childhood through adolescence and peaks at about the age of 30 years in both men and women [39]. A lower amount of peak bone mass has been associated with a greater risk for osteoporosis in later life; [50] therefore sex differences in the development of bone tissue during youth may partially explain some of the bone tissue differences between adult men and women. Several factors influence bone mass accrual, including nutrition (e.g., calcium, vitamin D), physical activity, lifestyle behaviors (e.g., smoking), genetic factors (race, sex), and hormonal factors (e.g., estrogen) [62, 64].

In children, serum markers of bone turnover have been shown to change significantly with pubescence in both boys and girls [50]. In girls, these markers were shown to peak at mid-puberty and decrease thereafter; in boys, the markers continued to increase through late puberty [50]. Moreover, the serum markers of bone turnover were overall higher in boys than girls even after adjustment for age, body weight, and pubertal stage [50]. These sex differences in bone turnover more than likely influence differences in peak bone mass [50], volumetric bone mineral density [51], selected measures of bone area [32], cortical thickness [32], plus ultimately compressive [51] and bending [58] strength. However, some authors have suggested that sex differences in some of these characteristics can be explained by differences in anthropometric dimensions (e.g., height and total lean body mass) [58].

In adults, there are several reported sex differences in bone tissue. Differences between men and women have been reported in bone mineral density (men greater than women) [60], peak bone mass (men greater than women) [39], cortical thickness (men greater than women) [54], age-dependent hormonal responsiveness of osteoblasts (less responsiveness in older cells from women when compared to older cells from men) [52], bone turnover (men greater than women) [60], and bone strength (men greater than women) [39, 54]. In older adults, sex differences in bone mineral density [55, 57], cross-sectional area [55], cortical thickness [55], bone width [55], and strength [39, 55, 59] remain considerable, with some differences (i.e., bone mineral density and strength) further diverging as compared to younger adult values [39, 55].

Furthermore, many of these sex differences do not disappear after adjusting for anthropometric factors such as height and weight [55].

The role of sex hormones in the loss of bone mass in older women has been explored widely in the literature. It is well known that a decrease in estrogen production following menopause is a primary contributing factor to the accelerated loss of bone mass in older women when compared to older men [62, 63]. However, older men also lose bone mass and are at greater risk for hip and vertebral fractures compared to younger men [53]. Even though men do not experience a physiological event comparable to menopause and therefore do not undergo a substantial decline in total serum testosterone or estrogen, some evidence suggests that a decline in the bioavailable estrogen (non-sex hormone-binding globulin bound) might explain the loss of bone mass in both women and men [53].

### 3.3.3 Selected Regional Considerations in Female Functional Pathoanatomy

#### 3.3.3.1 Upper Extremity

##### Shoulder

Sex-based anatomical differences of the shoulder complex are tissue- and pathology-specific. For example, Pandley et al. [65] observed significant differences between men and women in the distribution of the articular branch of the axillary artery, which may influence decisions made during shoulder surgery. Differences have been associated with the incidence of external impingement of the shoulder. While women appear to have less prominent coracoid processes [66], no sex-based differences have been found in the role of the coracoid process in subscapularis impingement [67]. Furthermore, differences in the role of the acromion with external impingement have been observed. Historically, Bigliani [68] classified the acromial shape into three types. Type I processes are flat, Type II are curved, and Type III are hooked. Bigliani [68] suggested that these differences could lend to the incidence of impingement and any subsequent rotator cuff tearing. Later, Bigliani et al. [69] reported that 78 % of all full thickness rotator cuff tears were associated with a Type III acromion. More recent investigations have reported an increased incidence of full thickness rotator cuff tears in women versus men [70]. Selected investigators have suggested that acromial differences are acquired, resulting from altered tension loads imposed by the coracoacromial ligament and deltoid insertions [71]. Getz et al. [72] observed that Type III acromia were more common in female patients and discovered that Type II acromia were related to adaptive shortening of the glenohumeral joint posterior capsule.

Although external impingement has been associated with sex and age, the relationship between age, sex, and incidence of acromial type is controversial [72–74]. More recently investigators have suggested that while the inferior surface of the acromion changes increased with age, they were not different between sexes [75]. However, investigators have observed limited sex-based differences in the acromiohumeral distance with the shoulder at rest, where women exhibited a reduced space compared to men [76]. The influence of gender-based differences in scapular position at rest on impingement pathology needs further exploration, where women appear to demonstrate less protraction versus their male counterparts [77].

While men appear to experience more frequent anterior dislocations of the glenohumeral joint [78], women appear to be more predisposed to glenohumeral instability [79]. This disparity appears to be related to the notion that not all joint instability results in dislocation, where grade I and II instability represents increased motion and possible humeral head perching on the anterior labrum, versus the frank dislocation of the head in grade III [80]. While glenoid fossa inclination appears to influence instability incidence [81], few sex differences in this architectural feature have been noted [82]. Recently investigators found that women demonstrate differences in glenoid fossa shape, by being more oval and exhibiting deeper anterior glenoid notches [83]. Although the woman's predisposition appears to be more related to increased

anterior capsular laxity and resultant hypermobility along with decreased joint stiffness [79], further research is merited for studying the relationship between these architectural findings and the onset and persistence of glenohumeral pathology.

Finally, women between the ages of 40 and 60 years are more predisposed to developing idiopathic capsulitis [84]. This condition is associated with increased thickening of the anterior-superior joint capsule at the coracohumeral ligament [85], along with a noninflammatory synovial reaction in the proximity of the subscapularis tendon [86]. These changes demonstrate active fibroblastic proliferation accompanied by tissue transformation into a smooth muscle-like phenotype that is similar to Dupuytren's disease [87].

### Elbow

Women appear to be at greater risk for developing tennis elbow due to tendinosis that emerges from mesenchymal changes in the collagenous constituents of tendons [88]. This condition, typically lasting greater than 12 months in duration, is more likely noninflammatory in nature [89] and affects one of four possible different regions of the tendinous insertions at the lateral elbow. The tendons that are at risk are specifically located about the lateral epicondyle of the distal humerus. The extensor carpi radialis longus (ECRL) originates on the distal 1/3 of anterior supracondylar ridge, possesses almost no tendon at the origin, and demonstrates an immediate transition into muscle. Extensor carpi radialis brevis (ECRB) starts from a 5 mm by 5 mm square area on the superior surface of the lateral epicondyle (10 % of origin) and collagen/fascial layers of intra-compartmental septa that share fascia with the extensor digitorum communis (EDC) coursing distally to the second and third metacarpals, especially fascia associated with third metacarpal. Thus, resistive wrist extension and resistive extension of the second and third metacarpophalangeal joints may be suggestive of tendinopathy at either EDC or ECRB. The ECRB tendon is juxtaposed between the muscle bellies of ECRL and EDC. This common physical finding merits palpatory discrimination between the two regions for a differential diagnosis. The ECRB can exhibit tendinopathy at its origin, along the tendon between ECRL and EDC, or at its musculotendinous junction more distally. Finally, the EDC can be found at the anterior surface of the lateral epicondyle. If involved in a lateral tendinopathy, resistive extension of the second through fifth metacarpophalangeal joints (MCPJ 4 and 5 differentiating this lesion from a lesion of the ECRB). This condition seldom occurs in isolation, but is typically discovered in combination with affliction to the ECRB [90].

The woman's predisposition for lateral elbow tendinosis is increased when her estrogen decreases, especially after premature hysterectomy (at less than 35 years of age) and/or lowered estrogen levels from other causes [88]. While inflammatory tendinitis involves a chemically mediated inflammation due to tendon injury [88], the tendinosis to which women are more predisposed produces a non-chemically mediated degenerative change associated with long-term tendon stress [91], resulting in a condition that could persist long after 12–14 months. This process produces tissue necrosis that manifests as a "moth-eaten" appearance in the tendon [92]. As a consequence, the tendon becomes friable, along with possible bony exostosis at the lateral epicondyle [88]. This chronic condition can be accompanied by an imbalance between vasodilatory and vasoconstrictive variations [92], substance P, and CGRP proliferation in the vicinity of the affected tendon [92], accompanied by a high concentration of glutamate in the surrounding tissue [93].

Women also appear to be more predisposed to ulnar nerve lesions at the medial elbow [94]. The medial elbow anatomy affords three different predilection sites for ulnar nerve entrapment. The nerve first courses under the arcade of Struthers just dorsal to the medial intermuscular septum. Distally, the nerve courses through the cubital tunnel, whose boundaries are the medial collateral ligament complex (ceiling), medial epicondyle (medial wall), olecranon process (lateral wall), and cubital tunnel retinaculum (floor). In selected individuals, the retinaculum is dorsally bordered by the anconeus epitrochlearis muscle that is innervated by the radial nerve and is activated simultaneous with the triceps, lending to possible entrapment symptoms during

resisted elbow extension. The retinaculum that courses from the medial epicondyle to the olecranon tightens with passive elbow flexion, creating increased nerve entrapment symptoms at end-range passive flexion. Finally, the nerve must course under the retinaculum between the two heads of the flexor carpi ulnaris. As a consequence, entrapment symptoms may increase during resistive wrist flexion-ulnar deviation as well.

#### Wrist and Hand

Only a few afflictions of the wrist and hand appear to differ between men and women. Tendon pathologies, including tenosynovitis and tendinosis, seem to be more frequent in women [95], but data related to the pathoanatomical and physiological influences on these differences have not been explored. Similarly, carpal tunnel syndrome (CTS) is more common [96, 97] and prolonged [98] in women. However, multiple factors have been elucidated that may contribute to this difference. The etiology of carpal tunnel syndrome is multifactorial, resulting from anatomical, biomechanical, pathophysiological, neuropathological, and psychosocial influences. Anatomical factors, such as tunnel architecture and volume [99], lumbrical anatomy [100], and the shape of the hamate hook [101], have been associated with CTS. Specific anatomical and anthropometric factors appear to influence a woman's greater predisposition to CTS [102, 103]. While carpal bone size and scaling do not appear to differ between men and women [104], hand-length ratios, space indices at the wrist, and digital features appear to differ between the sexes [102]. Along with these, differences in body mass index seem to predispose women to CTS [102].

The onset and progression of CTS appear to be related to an increase in intra-tunnel pressure [105]. Different factors appear to increase this pressure, including tunnel space narrowing associated with wrist movements [106, 107], carpal instability [108], increased muscle force production [109], and trauma that produces perineural edema and fibrosis [110]. Women may be more susceptible to these influences versus men, due to reduced available space for the median nerve within the tunnel. The median nerve appears to increase in cross-sectional diameter with sustained repetitive hand movement in women as compared to men [103], thus compromising the relative tunnel size and potentially increasing pressure within the tunnel in context with the previously discussed factors.

The individual suffering from CTS may experience sensory and/or motor changes, including paresthesias or true numbness that reflects deficits in neurophysiological function. Women have demonstrated greater deficits in neurophysiological function involving the median nerve when compared to men [111, 112]. However, controversy exists over the value of neurophysiological testing for the diagnosis of CTS [113]. Orthodromic median sensory latency is typically prolonged with CTS patients [114], and median nerve motor amplitudes are decreased in patients with CTS [112]. Yet, Glowacki [115] discovered a poor relationship between electrodiagnostic test outcomes and final symptom consternations. Differences between the median and ulnar motor latencies appear to be important for the diagnosis of CTS [111]. Padula [116] found that the difference between the median and ulnar motor latencies was greater in patients experiencing CTS versus controls.

The presence of autonomic disturbances appears to be associated with the woman's predisposition to CTS [117]. This disturbance in neural function could be related to local sympathetic fiber stimulation and/or brachial plexus irritation associated with a double crush phenomenon, which has been observed in as many as 40 % of all patients suffering from CTS [118]. As result, a vasoconstrictive event could lead to decreased perineural microvascular flow and increased protein leakage from the vascular supply that produce epineural and perineural edema [110], as well as increased endoneural pressure and ischemia [119], contributing to the symptoms of CTS.

### 3.3.3.2 Lower Extremity

#### Hip Joint

Women are at greater risk for both microtraumatic stress fracture [120] and macrotraumatic frank fracture at the hip [121] especially involving the femoral neck [120]. This predilection appears to be influenced by differences in bony

architecture about the hip and pelvis [122]. Acetabular depth and femoral head width appear to be less in women versus men [123]. The coxa-diaphyseal angle has been reported to be wider in men versus women in selected races, thus potentially predisposing the women to higher incidence of stress reactions [124]. Women appear to have decreased femoral neck strength versus men, as evidenced by decreased femoral neck cross-sectional moment of inertia (CSMI) [125, 126]. Compressive stress (Cstress), defined as the stress in the femoral neck at its weakest cross section arising from a fall, is higher in women [126]. These features interact with women's altered estrogen associated with menstrual irregularities [127] and menopause [121], thus enhancing their fracture risk predisposition. Over the past decade, postmenopausal women have relied upon the long-acting, bone density-maintaining effects of bisphosphonate administration for reducing the rate of fragility fractures in this population [128]. However, this benefit has been accompanied by an increase in atypical subtrochanteric fractures at a younger age in response to chronic use [128], especially witnessed in Asian females [129, 130]. The risk associated with bisphosphonate use continues to be small but controversial [131], where comorbidities and management strategies should be assessed when its usage is considered [128].

The outer margin of the hip acetabulum is completely lined with the cartilaginous labrum that serves to enlarge the articular surface [132, 133]. The labrum enhances the articular seal, fluid pressurization, load support, and joint lubrication of the hip joint [134]. The labrum possesses a variety of sensory endings important to proprioception and nociception [135]. The labrum is vascularized in a fashion similar to the meniscus of the knee, where the outer margins are well vascularized and the inner margin is lacking in blood vessels [136]. The labrum is at risk for traumatic vertical, as well horizontal, degenerative tears [133, 137]. The propensity for tears is increased by the deficiencies in the mechanical properties of the labral tissue, especially in women. Labra obtained from male patients have stronger tensile stress than those from female patients [138]. Moreover, labral degenerative changes may influence those same mechanical properties, adding to the risk of tearing [139, 140].

Labral tears appear to occur more frequently in the superior region of the acetabular structure, due to decreased mechanical properties accompanied by increased demand [139, 141]. The superior region of the labrum appears to be less well vascularized, lending to the susceptibility of that region to traumatic and degenerative tears [139, 141]. One significant mechanical contribution to this loading demand is the impact of the femoral neck against this region during full flexion of the hip [140]. Femoral neck architecture also appears to differ between men and women, where increased thickening and decreased coxa-diaphyseal angulation of the neck and deformation/fullness of the neck diameter in older women predispose them to anterior acetabular labral trauma, especially when the hip is positioned in full flexion [142]. However, severity of such deformations and changes observed with imaging do not appear to correlate with the incidence of femoral-acetabular impingement and subsequent labral lesions [143], making the clinical examination paramount to diagnosis.

### Knee Complex

Little evidence is available to describe sex-based differences in the patellofemoral complex of the knee. One might explain differences in terms of cartilage volume, where sex explains 33–42 % of the variation in knee cartilage volumes with women demonstrating decreased cartilage volume versus men [42]. However, T2 MRI examination of young, healthy volunteers did not reveal sex-based differences in the magnitude or spatial dependency of cartilage [144].

Investigators have attempted to describe sex-based differences in terms of patellofemoral contact areas at various positions of knee flexion. In males, Csintalan et al. [145] observed larger contact areas of posterior patellar surfaces with the knee flexed to 30°. In addition, they observed a greater change in the female's contact pressures in response to varying vastus medialis activity with the knee positioned at 0°, 30°, and 60° flexion.

While no differences were seen by Besier et al. [146] with the knee in full extension, they observed larger contact areas in male patellofemoral joints with the knee flexed to 30° and 60°. However, the contact areas were not different when the data were normalized by patellar dimensions of height and width. Investigators have turned their attention to the role of hip control deficits in landing in the development of anterior knee pain syndrome (AKPS), where decreases in eccentric control from the hip external rotators and abductors were shown to be associated with increased AKPS [147]. However, Cowan and Crossley [148] found no relationship between hip control deficits and gender in subjects suffering from AKPS.

More striking is the relationship between sex and knee ligament injury. Injury to the anterior cruciate ligament (ACL) can be a devastating event, and a woman's increased risk for this injury over male counterparts is well documented [149, 150]. It has been reported that 70 % of all ACL injuries are a result of a non-contact mechanism [149, 151], where girls and women appear to tear their ACL two to eight times more frequently than men [152].

Since the reason for this increased ACL injury risk is unclear, investigators have explored many possible causes including anatomical, hormonal, and mechanical differences. One of the classic anatomical factors attributed to sex-based differences in ACL injury is the width of the femoral intercondylar notch or Grant's notch. The intercondylar notch is found in the roof of the space between the femoral condyles, lending a point where the ACL could crimp or tear during forced rotational non-contact loading [150]. The female knee was once thought to possess a smaller notch versus men, lending them to greater vulnerability for traumatic tears [12, 14]. However, other investigators have suggested that increased female risk was based on differences in the ratio between the notch width and width of the femoral condyles (notch width index) [153].

The role of the notch width has remained controversial. Charlton et al. [154] have suggested that the narrower notch width in the female knee simply reflects the smaller diameter ACL within the notch, which still must constrain the same relative loads and stresses as the male ACL. This difference in diameter, along with an increase in creep deformation under sustained loading [155], subsequently renders the ligament to greater injury potential in female athletes. Murshed et al. [156] found no differences in notch width characteristics between the sexes, and Ireland et al. [13] suggested that any individual with a smaller notch width is at higher risk for injury, regardless of sex. More recently investigators have suggested that different regions of the notch may vary in width, where women appear to demonstrate greater narrowing at the base and middle of the notch versus their male counterparts [157]. In response, investigators turned to MRI three-dimensional (3-D) notch volume analysis to better describe differences. Van Eck et al. [157] found that males exhibited a larger 3-D notch volume versus females, furthering the disagreement regarding the role of the notch in female ACL injury risk.

Other morphological characteristics have been examined in terms of their contribution to increased ACL injury risk in females. Investigators have noted that the disparity between the intercondylar axis and joint motion axis of the femur at the knee to be greater in females and compounded in those females with ACL injury history, suggesting its role in ACL tear risk [158]. Investigators discovered that the strain of the anterior–medial bundle of the ACL was increased in females versus males and that this relative strain pattern was positively correlated with ACL cross-sectional area and lateral tibial slope [159]. Finally, Hohmann et al. [160] discovered that ACL-injured females demonstrate a significantly greater posterior tibial plateau slope versus an uninjured control group.

Static knee postural and alignment characteristics have been considered to be factors that could contribute to the woman's greater risk for ACL injury [161]. The Q angle is a clinical measure used to determine the position of the knee in the frontal plane [161]. Livingston and Mandigo [162] compared Q-angles between male and female lower extremities and found no significant sex or right-to-left lower limb differences.

Conversely, Tillman et al. [16] reported that women exceeded men in quadriceps angle (Q-angle) and thigh–foot angle (TF angle) [163]. Yet, the TF angle, which is a measurement of tibia external rotation (toeing out), is not clearly linked to ACL injury [164].

Biomechanical features have been linked to sex-based ACL injury predisposition [164]. Kinematically, differences in knee flexion angle at contact (<30°), tibial rotation in the coronal plane, and frontal plane motion have all been implicated [165]. Investigators have linked reduced contact and peak knee and hip flexion during selected load-bearing functional activities with female ACL injury [166, 167]. Similarly, investigators have observed decreased peak hip abduction in women when cutting during sports [166, 168].

Female athletes have exhibited increased valgus motion in the frontal plane during a landing or cutting maneuver, which may serve as a factor in female ACL injury predisposition [169]. Numerous investigators have observed this behavior [166, 167, 170], along with an increased variability in the valgus motion during the landing and/or cutting sequence [166]. Hewitt et al. [171] reported that these excessive lower limb motions could be reduced through appropriate jump training. Yet, sex-based sagittal [170] and frontal [172] plane movement differences have been disputed, where expected differences did not emerge, and the authors suggest that other factors are at play in producing the increased injury risk for the female ACL. Additionally, other authors have reported increased coronal plane excursion for the hip and knee in women versus men during drop-landing activities [173], producing increased internal rotation of the lower extremity during those activities. Finally, Joseph et al. [174] examined the timing of kinematic occurrences during a landing sequence in men and women. They found that maximal hip adduction, knee valgus, and ankle eversion occurred significantly earlier in women versus men. Moreover, maximal hip adduction and knee valgus occurred before maximal knee flexion in women versus after in men. Maximal ankle eversion occurred earlier in women than in men and women produced a significantly higher angular velocity of knee valgus versus men. The authors concluded that these differences predisposed the women to increased ACL injury.

Increased joint laxity and anterior tibial translation are associated with non-contact ACL injury [164, 175]. Trimble et al. [164] reported that sex and excessive subtalar joint pronation are the only predictors of knee joint laxity. Women exhibit increased anterior knee joint surface translation during extension [176]. This is accompanied by reduced protective hamstring activity during that translational movement [152, 176] that renders the female ACL less protected when exposed to anterior shear forces [177]. In a similar fashion, the female's ACL injury predisposition may be related to the excessive subtalar joint pronation in the ankle [164, 166, 178], which appears to promote the previously discussed increase in tibial internal rotation [178] and tibial anterior translation [164]. This behavior does not appear to relate to genu recurvatum and the tibiofemoral angle [164]. In contrast, other authors have not observed the sex-based differences in subtalar pronation [16].

Differences in kinetic behaviors when cutting or landing have been attributed to increasing the female ACL injury risk [179]. Female athletes have been found to exhibit reduced peak knee flexor moments [180] and increased peak knee valgus moments [167, 180, 181] during cutting tasks. Similarly, Kernozek et al. [167] found increased greater peak vertical and posterior force than men during landing. These altered kinetic behaviors are accompanied by reduced leg stiffness during rapid load-bearing [182], which could translate into a reduced ability to diffuse stress from the ACL [183].

Load management appears to be related to appropriate co-contractive behavior between the quadriceps and hamstrings during cutting and landing. Thus, differences in muscle activation, timing, coordination, and force production may serve as a contributing factor to the female ACL injury predisposition [184, 185]. The woman's difference in muscle activity may begin early, as girls developmentally increase the quadriceps strength disproportionately more than the hamstring strength [186].

While da Fonseca et al. [187] questioned the role of sex in co-contractive disturbances, several authors have suggested that female athletes exhibit greater quadriceps versus hamstring activity during landing and cutting [188–190]. Similarly, women appear to demonstrate prolonged quadriceps recruitment and reduced hamstring activation during the post-contact phase of cutting versus male counterparts [191]. Increased soleus [188] and gastrocnemius [189] activity may contribute to the woman's muscular recruitment differences, while decreased hamstring activity may reduce the woman's ability to decelerate and control tibial translation, internal rotation, and anterior shearing [176, 192]. These differences may be exaggerated by prolonged exercise causing muscular fatigue. Stern et al. [193] found that after exercise, females exhibited significantly less quadriceps motor-evoked potential EMG amplitude compared to males, which may contribute to females' increased risk for ACL injury in response to changes in central nervous system drive capacity.

As a consequence of the woman's differences in kinematic, kinetic, and neuromuscular control strategies, the ACL potentially sustains greater loads with athletic activity. Unfortunately, the female ACL demonstrates different mechanical behaviors amidst these altered strategies. During passive cyclic loading, the female ACL appears to exhibit greater creep versus the male ligament [155]. This difference in ligament creep could compound the previously discussed deleterious mechanical effects, as quadriceps electromyographic activity may increase after ACL creep, while hamstring co-activation is not likely to change [194].

Another factor that appears to interact with the anatomical and biomechanical influences contributing to female ACL injury predisposition revolves around changes in sex hormones. Ovarian sex hormone fluctuations have been related to increased non-contact ACL injury [177, 195]. Estrogen and progesterone receptors have been identified within the substance of the ACL [196], likely responsible for the relationship between peaks in estrogen levels and increased laxity [197]. Exposure to estrogen appears to increase metallomatrix protease activity and decrease fibroblastic activity within the ligament, lending to increased tissue laxity [197]. More recent findings suggest that increased estrogen levels negatively correlate with hamstring rate of force production, suggesting a reduced protective response from that important muscle group during upswings of estrogen [198]. However, correlation between injury risk and specific menstrual phases is controversial at best, since other authors have reported increased ACL injury during the follicular [177, 199] and luteal [199] phases.

### Ankle and Foot

Women appear to be more predisposed to ankle and foot injuries than men in selected populations. Heir [200] found that women in military physical training were at greater risk for developing Achilles tendinopathy and ankle sprains than their male counterparts. Moreover, Knobloc et al. [201] found that symptomatic females suffering Achilles tendinopathy do not benefit as much as symptomatic males from 12 weeks of eccentric training in terms of pain reduction or improvement in functional scores. Structural differences have been noted in the female foot, which demonstrate smaller width and length, as well as specific shape [202]. However, sex differences are not observed in terms of medial longitudinal arch measurements [203] or overall arch height [204].

Women appear to be at greater risk for ankle inversion trauma. Hosea et al. [205] found that female athletes are at greater risk for grade I inversion trauma, where there is disruption of the anterior talofibular ligament. The same authors found no sex-based differences in grade II (anterior talofibular and calcaneofibular ligament involvement) or grade III (the same ligaments plus the posterior talofibular ligament). This predisposition is related not only to the sports in which female athletes participate [206] but also by selected structural differences in the lower extremity. Female athletes' risk of ankle inversion trauma is increased by increased tibia varum and rearfoot eversion while weight bearing [206]. Neuromuscular responses may contribute to the female's predisposition to ankle inversion trauma. Investigators reported that while males demonstrate

decreased peroneus longus reflex amplitude following neuromuscular fatigue, the same reflex in females increased, suggesting the female's reduced protective response during a sudden inversion perturbation [207].

Other structural differences have been noted that add to the female's predisposition to ankle and foot affliction. Women demonstrate decreased cartilage thickness over the talar dome, which is at risk for developing osteochondritis and necrotic changes [208]. Women also appear to demonstrate increased obliquity of the first metatarsal base, resulting in increased metatarsus primus varus and potential increased incidence of clinical hallux valgus [209]. Additionally, women demonstrate increased incidence of hallux rigidus in the first metatarsophalangeal joint, with the vast majority of the subjects demonstrating a flat joint configuration.

Achilles tendinopathy has been attributed to numerous factors, including histochemical, pathomechanical, and neurophysiological influences. In addition, sex has been touted as a factor lending to the development of Achilles tendinopathy, possibly interacting with other factors. Marked deficiencies have been noted for tissue histochemical responsiveness in female rabbit tendons [210]. Sex-based factors may contribute to differences in tendon pathology and response. For example, a female's tendon may experience increased load in response to footwear with hard soles and insufficient rearfoot control or high heels [211], all of which have been associated with increased incidence of tendinopathy. However, this sex-based predilection for Achilles tendinopathy is controversial, where more recent studies have questioned differential female predisposition [212]. Sex-based differences in Achilles tendon properties and pathology may be related to muscle and tendon strength differences, rather than other sex-specific tissue characteristics [213].

#### 3.3.3.3 Spine

##### Cervical

Sex-related anatomical differences in the cervical spine can be observed in the vertebral structure [214, 215], lending to clinically relevant differences in bony processes and the joints they form, as well as the foraminal spaces through which important neurovascular components course. For example, investigators have observed sex differences in dimensions of lower cervical vertebral laminae and pedicles. Rezcallah et al. [214] found that women demonstrated smaller pedicular widths, lengths, and transverse angles at C3 through C7 when compared to men. Similarly, Xu et al. [215] observed smaller laminar height, width, thickness, and angulation in women at levels C2 through C5. The role of sex-based cervical vertebral structure differences is not clear [216]. Yet, men appear to have a smaller vertebral canal-to-body anterior–posterior diameter ratio versus women, potentially predisposing them to a smaller canal in proportion to their overall axial skeletal morphism and decreased incidence of cervical myelopathy [217]. Conversely, women appear to demonstrate greater spinal canal narrowing after whiplash-type injury versus their male counterparts [218], possibly contributing to their increased incidence of whiplash-related disorders [219], as well as the latent clinical sequelae and delayed recovery status post-whiplash trauma [220, 221].

Female cervical zygapophyseal (facet) joints may be at greater risk for injury during a whiplash trauma versus their male counterparts. Excessive segmental translation has been shown to be a potential cause of injury. Simulated rear-impact vehicular accidents using human volunteer subjects showed greater degrees of cervical retraction in women that were unaware at time of rear-end impact [222].

Yoganandan [223] found that the facet articular surfaces in female cadavers were less adequately covered by cartilage than similar specimens in men. In addition, these joints exhibited a greater distance from the dorsal-most region of the facet joint to the location where the cartilage began to appear (called a cartilage gap), potentially lending these joints to greater translation during unanticipated loads. Stemper et al. [224] found that female cadaveric specimens exhibited increased compression in the dorsal region of the facet joint during the early phase of whiplash. These biomechanical behaviors could

predispose the female facets to injury in the subchondral bone during normal physiological and traumatic loads, especially when accompanied by endplate perforations and older age.

Female cervical discs may be at greater risk for failure when exposed to unexpected abnormal loads. Truumees et al. [225] examined the geometric characteristics and loading response of the cartilaginous endplates found in cadaveric cervical discs. They found that the female sex was associated with significantly lower endplate fracture loads when exposed to compression.

### Thoracic

The primary sex-based differences that have been observed in the thoracic spine appear to center around differences in postural alignment. Fundamental sex-based differences have been observed in children and adolescents regarding the extent of the thoracic kyphosis in the sagittal plane. The presence and severity of kyphosis is especially more marked in women [226]. Thoracic kyphosis changes as children age, where the rate of change is greater in women versus men [227]. This change in the kyphotic curve seems to progress in a fashion similar to the lumbar spine lordotic curve during childhood. However, the relationship between the change in kyphosis and lordosis decreases in girls by the age of 15, but not in boys [228].

Of greater interest are the sex-based differences in the development and progression of adolescent idiopathic scoliosis (AIS), where the individual develops a rotatory 3-D deformation in the thoracolumbar spine, especially in the frontal plane. A single thoracic curve is the most common in selected populations, followed by other configurations of single and double curves in the thoracolumbar spine [229]. Investigators have reported increased incidence of AIS in female adolescents [229–231], where female prevalence appears to be genetically coded [232]. Girls appear to be at greater risk for developmental curve progression versus boys, especially in the age prior to the onset of menses [231]. Similarly, girls with scoliosis generally grow faster than girls without the same condition [233]. Yet while an age of more than 15 years, skeletal maturity, postmenarchal status, and a history of spine injury are all associated with the prevalence of back pain in people with AIS, sex, family history of scoliosis, leg length discrepancy, curve magnitude, and spinal alignment are not [234].

Investigators have looked at not only the interactions of physical changes with sex in scoliotic patients but also the role of sex affecting psychosocial factors associated with the condition. Girls with scoliosis seem to be at greater risk for psychosocial stresses, including feelings about poor body development, troubled peer interactions, and health compromising behaviors [230]. Yet, while investigators have evaluated the impact of scoliosis on health-related quality of life (HRQoL), the role of sex in that evaluation is controversial. While Ugwonali et al. [235] discovered that male adolescents scored higher on validated self-report instruments that measured HRQoL, Bunge et al. [236] found no effect of sex, curve type, and curve size on a similar battery of measures. Additionally, adolescents undergoing brace-based management did not appear to score on HRQoL instruments differently from age-adjusted norms.

### Lumbar

Premenopausal women demonstrate decreased bone density in the lumbar vertebrae versus men [237]. Ebbesen et al. [238] found that adult women demonstrate lower vertebral bone mass than age-matched men. Additionally, women exhibited decreased compressive load tolerances, accompanied by increased mechanical stress [238, 239]. However, bony differences may not be limited only to adults. Gilsanz et al. [240] found similar bone mass differences in preadolescent and adolescent girls, who demonstrate lower vertebral bone mass than age-matched boys. However, these investigators reported that these differences were likely related to differences in bone size versus bone density [239, 240]. Naganathan and Sambrook [241] went further to report that volumetric bone density of the third lumbar vertebra did not differ between the sexes, whereas observed differences in areal bone density were likely related to differences in bone size.

An account of sex-based differences in low back pain (LBP) is controversial. However, Korovessis et al. [242] has reported a higher incidence of LBP and dorsal pain (DP) in female youth, especially with those girls involved in sports. However, the structural etiology of sex-based differences in back symptoms has not been fully elucidated. Evidence of differences in intervertebral disc structure or function is scarce. Gruber et al. [243] found that the female sex was a contribution to the cellular proliferation potential within the annulus fibrosis surrounding the disc nucleus pulposus, along with a contribution from increased age, degree of degeneration, and surgical modification. Investigators have found differences in lumbar zygapophyseal facet size, pedicle facet angle, and facet shape [244, 245]. These differences appear to be related to a greater incidence of degenerative anterolisthesis at L4 in women versus their male counterparts [244].

Sex differences are observable in the posture and postural control of the lumbar spine. Norton et al. [246] found a greater lumbar spine lordotic curve in women versus men. O'Sullivan et al. [247] examined the impact of unstable versus stable sitting surfaces on recruitment and control of the superficial lumbar multifidus, transverse fibers of internal oblique, and iliocostalis lumborum pars thoracis. While these investigators found no sex-based electromyographic (EMG) differences, they did observe that women exhibited greater medial–lateral postural sway versus men on an unstable surface. While the role of posture and postural control in the development of LBP is inconclusive, future studies could examine the role of these neuromuscular factors on the development of lumbar pathology.

Investigators have observed sex-based differences in lumbar muscle cross-sectional area and muscle geometry [248]. These differences, coupled with differences in trunk motor control strategies, could have an influence on biomechanical behaviors of the lumbar spine. Women exhibit decreased type II fiber diameter versus men, lending to decreased strength and increased endurance of the lumbar muscle groups [249]. Moreover, women appear to experience greater compressive and anterior–posterior shear loading at the lower lumbar spine [250]. These loading differences appear to be related to altered coactivation of the muscles surrounding the lumbar segments, where women produce greater flexor antagonistic coactivation than men [251]. It is likely that these altered behaviors result in distorted strategies for controlling dynamic spinal loading conditions. For example, Granata et al. [252] examined the effect of sustained flexion postures on protective paraspinal muscle reflexes. These investigators not only observed a detrimental alteration in the reflexive activity after sustained trunk flexion but also found that women demonstrated greater detriments in the protective reflexive response. Moreover, they appear to have decreased stiffness and increased segmental motion in the lumbar spine versus men [253]. These factors added together could lend the female lumbar segments to the development of clinical lumbar instability [254].

### Sacroiliac and Pelvis

Women appear to suffer from pain associated with the sacroiliac joint (SIJ) more frequently than men, most likely associated with anatomical differences and hormonal fluctuations. The incidence of SIJ clinical hypermobility in the joint is greatest between the ages of 18 to 35 years. However, this prevalence appears to be sex-specific, where the SIJ mobility begins to decline at 35 years old in men and 45 years old in women. Thus, SIJ-related pain that is associated with clinical instability could persist in women after 45 years, especially when the individual is on estrogen replacement.

Anatomical changes are seen in the SIJ throughout the course of life, and those changes appear to be different between men and women [255–257]. By the second decade, differences between the sexes are observable [258]. While the male synovial capsule thickens and the joint architecture visibly adapts, the female SIJ soft tissues become more pliable as hormones fluctuate with the onset of menses.

Although the sacral vertebrae start ossifying in the third decade, the mobility of the female SIJ continues to increase, producing a ratio of mobility of approximately 5:1 compared to men.

Pregnancy can increase mobility of the sacroiliac joint 2.5 times, increasing the dynamic movement disparity between women and men [258]. Movement persists in the female SIJ through the fourth and fifth decades, whereas the male SIJ demonstrates a further decline of motion in the same time frame [259, 260]. While complementary ridges and depressions form on the iliac and sacral cartilages and the synovial membranes thicken in both men and women, men appear to be more prone to the development of periarticular osteophytes and sacroiliac bridging, further lending the male SIJ to decreased mobility [258, 261–263].

The external contours of the SI joint articular surfaces are generally a C-shape in men and an L-shape in women, lending the female articulation to greater translation during select situations such as pregnancy and delivery. The joint surfaces at the S1 level are the largest compared to the smaller surfaces at S2 and S3. Each SIJ surface is approximately 17.5 cm [2] in surface area, well suited for absorption and transfer of large forces [264]. The sacroiliac joint itself is found deep within the sacrum and ilium. The iliac cartilage is thin (0.5 mm), bluish, dull, and rough, compared to the sacral cartilage which is thick (3 mm), white, shiny, and smooth [261]. The iliac cartilage is the same relative thickness in both sexes, in contrast to the sacral cartilage which is thicker in women [265].

Women present with SIJ-related pain in the last trimester of pregnancy [266, 267], in response to increased relaxin that changes the stiffness of the elaborate ligament system and produces a hypermobile state in the joint [268, 269]. The ligamentous system of the SIJ enhances stability by increasing the friction in the SI joint and contributes to a self-locking mechanism [270–272]. In addition, the system offers proprioceptive feedback in response to activity due to a rich plexus of articular receptors.

The SIJ ligament system can be divided into four different layers, the most superficial layer being the thoracolumbar fascia to which numerous muscles attach and impose control, including the latissimus dorsi, gluteus maximus, transverse abdominis, and serratus posterior inferior [272]. The next layer associated with the SIJ includes the sacrospinous and the sacrotuberous ligaments that constrain sacral nutation (or anterior sacral rotation about its internal transverse axis of rotation at S2) and control movement of the pubic symphysis on the anterior aspect of the pelvic ring [273, 274].

The long dorsal SI ligament (also known as the longissimus ligament) courses from the posterior superior iliac spine to the inferior lateral sacrum outside the coccyx. This ligament is approximately 2 cm wide and 6 cm long. It is the only ligament that maximally tightens during counter-nutation, lending it to strain and clinical symptoms during a woman's third trimester of pregnancy after the fetus descends [275]. The iliolumbar ligament constrains both SIJ movements and movement of the lower lumbar segments with respect to the sacrum [276, 277]. Along with the dorsal SIJ ligaments that are less developed in females [278], the self-locking mechanism is further enhanced through the constraints imposed by the deep interosseus ligaments, especially during nutation [270–272]. These ligaments are found to be thicker in females [278], and their stiffness decreases under the influence of hormonal changes in the final stages of pregnancy, so that the birthing process can be enabled. Moreover, the SIJ tends to counter-nutate during these stages, where the sacral base tips posterior and opens the pelvic inlet for fetal decent. Counter-nutation reduces ligament constraint and promotes joint hypermobility that can contribute to postpartum pelvic pain [268, 279, 280], which can persist several years after birth [281].

## 3.4 Contemporary Understanding of the Issues

Sexual dimorphism in the human musculoskeletal system is apparent, but more subtle than sex differences often observed in other species. Some musculoskeletal sex differences in humans are present at an early age, while others tend to appear later in life, especially at puberty and menopause. Sex differences in gross skeletal

geometry and specific tissue characteristics are common. For example, women are generally shorter, have less body mass, and have a different general morphological appearance than men. Women tend to have different characteristics of specific bones and skeletal features than men, which have been explained by both genetic and environmental factors. In the pelvis, women tend to have a larger inlet and outlet, greater interacetabular distance, and greater hip width normalized to femur length. In the femur, women tend to have greater femoral anteversion and narrower intercondylar notch features. Additionally, there are several differences in specific characteristics of the femur, such as head diameter and circumference that are relatively strong predictors of sex. In the knee, tibia, and foot, women tend to exhibit greater genu recurvatum, greater quadriceps angle, more lateral patellar alignment, increased tibial torsion, greater tibial slope angles, and more bunions and deformities of the toes. Additionally, women and men appear to have several differences in collagenous, cartilage, and bone tissues, which may predispose women to certain pathologies such as osteoarthritis and osteoporosis later in life. In other collagenous tissues, there are sex differences in collagen thickness, orientation, content, diameter, volume, and metabolism. In cartilage, women tend to have less cartilage volume, thickness, and surface area at specific sites. Additionally, prepubescent girls tend to have slower cartilage accrual rate, and postmenopausal women tend to have greater cartilage degradation than their male counterparts at either age, respectively. In bone tissue, women tend to have a slower accrual rate in youth, less peak bone mass, and slower bone turnover in adulthood as compared to men. Additionally, women tend to have decreased volumetric bone mineral density, less bone area, decreased cortical thickness, plus less compressive and bending strength at some bony sites compared to men, even after correction for anthropometric differences such as height and weight.

Sexual dimorphism can manifest itself by specific differences in each joint system throughout the body, possibly resulting in differences in clinical pathology and symptomology. While differences in subacromial space have been attributed to sex-based differences in clinical impingement at the shoulder, the role of those variations remains controversial. More trustworthy are the female glenohumeral capsular responses that appear to contribute to sex-based differences in the incidence of joint laxity and/or idiopathic capsulitis. Hormonal differences appear to affect tissue changes related to the woman's higher incidence of tendinosis in the lateral elbow tendon structures, while the female predisposition for increased incidence of carpal tunnel syndrome seems to relate to differences in architectural shapes in the wrist and hand especially found around the tunnel. Similarly, architectural differences are at the root of the female predilection for fracture responses at the hip joint, while tissue biomechanical differences accompany architectural distinctions in contributing to the female incidence of acetabular labral tears.

The woman's increased risk for anterior cruciate ligament injury has received special attention in the literature, which has suggested that several factors are responsible for this elevated incidence. Anatomical, hormonal, mechanical, and neurophysiological differences have all been examined, and multiple mechanisms have been proposed. While the femoral intercondylar notch has been examined, its role remains controversial. Similarly, the role of static measures including Q-angle and thigh–foot angle has remained questionable, while differences in joint movement at both the knee and hip during cutting and landing have been deemed partially responsible for sex-based differences in ACL injury. Joint laxity and tibial translation behaviors appear to contribute to this disparity, along with altered joint motion responses in the subtalar joint. Moreover, locomotor control strategy differences are exhibited by female athletes, lending to their heightened predisposition. Finally, the woman's hormonal fluctuations affect not only changes in ACL architecture but also the biomechanical response of the tissue to stress, along with neuromuscular control of the extremities.

Selected sex-based differences have been observed in the structural and mechanical features

of the ankle and foot. These differences appear to contribute to the woman's increased risk for both ankle sprains and Achilles tendinopathy, especially in those who are athletically inclined. The role of cervical spinal architectural differences in female musculoskeletal health is unclear. However, the woman's cervical spine structures that include the facets and intervertebral discs appear to respond more poorly to macrotrauma, such as whiplash. The preadolescent female is more susceptible to developing thoracolumbar postural changes that include excessive kyphosis, lordosis, and/or scoliosis. These differences not only are influenced by physical differences in the vertebrae, articular structures, intervertebral discs, and/or attached musculature but are apparently influenced by psychomotor control and psychobehavioral variations as well. Finally, sacroiliac joint differences between men and women are influenced not only by architectural disparities but additionally by the influence that hormones have on the integrity of the complex capsuloligamentous structures surrounding the joint itself.

## 3.5 Future Directions

Sex differences in musculoskeletal anatomy are evident in gross body structure, regionally and at the tissue level. Much is known about sex-based differences in musculoskeletal anatomy and how these differences manifest in functional and health-related disparities. However, a great deal of information remains unknown. Future research should continue to investigate the relationships among structure, function, and health, especially in relation to sex-based differences. A few specific recommendations for future directions include the need to better understand:

- The influence of sex-based differences on scapular position at rest and during elevation with external impingement of the shoulder
- The influence of sex differences on the relationships among glenohumeral structure, hypermobility, and pathology
- The influence of pathoanatomical and pathophysiological mechanisms on sex-based differences in tendon pathologies such as tenosynovitis and tendinosis
- The comorbid fracture risks associated with chronic bisphosphonate administration used for treating osteoporosis
- The role and influence of sex-based differences in the development of hip neuromuscular control deficits in the development of knee disorders such as anterior knee pain syndrome and ACL injury
- The influence of sex-based differences on posture and postural control in the development of low back pain and lumbar pathology

## 3.6 Conclusion

There exists an abundance of literature that documents sex differences in general body characteristics, skeletal geometry, musculoskeletal tissue characteristics, and joint-specific functional anatomy and pathomechanics. While the musculoskeletal anatomy of men and women is grossly similar, important differences exist that may influence the way in which the general public views and health care professionals respond to women's musculoskeletal health issues.

## References

1. Walker PBM. Chambers science and technology dictionary. Cambridge: W & R Chambers Ltd and Cambridge University Press.
2. Anonymous. Webster's online dictionary. *Internet* [serial online] (2011). Accessed 15 Feb 2012.
3. Smith FW, Smith PA. Musculoskeletal differences between males and females. Sports Med Arthrosc Rev. 2002;10:98–100.
4. Chmielewski T, Ferber R. Rehabilitation considerations for the female athlete. In: Andrews JR, Harrelson GL, Wilk KE, editors. Physical rehabilitation of the injured athlete. 3rd ed. Philadelphia, PA: Saunders; 2004. p. 315–28.
5. Anonymous. Clinical growth charts. CDC gov [serial online] (2006).
6. Anonymous. Sexual dimorphism. Answers com [serial online] (2006).
7. Horton MG, Hall TL. Quadriceps femoris muscle angle: normal values and relationships with gender and selected skeletal measures. Phys Ther. 1989;69:897–901.

8. Kersnic B, Iglic A, Kralj-Iglic V, et al. Determination of the femoral and pelvic geometrical parameters that are important for the hip joint contact stress: differences between female and male. Pflugers Arch. 1996;431:R207–8.
9. Souryal TO, Freeman TR. Intercondylar notch size and anterior cruciate ligament injuries in athletes. A prospective study. Am J Sports Med. 1993;21:535–9.
10. Woodland LH, Francis RS. Parameters and comparisons of the quadriceps angle of college-aged men and women in the supine and standing positions. Am J Sports Med. 1992;20:208–11.
11. Yoshioka Y, Siu DW, Scudamore RA, Cooke TD. Tibial anatomy and functional axes. J Orthop Res. 1989;7:132–7.
12. Shelbourne KD, Davis TJ, Klootwyk TE. The relationship between intercondylar notch width of the femur and the incidence of anterior cruciate ligament tears. A prospective study. Am J Sports Med. 1998;26:402–8.
13. Ireland ML, Ballantyne BT, Little K, McClay IS. A radiographic analysis of the relationship between the size and shape of the intercondylar notch and anterior cruciate ligament injury. Knee Surg Sports Traumatol Arthrosc. 2001;9:200–5.
14. LaPrade RF, Burnett QM. Femoral intercondylar notch stenosis and correlation to anterior cruciate ligament injuries. A prospective study. Am J Sports Med. 1994;22:198–202.
15. Hashemi J, Chandrashekar N, Gill B, et al. The geometry of the tibial plateau and its influence on the biomechanics of the tibiofemoral joint. J Bone Joint Surg Am. 2008;90:2724–34.
16. Tillman MD, Bauer JA, Cauraugh JH, Trimble MH. Differences in lower extremity alignment between males and females. Potential predisposing factors for knee injury. J Sports Med Phys Fitness. 2005;45: 355–9.
17. Gualdi-Russo E. Study on long bones: variation in angular traits with sex, age, and laterality. Anthropol Anz. 1998;56:289–99.
18. LaVelle M. Natural selection and developmental sexual variation in the human pelvis. Am J Phys Anthropol. 1995;98:59–72.
19. Patriquin ML, Loth SR, Steyn M. Sexually dimorphic pelvic morphology in South African whites and blacks. Homo. 2003;53:255–62.
20. Igbigbi PS, Msamati BC, Ng'Ambi TM. Intercondylar shelf angle in adult black Malawian subjects. Clin Anat. 2001;14:254–7.
21. Iscan MY, Shihai D. Sexual dimorphism in the Chinese femur. Forensic Sci Int. 1995;74:79–87.
22. King CA, Iscan MY, Loth SR. Metric and comparative analysis of sexual dimorphism in the Thai femur. J Forensic Sci. 1998;43:954–8.
23. Macho GA. Is sexual dimorphism in the femur a "population specific phenomenon"? Z Morphol Anthropol. 1990;78:229–42.
24. Mall G, Graw M, Gehring K, Hubig M. Determination of sex from femora. Forensic Sci Int. 2000;113:315–21.
25. Purkait R, Chandra H. A study of sexual variation in Indian femur. Forensic Sci Int. 2004;146:25–33.
26. Iscan MY, Miller-Shaivitz P. Determination of sex from the tibia. Am J Phys Anthropol. 1984;64: 53–7.
27. Iscan MY, Yoshino M, Kato S. Sex determination from the tibia: standards for contemporary Japan. J Forensic Sci. 1994;39:785–92.
28. Ruff CB, Hayes WC. Cross-sectional geometry of Pecos Pueblo femora and tibiae–a biomechanical investigation: II. Sex, age, side differences. Am J Phys Anthropol. 1983;60:383–400.
29. Steele DG. The estimation of sex on the basis of the talus and calcaneus. Am J Phys Anthropol. 1976; 45:581–8.
30. Bidmos MA, Asala SA. Discriminant function sexing of the calcaneus of the South African whites. J Forensic Sci. 2003;48:1213–8.
31. Riepert T, Drechsler T, Schild H, Nafe B, Mattern R. Estimation of sex on the basis of radiographs of the calcaneus. Forensic Sci Int. 1996;77:133–40.
32. Hogler W, Blimkie CJ, Cowell CT, et al. A comparison of bone geometry and cortical density at the mid-femur between prepuberty and young adulthood using magnetic resonance imaging. Bone. 2003;33: 771–8.
33. Ashizawa K, Kumakura C, Kusumoto A, Narasaki S. Relative foot size and shape to general body size in Javanese, Filipinas and Japanese with special reference to habitual footwear types. Ann Hum Biol. 1997;24:117–29.
34. Axer H, von Keyserlingk DG, Prescher A. Collagen fibers in linea alba and rectus sheaths. J Surg Res. 2001;96:239–45.
35. Tzaphlidou M. Diameter distributions of collagenous tissues in relation to sex. A quantitative ultrastructural study. Micron. 2001;32:333–6.
36. Vitellaro-Zuccarello L, Cappelletti S, Dal Pozzo Rossi V, Sari-Gorla M. Stereological analysis of collagen and elastic fibers in the normal human dermis: variability with age, sex, and body region. Anat Rec. 1994;238:153–62.
37. Zanze M, Souberbielle JC, Kindermans C, Rossignol C, Garabedian M. Procollagen propeptide and pyridinium cross-links as markers of type I collagen turnover: sex- and age-related changes in healthy children. J Clin Endocrinol Metab. 1997;82:2971–7.
38. Whiting WC, Zernicke RF. Biomechanics of musculoskeletal injury. Champaign, IL: Human Kinetics; 2008.
39. Nordin M, Frankel VH. Basic biomechanics of the musculoskeletal system. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2001.
40. Osakabe T, Hayashi M, Hasegawa K, et al. Age- and gender-related changes in ligament components. Ann Clin Biochem. 2001;38:527–32.
41. Tamir E, Brenner S. Gender differences in collagen diseases. Skinmed. 2003;2:113–7.
42. Ding C, Cicuttini F, Scott F, Glisson M, Jones G. Sex differences in knee cartilage volume in adults: role of body and bone size, age and physical activity. Rheumatology (Oxford). 2003;42:1317–23.

43. Jones G, Glisson M, Hynes K, Cicuttini F. Sex and site differences in cartilage development: a possible explanation for variations in knee osteoarthritis in later life. Arthritis Rheum. 2000;43: 2543–9.
44. Lanyon P, Muir K, Doherty S, Doherty M. Age and sex differences in hip joint space among asymptomatic subjects without structural change: implications for epidemiologic studies. Arthritis Rheum. 2003;48: 1041–6.
45. Larbre JP, Da Silva JA, Moore AR, James IT, Scott DL, Willoughby DA. Cartilage contribution to gender differences in joint disease progression. A study with rat articular cartilage. Clin Exp Rheumatol. 1994;12:401–8.
46. Cicuttini FM, Wluka A, Bailey M, et al. Factors affecting knee cartilage volume in healthy men. Rheumatology (Oxford). 2003;42:258–62.
47. Faber SC, Eckstein F, Lukasz S, et al. Gender differences in knee joint cartilage thickness, volume and articular surface areas: assessment with quantitative three-dimensional MR imaging. Skeletal Radiol. 2001;30:144–50.
48. Mouritzen U, Christgau S, Lehmann HJ, Tanko LB, Christiansen C. Cartilage turnover assessed with a newly developed assay measuring collagen type II degradation products: influence of age, sex, menopause, hormone replacement therapy, and body mass index. Ann Rheum Dis. 2003;62:332–6.
49. Da Silva JA, Larbre JP, Seed MP, et al. Sex differences in inflammation induced cartilage damage in rodents. The influence of sex steroids. J Rheumatol. 1994;21:330–7.
50. Fares JE, Choucair M, Nabulsi M, Salamoun M, Shahine CH, Fuleihan G. Effect of gender, puberty, and vitamin D status on biochemical markers of bone remodeling. Bone. 2003;33:242–7.
51. Nuckley DJ, Eck MP, Carter JW, Ching RP. Spinal maturation affects vertebral compressive mechanics and vBMD with sex dependence. Bone. 2004;35: 720–8.
52. Katzburg S, Lieberherr M, Ornoy A, Klein BY, Hendel D, Somjen D. Isolation and hormonal responsiveness of primary cultures of human bone-derived cells: gender and age differences. Bone. 1999;25:667–73.
53. Khosla S, Melton III LJ, Atkinson EJ, O'Fallon WM, Klee GG, Riggs BL. Relationship of serum sex steroid levels and bone turnover markers with bone mineral density in men and women: a key role for bioavailable estrogen. J Clin Endocrinol Metab. 1998;83:2266–74.
54. Beck TJ, Ruff CB, Shaffer RA, Betsinger K, Trone DW, Brodine SK. Stress fracture in military recruits: gender differences in muscle and bone susceptibility factors. Bone. 2000;27:437–44.
55. Kaptoge S, Dalzell N, Loveridge N, Beck TJ, Khaw KT, Reeve J. Effects of gender, anthropometric variables, and aging on the evolution of hip strength in men and women aged over 65. Bone. 2003;32: 561–70.
56. Schuit SC, Van der KM, Weel AE, et al. Fracture incidence and association with bone mineral density in elderly men and women: the Rotterdam Study. Bone. 2004;34:195–202.
57. Krall EA, Dawson-Hughes B, Hirst K, Gallagher JC, Sherman SS, Dalsky G. Bone mineral density and biochemical markers of bone turnover in healthy elderly men and women. J Gerontol A Biol Sci Med Sci. 1997;52:M61–7.
58. Forwood MR, Bailey DA, Beck TJ, Mirwald RL, Baxter-Jones AD, Uusi-Rasi K. Sexual dimorphism of the femoral neck during the adolescent growth spurt: a structural analysis. Bone. 2004;35:973–81.
59. Mosekilde L. Sex differences in age-related loss of vertebral trabecular bone mass and structure–biomechanical consequences. Bone. 1989;10:425–32.
60. Henry YM, Eastell R. Ethnic and gender differences in bone mineral density and bone turnover in young adults: effect of bone size. Osteoporos Int. 2000;11:512–7.
61. Minisola S, Dionisi S, Pacitti MT, et al. Gender differences in serum markers of bone resorption in healthy subjects and patients with disorders affecting bone. Osteoporos Int. 2002;13:171–5.
62. Smith EL, Smith KA, Gilligan C. Exercise, fitness, osteoarthritis, and osteoporosis. In: Bouchard C, Shephard RJ, Stephens T, Sutton JR, McPherson BD, editors. Exercise, fitness, and health: a consensus of current knowledge. Champaign, IL: Human Kinetics; 1990. p. 517–28.
63. Harrison JE, Chow R. Discussion: exercise, fitness, osteoarthritis, and osteoporosis. In: Bouchard C, Shephard RJ, Stephens T, Sutton JR, McPherson BD, editors. Exercise, fitness, and health: a consensus of current knowledge. Champaign, IL: Human Kinetics; 1990. p. 529–32.
64. Anonymous. Osteoporosis: peak bone mass in women. Osteo org [serial online] (2005).
65. Pandley SK, Shamal S, Kuman S, Shukla VK. Articular branch of the axillary artery and its clinical implication. Nepal Med Coll J. 2003;5:61–3.
66. Bhatia DN, de Beer JF, du Toit DF. Coracoid process anatomy: implications in radiographic imaging and surgery. Clin Anat. 2007;20:774–84.
67. Radas CBPHG. The coracoid impingement of the subscapularis tendon: a cadaver study. J Shoulder Elbow Surg. 2004;13:154–9.
68. Bigliani LU, Morrison DS, April E. The morphology of the acromion and its relationship to rotator cuff tears. Orthop Trans. 1986;10:228.
69. Bigliani LU, Ticker JB, Flatow EL, Soslowsky LJ, Mow VC. The relationship of acromial architecture to rotator cuff disease. Clin Sports Med. 1991;10: 823–38.
70. Berbig R, Weishaupt D, Prim J, Shahin O. Primary anterior shoulder dislocation and rotator cuff tears. J Shoulder Elbow Surg. 1999;8:220–5.
71. Speer KP, Osbahr DC, Montella BJ, Apple AS, Mair SD. Acromial morphotype in the young asymptomatic athletic shoulder. J Shoulder Elbow Surg. 2001; 10:434–7.

72. Getz JD, Recht MP, Piraino DW, et al. Acromial morphology: relation to sex, age, symmetry, and subacromial enthesophytes. Radiology. 1996;199:737–42.
73. Gill TJ, McIrvin E, Kocher MS, Homa K, Mair SD, Hawkins RJ. The relative importance of acromial morphology and age with respect to rotator cuff pathology. J Shoulder Elbow Surg. 2002;11:327–30.
74. Wang JC, Shapiro MS. Changes in acromial morphology with age. J Shoulder Elbow Surg. 1997; 6:55–9.
75. Mahakkanukrauh P, Surin P. Prevalence of osteophytes associated with the acromion and acromioclavicular joint. Clin Anat. 2003;16:506–10.
76. Graichen H, Bonel H, Stammberger T, Englmeier KH, Reiser M, Eckstein F. Sex-specific differences of subacromial space width during abduction, with and without muscular activity, and correlation with anthropometric variables. J Shoulder Elbow Surg. 2001;10:129–35.
77. McKenna L, Straker L, Smith A, Cunningham J. Differences in scapular and humeral head position between swimmers and non-swimmers. Scand J Med Sci Sports. 2011;21:206–14.
78. Gill TJ, Zarins B. Open repairs for the treatment of anterior shoulder instability. Am J Sports Med. 2003;31:142–53.
79. Borsa PA, Sauers EL, Herling DE. Patterns of glenohumeral joint laxity and stiffness in healthy men and women. Med Sci Sports Exerc. 2000;32:1685–90.
80. Hawkins RJ, Mohtadi NG. Controversy in anterior shoulder instability. Clin Orthop Relat Res. 1991; (272):152–61.
81. Kronberg M, Brostrom LA. Humeral head retroversion in patients with unstable humeroscapular joints. Clin Orthop Relat Res. 1990;(260):207–11.
82. Churchill RS, Brems JJ, Kotschi H. Glenoid size, inclination, and version: an anatomic study. J Shoulder Elbow Surg. 2001;10:327–32.
83. Merrill A, Guzman K, Miller SL. Gender differences in glenoid anatomy: an anatomic study. Surg Radiol Anat. 2009;31:183–9.
84. Arkkila PE, Kantola IM, Viikari JS, Ronnemaa T. Shoulder capsulitis in type I and II diabetic patients: association with diabetic complications and related diseases. Ann Rheum Dis. 1996;55:907–14.
85. Omari A, Bunker TD. Open surgical release for frozen shoulder: surgical findings and results of the release. J Shoulder Elbow Surg. 2001;10:353–7.
86. Mengiardi B, Pfirrmann CW, Gerber C, Hodler J, Zanetti M. Frozen shoulder: MR arthrographic findings. Radiology. 2004;233:486–92.
87. Hutchinson JW, Tierney GM, Parsons SL, Davis TR. Dupuytren's disease and frozen shoulder induced by treatment with a matrix metalloproteinase inhibitor. J Bone Joint Surg Br. 1998;80:907–8.
88. Nirschl RP. Tennis elbow tendinosis: pathoanatomy, nonsurgical and surgical management. In: Gordon SLBSJFLJ, editor. Repetitive motion disorders of the upper extremity. Rosemont, IL: American Academy of Orthopaedic Surgeons; 1995. p. 467–78.
89. Svernlov B, Adolfsson L. Non-operative treatment regime including eccentric training for lateral humeral epicondylalgia. Scand J Med Sci Sports. 2001;11:328–34.
90. Winkel D, Matthijs O, Phelps V. Part 2: The knee. Diagnosis and treatment of the lower extremities. Gaithersburg, MD: Aspen Publishers, Inc.; 1997
91. Solveborn SA. Radial epicondylalgia ('tennis elbow'): treatment with stretching or forearm band. A prospective study with long-term follow-up including range-of-motion measurements. Scand J Med Sci Sports. 1997;7:229–37.
92. Ljung BO, Lieber RL, Friden J. Wrist extensor muscle pathology in lateral epicondylitis. J Hand Surg [Br]. 1999;24:177–83.
93. Alfredson H, Ljung BO, Thorsen K, Lorentzon R. In vivo investigation of ECRB tendons with microdialysis technique—no signs of inflammation but high amounts of glutamate in tennis elbow. Acta Orthop Scand. 2000;71:475–9.
94. Richardson JK, Green DF, Jamieson SC, Valentin FC. Gender, body mass and age as risk factors for ulnar mononeuropathy at the elbow. Muscle Nerve. 2001;24:551–4.
95. Tanaka S, Petersen M, Cameron L. Prevalence and risk factors of tendinitis and related disorders of the distal upper extremity among U.S. workers: comparison to carpal tunnel syndrome. Am J Ind Med. 2001;39:328–35.
96. Moghtaderi A, Izadi S, Sharafadinzadeh N. An evaluation of gender, body mass index, wrist circumference and wrist ratio as independent risk factors for carpal tunnel syndrome. Acta Neurol Scand. 2005;112:375–9.
97. McDiarmid M, Oliver M, Ruser J, Gucer P. Male and female rate differences in carpal tunnel syndrome injuries: personal attributes or job tasks? Environ Res. 2000;83:23–32.
98. Mondelli M, Aprile I, Ballerini M, et al. Sex differences in carpal tunnel syndrome: comparison of surgical and non-surgical populations. Eur J Neurol. 2005;12:976–83.
99. Pierre-Jerome C, Bekkelund SI, Nordstrom R. Quantitative MRI analysis of anatomic dimensions of the carpal tunnel in women. Surg Radiol Anat. 1997;19:31–4.
100. Siegel DB, Kuzma G, Eakins D. Anatomic investigation of the role of the lumbrical muscles in carpal tunnel syndrome. J Hand Surg [Am]. 1995;20: 860–3.
101. Richards RS, Bennett JD. Abnormalities of the hook of the hamate in patients with carpal tunnel syndrome. Ann Plast Surg. 1997;39:44–6.
102. Boz C, Ozmenoglu M, Altunayoglu V, Velioglu S, Alioglu Z. Individual risk factors for carpal tunnel syndrome: an evaluation of body mass index, wrist index and hand anthropometric measurements. Clin Neurol Neurosurg. 2004;106:294–9.
103. Massy-Westropp N, Grimmer K, Bain G. The effect of a standard activity on the size of the median nerve

as determined by ultrasound visualization. J Hand Surg [Am]. 2001;26:649–54.
104. Crisco JJ, Coburn JC, Moore DC, Upal MA. Carpal bone size and scaling in men versus in women. J Hand Surg [Am]. 2005;30:35–42.
105. Hamanaka I, Okutsu I, Shimizu K, Takatori Y, Ninomiya S. Evaluation of carpal canal pressure in carpal tunnel syndrome. J Hand Surg [Am]. 1995; 20:848–54.
106. Ham SJ, Kolkman WF, Heeres J, den Boer JA. Changes in the carpal tunnel due to action of the flexor tendons: visualization with magnetic resonance imaging. J Hand Surg [Am]. 1996;21:997–1003.
107. Rempel D, Keir PJ, Smutz WP, Hargens A. Effects of static fingertip loading on carpal tunnel pressure. J Orthop Res. 1997;15:422–6.
108. Chen WS. Median-nerve neuropathy associated with chronic anterior dislocation of the lunate. J Bone Joint Surg Am. 1995;77:1853–7.
109. Seradge H, Jia YC, Owens W. In vivo measurement of carpal tunnel pressure in the functioning hand. J Hand Surg [Am]. 1995;20:855–9.
110. Gelberman RH, Hergenroeder PT, Hargens AR, Lundborg GN, Akeson WH. The carpal tunnel syndrome. A study of carpal canal pressures. J Bone Joint Surg Am. 1981;63:380–3.
111. Buschbacher RM. Mixed nerve conduction studies of the median and ulnar nerves. Am J Phys Med Rehabil. 1999;78:S69–74.
112. Vennix MJ, Hirsh DD, Chiou-Tan FY, Rossi CD. Predicting acute denervation in carpal tunnel syndrome. Arch Phys Med Rehabil. 1998;79:306–12.
113. Tetro AM, Evanoff BA, Hollstien SB, Gelberman RH. A new provocative test for carpal tunnel syndrome. Assessment of wrist flexion and nerve compression. J Bone Joint Surg Br. 1998;80:493–8.
114. Vogt T. Median-ulnar motor latency difference in the diagnosis of CTS. Zeitschr Elektroenzephalogr Diagn verwandte Gebiete. 1995;26:141–5.
115. Glowacki KA, Breen CJ, Sachar K, Weiss AP. Electrodiagnostic testing and carpal tunnel release outcome. J Hand Surg [Am]. 1996;21:117–21.
116. Padula L. A useful electrophysiologic parameter for diagnosis of CTS. Muscle Nerve. 1995;19:48–53.
117. Verghese J, Galanopoulou AS, Herskovitz S. Autonomic dysfunction in idiopathic carpal tunnel syndrome. Muscle Nerve. 2000;23:1209–13.
118. Golovchinsky V. Frequency of ulnar-to-median nerve anastomosis revisited. Electromyogr Clin Neurophysiol. 1995;35:67–8.
119. Szabo RM, Gelberman RH. The pathophysiology of nerve entrapment syndromes. J Hand Surg [Am]. 1987;12:880–4.
120. Kiuru MJ, Pihlajamaki HK, Ahovuo JA. Fatigue stress injuries of the pelvic bones and proximal femur: evaluation with MR imaging. Eur Radiol. 2003;13:605–11.
121. Seeman E. The structural and biomechanical basis of the gain and loss of bone strength in women and men. Endocrinol Metab Clin North Am. 2003;32:25–38.
122. Genser-Strobl B, Sora MC. Potential of P40 plastination for morphometric hip measurements. Surg Radiol Anat. 2005;27:147–51.
123. Wang SC, Brede C, Lange D, et al. Gender differences in hip anatomy: possible implications for injury tolerance in frontal collisions. Annu Proc Assoc Adv Automot Med. 2004;48:287–301.
124. Igbigbi PS. Collo-diaphysial angle of the femur in East African subjects. Clin Anat. 2003;16:416–9.
125. Beck TJ, Ruff CB, Scott Jr WW, Plato CC, Tobin JD, Quan CA. Sex differences in geometry of the femoral neck with aging: a structural analysis of bone mineral data. Calcif Tissue Int. 1992;50:24–9.
126. Crabtree N, Lunt M, Holt G, et al. Hip geometry, bone mineral distribution, and bone strength in European men and women: the EPOS study. Bone. 2000;27:151–9.
127. Korpelainen R, Orava S, Karpakka J, Siira P, Hulkko A. Risk factors for recurrent stress fractures in athletes. Am J Sports Med. 2001;29:304–10.
128. Curtin BM, Fehring TK. Bisphosphonate fractures as a cause of painful total hip arthroplasty. Orthopedics. 2011;34:e939–44.
129. Lo JC, Huang SY, Lee GA, et al. Clinical correlates of atypical femoral fracture. Bone. 2012;51:181–4.
130. Yoon RS, Hwang JS, Beebe KS. Long-term bisphosphonate usage and subtrochanteric insufficiency fractures: a cause for concern? J Bone Joint Surg Br. 2011;93:1289–95.
131. Schilcher J, Michaelsson K, Aspenberg P. Bisphosphonate use and atypical fractures of the femoral shaft. N Engl J Med. 2011;364:1728–37.
132. Horii M, Kubo T, Inoue S, Kim WC. Coverage of the femoral head by the acetabular labrum in dysplastic hips: quantitative analysis with radial MR imaging. Acta Orthop Scand. 2003;74:287–92.
133. Seldes RM, Tan V, Hunt J, Katz M, Winiarsky R, Fitzgerald RH, Jr. Anatomy, histologic features, and vascularity of the adult acetabular labrum. Clin Orthop Relat Res. 2001;(382):232–40.
134. Ferguson SJ, Bryant JT, Ganz R, Ito K. An in vitro investigation of the acetabular labral seal in hip joint mechanics. J Biomech. 2003;36:171–8.
135. Narvani AA, Tsiridis E, Tai CC, Thomas P. Acetabular labrum and its tears. Br J Sports Med. 2003;37:207–11.
136. Kelly BT, Shapiro GS, Digiovanni CW, Buly RL, Potter HG, Hannafin JA. Vascularity of the hip labrum: a cadaveric investigation. Arthroscopy. 2005;21:3–11.
137. Stiris MG. Magnetic resonance arthrography of the hip joint in patients with suspected rupture of labrum acetabulare. Tidsskr Nor Laegeforen. 2001;121: 698–700.
138. Ishiko T, Naito M, Moriyama S. Tensile properties of the human acetabular labrum-the first report. J Orthop Res. 2005;23:1448–53.
139. McCarthy JC, Lee JA. Acetabular dysplasia: a paradigm of arthroscopic examination of chondral injuries. Clin Orthop Relat Res. 2002;(405):122–8.

140. Leunig M, Sledge JB, Gill TJ, Ganz R. Traumatic labral avulsion from the stable rim: a constant pathology in displaced transverse acetabular fractures. Arch Orthop Trauma Surg. 2003;123:392–5.
141. Mintz DN, Hooper T, Connell D, Buly R, Padgett DE, Potter HG. Magnetic resonance imaging of the hip: detection of labral and chondral abnormalities using noncontrast imaging. Arthroscopy. 2005;21: 385–93.
142. Ito K, Minka MA, Leunig M, Werlen S, Ganz R. Femoroacetabular impingement and the cam-effect. A MRI-based quantitative anatomical study of the femoral head-neck offset. J Bone Joint Surg Br. 2001;83:171–6.
143. Kappe T, Kocak T, Bieger R, Reichel H, Fraitzl CR. Radiographic risk factors for labral lesions in femoroacetabular impingement. Clin Orthop Relat Res. 2011;469:3241–7.
144. Mosher TJ, Collins CM, Smith HE, et al. Effect of gender on in vivo cartilage magnetic resonance imaging T2 mapping. J Magn Reson Imaging. 2004; 19:323–8.
145. Csintalan RP, Schulz MM, Woo J, McMahon PJ, Lee TQ. Gender differences in patellofemoral joint biomechanics. Clin Orthop Relat Res. 2002;(402):260–9.
146. Besier TF, Draper CE, Gold GE, Beaupre GS, Delp SL. Patellofemoral joint contact area increases with knee flexion and weight-bearing. J Orthop Res. 2005;23:345–50.
147. Souza RB, Powers CM. Predictors of hip internal rotation during running: an evaluation of hip strength and femoral structure in women with and without patellofemoral pain. Am J Sports Med. 2009; 37:579–87.
148. Cowan SM, Crossley KM. Does gender influence neuromotor control of the knee and hip? J Electromyogr Kinesiol. 2009;19:276–82.
149. Arendt E, Dick R. Knee injury patterns among men and women in collegiate basketball and soccer. NCAA data and review of literature. Am J Sports Med. 1995;23:694–701.
150. Fayad LM, Parellada JA, Parker L, Schweitzer ME. MR imaging of anterior cruciate ligament tears: is there a gender gap? Skeletal Radiol. 2003;32: 639–46.
151. Boden BP, Dean GS, Feagin Jr JA, Garrett Jr WE. Mechanisms of anterior cruciate ligament injury. Orthopedics. 2000;23:573–8.
152. Rozzi SL, Lephart SM, Gear WS, Fu FH. Knee joint laxity and neuromuscular characteristics of male and female soccer and basketball players. Am J Sports Med. 1999;27:312–9.
153. Muneta T, Takakuda K, Yamamoto H. Intercondylar notch width and its relation to the configuration and cross-sectional area of the anterior cruciate ligament. A cadaveric knee study. Am J Sports Med. 1997; 25:69–72.
154. Charlton WP, St John TA, Ciccotti MG, Harrison N, Schweitzer M. Differences in femoral notch anatomy between men and women: a magnetic resonance imaging study. Am J Sports Med. 2002;30:329–33.
155. Sbriccoli P, Solomonow M, Zhou BH, Lu Y, Sellards R. Neuromuscular response to cyclic loading of the anterior cruciate ligament. Am J Sports Med. 2005; 33:543–51.
156. Murshed KA, Cicekcibasi AE, Karabacakoglu A, Seker M, Ziylan T. Distal femur morphometry: a gender and bilateral comparative study using magnetic resonance imaging. Surg Radiol Anat. 2005; 27:108–12.
157. van Eck CF, Martins CA, Vyas SM, Celentano U, van Dijk CN, Fu FH. Femoral intercondylar notch shape and dimensions in ACL-injured patients. Knee Surg Sports Traumatol Arthrosc. 2010;18:1257–62.
158. Hoshino Y, Wang JH, Lorenz S, Fu FH, Tashman S. Gender difference of the femoral kinematics axis location and its relation to anterior cruciate ligament injury: a 3D-CT study. Knee Surg Sports Traumatol Arthrosc. 2012;20:1282–8.
159. Lipps DB, Oh YK, Ashton-Miller JA, Wojtys EM. Morphologic characteristics help explain the gender difference in peak anterior cruciate ligament strain during a simulated pivot landing. Am J Sports Med. 2012;40:32–40.
160. Hohmann E, Bryant A, Reaburn P, Tetsworth K. Is there a correlation between posterior tibial slope and non-contact anterior cruciate ligament injuries? Knee Surg Sports Traumatol Arthrosc. 2011;19 Suppl 1:S109–14.
161. Loudon JK, Jenkins W, Loudon KL. The relationship between static posture and ACL injury in female athletes. J Orthop Sports Phys Ther. 1996;24:91–7.
162. Livingston LA, Mandigo JL. Bilateral Q angle asymmetry and anterior knee pain syndrome. Clin Biomech (Bristol, Avon). 1999;14:7–13.
163. Stuberg W, Temme J, Kaplan P, Clarke A, Fuchs R. Measurement of tibial torsion and thigh-foot angle using goniometry and computed tomography. Clin Orthop Relat Res. 1991;(272):208–12.
164. Trimble MH, Bishop MD, Buckley BD, Fields LC, Rozea GD. The relationship between clinical measurements of lower extremity posture and tibial translation. Clin Biomech (Bristol Avon). 2002;17:286–90.
165. McNair PJ, Marshall RN. Landing characteristics in subjects with normal and anterior cruciate ligament deficient knee joints. Arch Phys Med Rehabil. 1994;75:584–9.
166. McLean SG, Lipfert SW, van den Bogert AJ. Effect of gender and defensive opponent on the biomechanics of sidestep cutting. Med Sci Sports Exerc. 2004;36:1008–16.
167. Kernozek TW, Torry MR, Van Hoof H, Cowley H, Tanner S. Gender differences in frontal and sagittal plane biomechanics during drop landings. Med Sci Sports Exerc. 2005;37:1003–12.
168. Pollard CD, Davis IM, Hamill J. Influence of gender on hip and knee mechanics during a randomly cued cutting maneuver. Clin Biomech (Bristol, Avon). 2004;19:1022–31.

169. Hewett TE, Lindenfeld TN, Riccobene JV, Noyes FR. The effect of neuromuscular training on the incidence of knee injury in female athletes. A prospective study. Am J Sports Med. 1999;27:699–706.
170. Ford KR, Myer GD, Toms HE, Hewett TE. Gender differences in the kinematics of unanticipated cutting in young athletes. Med Sci Sports Exerc. 2005;37:124–9.
171. Hewett TE, Stroupe AL, Nance TA, Noyes FR. Plyometric training in female athletes. Decreased impact forces and increased hamstring torques. Am J Sports Med. 1996;24:765–73.
172. Barber-Westin SD, Noyes FR, Galloway M. Jump-land characteristics and muscle strength development in young athletes: a gender comparison of 1140 athletes 9 to 17 years of age. Am J Sports Med. 2006;34:375–84.
173. Ford KR, Myer GD, Smith RL, Vianello RM, Seiwert SL, Hewett TE. A comparison of dynamic coronal plane excursion between matched male and female athletes when performing single leg landings. Clin Biomech (Bristol, Avon). 2006;21:33–40.
174. Joseph MF, Rahl M, Sheehan J, et al. Timing of lower extremity frontal plane motion differs between female and male athletes during a landing task. Am J Sports Med. 2011;39:1517–21.
175. Ramesh R, Von Arx O, Azzopardi T, Schranz PJ. The risk of anterior cruciate ligament rupture with generalised joint laxity. J Bone Joint Surg Br. 2005; 87:800–3.
176. Hollman JH, Deusinger RH, Van Dillen LR, Matava MJ. Gender differences in surface rolling and gliding kinematics of the knee. Clin Orthop Relat Res. 2003;(413):208–21.
177. Wojtys EM, Ashton-Miller JA, Huston LJ. A gender-related difference in the contribution of the knee musculature to sagittal-plane shear stiffness in subjects with similar knee laxity. J Bone Joint Surg Am. 2002;84-A:10–6.
178. Beckett ME, Massie DL, Bowers KD, Stoll DA. Incidence of hyperpronation in the ACL injured knee: a clinical perspective. J Athl Train. 1992;27: 58–62.
179. Chappell JD, Yu B, Kirkendall DT, Garrett WE. A comparison of knee kinetics between male and female recreational athletes in stop-jump tasks. Am J Sports Med. 2002;30:261–7.
180. Sigward SM, Powers CM. The influence of gender on knee kinematics, kinetics and muscle activation patterns during side-step cutting. Clin Biomech (Bristol, Avon). 2006;21:41–8.
181. McLean SG, Huang X, van den Bogert AJ. Association between lower extremity posture at contact and peak knee valgus moment during sidestepping: implications for ACL injury. Clin Biomech (Bristol, Avon). 2005;20:863–70.
182. Granata KP, Padua DA, Wilson SE. Gender differences in active musculoskeletal stiffness. Part II. Quantification of leg stiffness during functional hopping tasks. J Electromyogr Kinesiol. 2002;12:127–35.
183. Decker MJ, Torry MR, Wyland DJ, Sterett WI, Richard SJ. Gender differences in lower extremity kinematics, kinetics and energy absorption during landing. Clin Biomech (Bristol, Avon). 2003;18: 662–9.
184. Besier TF, Lloyd DG, Ackland TR. Muscle activation strategies at the knee during running and cutting maneuvers. Med Sci Sports Exerc. 2003;35:119–27.
185. Fagenbaum R, Darling WG. Jump landing strategies in male and female college athletes and the implications of such strategies for anterior cruciate ligament injury. Am J Sports Med. 2003;31:233–40.
186. Ahmad CS, Clark AM, Heilmann N, Schoeb JS, Gardner TR, Levine WN. Effect of gender and maturity on quadriceps-to-hamstring strength ratio and anterior cruciate ligament laxity. Am J Sports Med. 2006;34:370–4.
187. da Fonseca ST, Vaz DV, de Aquino CF, Bricio RS. Muscular co-contraction during walking and landing from a jump: comparison between genders and influence of activity level. J Electromyogr Kinesiol. 2006;16:273–80.
188. Padua DA, Carcia CR, Arnold BL, Granata KP. Gender differences in leg stiffness and stiffness recruitment strategy during two-legged hopping. J Mot Behav. 2005;37:111–25.
189. Hurd WJ, Chmielewski TL, Snyder-Mackler L. Perturbation-enhanced neuromuscular training alters muscle activity in female athletes. Knee Surg Sports Traumatol Arthrosc. 2006;14:60–9.
190. Myer GD, Ford KR, Hewett TE. The effects of gender on quadriceps muscle activation strategies during a maneuver that mimics a high ACL injury risk position. J Electromyogr Kinesiol. 2005;15:181–9.
191. Ebben WP, Fauth ML, Petushek EJ, et al. Gender-based analysis of hamstring and quadriceps muscle activation during jump landings and cutting. J Strength Cond Res. 2010;24:408–15.
192. Wojtys EM, Huston LJ, Schock HJ, Boylan JP, Ashton-Miller JA. Gender differences in muscular protection of the knee in torsion in size-matched athletes. J Bone Joint Surg Am. 2003;85-A:782–9.
193. Stern A, Kuenze C, Herman D, Sauer LD, Hart JM. A gender comparison of central and peripheral neuromuscular function after exercise. J Sport Rehabil. 2011;21(3):209–17.
194. Chu D, LeBlanc R, D'Ambrosia P, D'Ambrosia R, Baratta RV, Solomonow M. Neuromuscular disorder in response to anterior cruciate ligament creep. Clin Biomech Bristol, Avon. 2003;18:222–30.
195. Slauterbeck JR, Fuzie SF, Smith MP, et al. The menstrual cycle, sex hormones, and anterior cruciate ligament injury. J Athl Train. 2002;37:275–8.
196. Liu SH, al Shaikh R, Panossian V, et al. Primary immunolocalization of estrogen and progesterone target cells in the human anterior cruciate ligament. J Orthop Res. 1996;14:526–33.
197. Slauterbeck JR, Hardy DM. Sex hormones and knee ligament injuries in female athletes. Am J Med Sci. 2001;322:196–9.

198. Bell DR, Blackburn JT, Norcorss MF, et al. Estrogen and muscle stiffness have a negative relationship in females. Knee Surg Sports Traumatol Arthrosc. 2012;20:361–7.
199. Deie M, Sakamaki Y, Sumen Y, Urabe Y, Ikuta Y. Anterior knee laxity in young women varies with their menstrual cycle. Int Orthop. 2002;26:154–6.
200. Heir T. Musculoskeletal injuries in officer training: one-year follow-up. Mil Med. 1998;163:229–33.
201. Knobloch K, Schreibmueller L, Kraemer R, Jagodzinski M, Vogt PM, Redeker J. Gender and eccentric training in Achilles mid-portion tendinopathy. Knee Surg Sports Traumatol Arthrosc. 2010;18: 648–55.
202. Wunderlich RE, Cavanagh PR. Gender differences in adult foot shape: implications for shoe design. Med Sci Sports Exerc. 2001;33:605–11.
203. Gilmour JC, Burns Y. The measurement of the medial longitudinal arch in children. Foot Ankle Int. 2001;22:493–8.
204. Michelson JD, Durant DM, McFarland E. The injury risk associated with pes planus in athletes. Foot Ankle Int. 2002;23:629–33.
205. Hosea TM, Carey CC, Harrer MF. The gender issue: epidemiology of ankle injuries in athletes who participate in basketball. Clin Orthop Relat Res. 2000;(372):45–9.
206. Beynnon BD, Renstrom PA, Alosa DM, Baumhauer JF, Vacek PM. Ankle ligament injury risk factors: a prospective study of college athletes. J Orthop Res. 2001;19:213–20.
207. Wilson EL, Madigan ML. Effects of fatigue and gender on peroneal reflexes elicited by sudden ankle inversion. J Electromyogr Kinesiol. 2007;17:160–6.
208. Sugimoto K, Takakura Y, Tohno Y, Kumai T, Kawate K, Kadono K. Cartilage thickness of the talar dome. Arthroscopy. 2005;21:401–4.
209. Hyer CF, Philbin TM, Berlet GC, Lee TH. The obliquity of the first metatarsal base. Foot Ankle Int. 2004;25:728–32.
210. Hart DA, Kydd A, Reno C. Gender and pregnancy affect neuropeptide responses of the rabbit Achilles tendon. Clin Orthop Relat Res. 1999;(365):237-246.
211. Wang YT, Pascoe DD, Kim CK, Xu D. Force patterns of heel strike and toe off on different heel heights in normal walking. Foot Ankle Int. 2001;22:486–92.
212. Haims AH, Schweitzer ME, Patel RS, Hecht P, Wapner KL. MR imaging of the Achilles tendon: overlap of findings in symptomatic and asymptomatic individuals. Skeletal Radiol. 2000;29:640–5.
213. Muraoka T, Muramatsu T, Fukunaga T, Kanehisa H. Elastic properties of human Achilles tendon are correlated to muscle strength. J Appl Physiol. 2005;99: 665–9.
214. Rezcallah AT, Xu R, Ebraheim NA, Jackson T. Axial computed tomography of the pedicle in the lower cervical spine. Am J Orthop. 2001;30:59–61.
215. Xu R, Burgar A, Ebraheim NA, Yeasting RA. The quantitative anatomy of the laminas of the spine. Spine. 1999;24:107–13.
216. Lim JK, Wong HK. Variation of the cervical spinal Torg ratio with gender and ethnicity. Spine J. 2004;4:396–401.
217. Hukuda S, Kojima Y. Sex discrepancy in the canal/body ratio of the cervical spine implicating the prevalence of cervical myelopathy in men. Spine. 2002;27:250–3.
218. Pettersson K, Karrholm J, Toolanen G, Hildingsson C. Decreased width of the spinal canal in patients with chronic symptoms after whiplash injury. Spine. 1995;20:1664–7.
219. Versteegen GJ, Kingma J, Meijler WJ, ten Duis HJ. Neck sprain after motor vehicle accidents in drivers and passengers. Eur Spine J. 2000;9:547–52.
220. Suissa S. Risk factors of poor prognosis after whiplash injury. Pain Res Manag. 2003;8:69–75.
221. Hendriks EJ, Scholten-Peeters GG, van der Windt DA, Neeleman-van der Steen CW, Oostendorp RA, Verhagen AP. Prognostic factors for poor recovery in acute whiplash patients. Pain. 2005; 114:408–16.
222. Siegmund GP, Sanderson DJ, Myers BS, Inglis JT. Awareness affects the response of human subjects exposed to a single whiplash-like perturbation. Spine. 2003;28:671–9.
223. Yoganandan N, Knowles SA, Maiman DJ, Pintar FA. Anatomic study of the morphology of human cervical facet joint. Spine. 2003;28:2317–23.
224. Stemper BD, Yoganandan N, Pintar FA. Gender- and region-dependent local facet joint kinematics in rear impact: implications in whiplash injury. Spine. 2004;29:1764–71.
225. Truumees E, Demetropoulos CK, Yang KH, Herkowitz HN. Failure of human cervical endplates: a cadaveric experimental model. Spine. 2003;28: 2204–8.
226. Ryan SD, Fried LP. The impact of kyphosis on daily functioning. J Am Geriatr Soc. 1997;45:1479–86.
227. Fon GT, Pitt MJ, Thies Jr AC. Thoracic kyphosis: range in normal subjects. AJR Am J Roentgenol. 1980;134:979–83.
228. Widhe T. Spine: posture, mobility and pain. A longitudinal study from childhood to adolescence. Eur Spine J. 2001;10:118–23.
229. Chiu YL, Huang TJ, Hsu RW. Curve patterns and etiologies of scoliosis: analysis in a university hospital clinic in Taiwan. Changgeng Yi Xue Za Zhi. 1998;21:421–8.
230. Payne III WK, Ogilvie JW, Resnick MD, Kane RL, Transfeldt EE, Blum RW. Does scoliosis have a psychological impact and does gender make a difference? Spine. 1997;22:1380–4.
231. Soucacos PN, Zacharis K, Soultanis K, Gelalis J, Xenakis T, Beris AE. Risk factors for idiopathic scoliosis: review of a 6-year prospective study. Orthopedics. 2000;23:833–8.
232. Axenovich TI, Zaidman AM, Zorkoltseva IV, Tregubova IL, Borodin PM. Segregation analysis of idiopathic scoliosis: demonstration of a major gene effect. Am J Med Genet. 1999;86:389–94.

233. Loncar-Dusek M, Pecina M, Prebeg Z. A longitudinal study of growth velocity and development of secondary gender characteristics versus onset of idiopathic scoliosis. Clin Orthop Relat Res. 1991;(270):278–2.
234. Ramirez N, Johnston CE, Browne RH. The prevalence of back pain in children who have idiopathic scoliosis. J Bone Joint Surg Am. 1997;79:364–8.
235. Ugwonali OF, Lomas G, Choe JC, et al. Effect of bracing on the quality of life of adolescents with idiopathic scoliosis. Spine J. 2004;4:254–60.
236. Bunge EM, Juttmann RE, de Kleuver M, van Biezen FC, de Koning HJ. Health-related quality of life in patients with adolescent idiopathic scoliosis after treatment: short-term effects after brace or surgical treatment. Eur Spine J. 2006;16(1):83–9.
237. Cheng WC, Yang RS, Huey-Jen HS, Chieng PU, Tsai KS. Effects of gender and age differences on the distribution of bone content in the third lumbar vertebra. Spine. 2001;26:964–8.
238. Ebbesen EN, Thomsen JS, Beck-Nielsen H, Nepper-Rasmussen HJ, Mosekilde L. Age- and gender-related differences in vertebral bone mass, density, and strength. J Bone Miner Res. 1999;14:1394–403.
239. Gilsanz V, Boechat MI, Gilsanz R, Loro ML, Roe TF, Goodman WG. Gender differences in vertebral sizes in adults: biomechanical implications. Radiology. 1994;190:678–82.
240. Gilsanz V, Boechat MI, Roe TF, Loro ML, Sayre JW, Goodman WG. Gender differences in vertebral body sizes in children and adolescents. Radiology. 1994;190:673–7.
241. Naganathan V, Sambrook P. Gender differences in volumetric bone density: a study of opposite-sex twins. Osteoporos Int. 2003;14:564–9.
242. Korovessis P, Koureas G, Papazisis Z. Correlation between backpack weight and way of carrying, sagittal and frontal spinal curvatures, athletic activity, and dorsal and low back pain in schoolchildren and adolescents. J Spinal Disord Tech. 2004;17:33–40.
243. Gruber HE, Norton HJ, Leslie K, Hanley Jr EN. Clinical and demographic prognostic indicators for human disc cell proliferation in vitro: pilot study. Spine. 2001;26:2323–7.
244. Iguchi T, Wakami T, Kurihara A, Kasahara K, Yoshiya S, Nishida K. Lumbar multilevel degenerative spondylolisthesis: radiological evaluation and factors related to anterolisthesis and retrolisthesis. J Spinal Disord Tech. 2002;15:93–9.
245. Masharawi Y, Rothschild B, Salame K, Dar G, Peleg S, Hershkovitz I. Facet tropism and interfacet shape in the thoracolumbar vertebrae: characterization and biomechanical interpretation. Spine. 2005;30:E281–92.
246. Norton BJ, Sahrmann SA, Van Dillen FL. Differences in measurements of lumbar curvature related to gender and low back pain. J Orthop Sports Phys Ther. 2004;34:524–34.
247. O'Sullivan P, Dankaerts W, Burnett A, et al. Lumbopelvic kinematics and trunk muscle activity during sitting on stable and unstable surfaces. J Orthop Sports Phys Ther. 2006;36:19–25.
248. Marras WS, Jorgensen MJ, Granata KP, Wiand B. Female and male trunk geometry: size and prediction of the spine loading trunk muscles derived from MRI. Clin Biomech (Bristol, Avon). 2001;16:38–46.
249. Ng JK, Richardson CA, Kippers V, Parnianpour M. Relationship between muscle fiber composition and functional capacity of back muscles in healthy subjects and patients with back pain. J Orthop Sports Phys Ther. 1998;27:389–402.
250. Marras WS, Davis KG, Jorgensen M. Spine loading as a function of gender. Spine. 2002;27:2514–20.
251. Granata KP, Orishimo KF. Response of trunk muscle coactivation to changes in spinal stability. J Biomech. 2001;34:1117–23.
252. Granata KP, Rogers E, Moorhouse K. Effects of static flexion-relaxation on paraspinal reflex behavior. Clin Biomech (Bristol, Avon). 2005;20:16–24.
253. Brown MD, Holmes DC, Heiner AD, Wehman KF. Intraoperative measurement of lumbar spine motion segment stiffness. Spine. 2002;27:954–8.
254. Cook C, Brismee JM, Sizer Jr PS. Subjective and objective descriptors of clinical lumbar spine instability: a Delphi study. Man Ther. 2006;11:11–21.
255. Bowen V, Cassidy JD. Macroscopic and microscopic anatomy of the sacroiliac joint from embryonic life until the eighth decade. Spine. 1981;6:620–8.
256. Lin WY, Wang SJ. Influence of age and gender on quantitative sacroiliac joint scintigraphy. J Nucl Med. 1998;39:1269–72.
257. Faflia CP, Prassopoulos PK, Daskalogiannaki ME, Gourtsoyiannis NC. Variation in the appearance of the normal sacroiliac joint on pelvic CT. Clin Radiol. 1998;53:742–6.
258. Brooke R. The sacro-iliac joint. J Anat. 1924;58:299–305.
259. Brunner C, Kissling R, Jacob HA. The effects of morphology and histopathologic findings on the mobility of the sacroiliac joint. Spine. 1991;16:1111–7.
260. Bellamy N, Park W, Rooney PJ. What do we know about the sacroiliac joint? Semin Arthritis Rheum. 1983;12:282–313.
261. Sashin D. A critical analysis of the anatomy and the pathologic changes of the sacroiliac joints. J Bone Joint Surg. 1930;12:891–910.
262. Vleeming A, van Wingerden JP, Snijders CJ, Stoeckart R, Dijkstra PF, Stijnen T. Mobility in the SI-joints in the elderly: a kinematic and roentgenologic study. Clin Biomech (Bristol, Avon). 1992;7:170–8.
263. Dar G, Peleg S, Masharawi Y, et al. Sacroiliac joint bridging: demographical and anatomical aspects. Spine. 2005;30:E429–32.
264. Dreyfuss P, Dryer S, Griffin J, Hoffman J, Walsh N. Positive sacroiliac screening tests in asymptomatic adults. Spine. 1994;19:1138–43.

265. Salsabili N, Valojerdy MR, Hogg DA. Variations in thickness of articular cartilage in the human sacroiliac joint. Clin Anat. 1995;8:388–90.
266. Kristiansson P, Svardsudd K. Discriminatory power of tests applied in back pain during pregnancy. Spine. 1996;21:2337–43.
267. Snijders CJ, Seroo JM, Snijder JGT, Hoedt HT. Change in form of the spine as a consequence of pregnancy. Digest of the 11th Int Conf Med Biol Eng. Ottawa: Conference Committee; 1976, pp. 670–1.
268. Mens JM, Vleeming A, Stoeckart R, Stam HJ, Snijders CJ. Understanding peripartum pelvic pain. Implications of a patient survey. Spine. 1996;21: 1363–9.
269. Rathmell JP, Viscomi CM, Bernstein IM. Managing pain during pregnancy and lactation. in *Practical management of pain*, 3rd edn. ed. by P. Raj (Mosby, Inc, St. Louis, 2000), pp. 196–211.
270. Gerlach UJ, Lierse W. Functional construction of the sacroiliac ligamentous apparatus. Acta Anat (Basel). 1992;144:97–102.
271. Lee D. Biomechanics of the lumbo-pelvic-hip complex. An approach to the examination and treatment of the lumbo-pelvic-hip region. New York: Churchill Livingstone; 1999. p. 43–72.
272. Vleeming A, Pool-Goudzwaard AL, Stoeckart R, van Wingerden JP, Snijders CJ. The posterior layer of the thoracolumbar fascia. Its function in load transfer from spine to legs. Spine. 1995;20:753–8.
273. Kapandji IA. The physiology of the joints. Edinburgh: Churchill Livingstone; 1974.
274. Williams PL. Gray's anatomy. 38th ed. London: Churchill Livingstone; 1995.
275. Vleeming A, Pool-Goudzwaard AL, Hammudoghlu D, Stoeckart R, Snijders CJ, Mens JM. The function of the long dorsal sacroiliac ligament: its implication for understanding low back pain. Spine. 1996;21:556–62.
276. Luk KD, Ho HC, Leong JC. The iliolumbar ligament. A study of its anatomy, development and clinical significance. J Bone Joint Surg Br. 1986;68: 197–200.
277. Pool-Goudzwaard AL, Kleinrensink GJ, Snijders CJ, Entius C, Stoeckart R. The sacroiliac part of the iliolumbar ligament. J Anat. 2001;199:457–63.
278. Steinke H, Hammer N, Slowik V, et al. Novel insights into the sacroiliac joint ligaments. Spine (Phila Pa 1976). 2010;35:257–63.
279. Ostgaard HC. Assessment and treatment of low back pain in working pregnant women. Semin Perinatol. 1996;20:61–9.
280. Damen L, Buyruk HM, Guler-Uysal F, Lotgering FK, Snijders CJ, Stam HJ. The prognostic value of asymmetric laxity of the sacroiliac joints in pregnancy-related pelvic pain. Spine. 2002;27: 2820–4.
281. Noren L, Ostgaard S, Johansson G, Ostgaard HC. Lumbar back and posterior pelvic pain during pregnancy: a 3-year follow-up. Eur Spine J. 2002;11: 267–71.

# The Human Menstrual Cycle

4

Reid L. Norman

**Abstract**

Regular menstrual cycles are the result of predictable cyclic changes in reproductive hormones produced by the pituitary and ovary. A few neurons at the base of the brain in the hypothalamus provide the ongoing stimulus, GnRH pulses, for the synthesis and secretion of LH and FSH which, in turn, stimulate the ovaries to produce the steroids estrogen and progesterone and the small peptides inhibin and activin. It is the feedback relationships between the pituitary and ovarian hormones that provide the basis for the cyclic function of the reproductive system. If fertilization occurs, there is the possibility of a pregnancy which will suppress menstrual cycles for the duration of the pregnancy. Nursing or lactation can also suppress the ovarian cycles, but should not be relied on for birth control. The negative feedback relationship between the ovarian steroids and pituitary gonadotropins is the basis by which hormonal birth control works. Synthetic steroid hormones that can be administered orally suppress the secretion of LH and FSH and thus interrupt the growth and development of ovarian follicles.

**Keywords**

Hypothalamus • Pituitary • Ovary • LH • FSH • Estradiol • Progesterone • Inhibin • Negative feedback • Pregnancy

## 4.1 Learning Objectives

After completing this chapter, you should have an understanding of:

- The hormonal changes that occur throughout a menstrual cycle
- Female reproductive hormones and where they are produced

R.L. Norman, Ph.D. (✉)
Department of Pharmacology and Neuroscience, Texas Tech University Health Sciences Center School of Medicine, 3601 4th St, Lubbock, TX, USA
e-mail: reid.normna@ttuhsc.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_4, © Springer Science+Business Media New York 2014

- How secretion of pituitary and ovarian hormones is regulated
- How birth control pills work

## 4.2 Introduction

Menstruation is the result of a complex series of hormonal events that causes the growth, demise, and the eventual sloughing off of the uterine lining and discharge through the vagina. Ultimately, the menstrual cycle is a reflection of cyclic changes in secretion of steroid hormones (estrogen and progesterone) from the ovary. The ovarian cycle in women and other female primates is coordinated by a series of chemical signals between the brain, pituitary, and ovary. The most conspicuous sign that occurs as a result of these events is menstruation or the monthly (usually) shedding of blood and cellular debris through the vagina. Monthly menstruation is a sign of regular ovulation, and cycles that are prolonged or not regular suggest that ovarian function is disrupted. Not only does the hypothalamic-pituitary-ovarian (HPO) axis need to function normally for regular cycles, but pathology of other endocrine organs such as the thyroid and adrenal can also result in menstrual cycle disturbances.

The environment within the body (internal milieu) is under tight homeostatic regulation and, in the absence of genetic defects or disease, is kept exceedingly constant. It is this internal homeostatic regulation that allows us to live in a widely fluctuating external environment with relative ease. However, there are environmental factors that can negatively impact reproduction, the foremost of these being availability of food [1]. Reduced energy availability (nutritional stress) is thought to be the main factor in menstrual irregularity [2, 3]. During times when food is limited, physiological functions not necessary for immediate survival, such as growth and reproduction, are put on hold until energy is again readily available. Along with nutritional stress, psychological and physical stress may also influence reproductive cycles. In times of social stress, such as war, there may be menstrual cycle disturbances in 50 % of sexually mature women [4].

## 4.3 Research Findings

### 4.3.1 How Is the Brain Involved in Reproduction?

At the transition (puberty) between childhood and adulthood, the initial signal that activates the reproductive system is thought to originate in the brain. It is not clear what event actually initiates the process, but it is the increased secretion of gonadotropin-releasing hormone (GnRH) from a few thousand neurons in the hypothalamus that begins the process of sexual maturation. GnRH is a small peptide (10 amino acids in length) produced in neurons and released from the base of the brain in pulses into a set of specialized vessels, the pituitary portal system, and travels a very short distance to the pituitary gland. These pulses of GnRH occur at about hourly intervals during the first half of the menstrual cycle and cause the release of LH and FSH from the pituitary. We cannot reliably measure GnRH in the peripheral circulation, but there is a very high coincidence of LH pulses in the circulation that follow GnRH pulses in the portal system [5, 6]. Therefore, because LH secretion is a faithful indicator of GnRH release, we can analyze GnRH secretion by measuring LH in the peripheral circulation. Even so, it has been difficult to determine if the amplitude of GnRH secretion changes throughout the cycle, but there is general agreement that the frequency of GnRH release is highest in the follicular phase and slows down in the luteal phase when progesterone is present.

### 4.3.2 Pituitary Hormones: Luteinizing Hormone and Follicle-Stimulating Hormone

In response to GnRH pulses, luteinizing hormone (LH) and follicle-stimulating hormone (FSH) are secreted from the gonadotroph cell in the pituitary. Gonadotroph cells can make either FSH or LH or both. FSH and LH are glycoprotein hormones composed of amino acids and sugars. They are released from the pituitary and travel

through the circulatory system where they bind to specific receptors on the cell surface of specific cells in the ovary in females and the testis in males. The secretion of FSH is regulated by GnRH, estrogen, progesterone, activin, and inhibin. Activin stimulates and inhibin suppresses FSH release. Activin and inhibin are protein hormones secreted by the ovary. Secretion of LH is regulated primarily by GnRH, estrogen, and progesterone. Both FSH and LH can easily be measured in the peripheral circulation, and after puberty, the circulating concentration of these hormones is a useful diagnostic tool for the clinician.

### 4.3.3 The Ovaries Secrete Steroid Hormone and Release Ova

The ovaries are located in the pelvis, a long distance from the pituitary. LH and FSH are released from the pituitary and travel through the circulatory system, and although almost all cells in the body are exposed to these hormones, they only bind to those cells that have the specific LH and FSH receptors on their surface. How these hormones interact with cells in the ovary is an intricate and fascinating story, but too detailed to describe here. However, the main points regarding the cyclic changes in the ovary are important for understanding the menstrual cycle. At the beginning of the cycle (day 1 of menstruation), FSH is elevated, and this stimulates several follicles (eight to ten) to begin their final stages of growth and development for ovulation. After a few days, one of these follicles becomes the dominant follicle and is destined to be the one that will ovulate or release the mature ovum (egg) into the fallopian tube. As the dominant follicle grows, cells within that structure called granulosa cells release estrogen into the circulation, and estrogen levels increase throughout the first 14 days of the cycle (follicular phase). Estrogen causes growth of the lining of the uterus (endometrium) in preparation for possible implantation of the developing embryo if fertilization occurs. High levels of estrogen are produced when the follicle is ready to ovulate, and this is the signal to the brain and pituitary to release LH to cause ovulation. When the ovum is released from the follicle, the granulosa cells within the follicle transform into luteal cells under the influence of LH. The luteal (yellow) cells primarily make progesterone, the dominant hormone during the luteal phase. Progesterone from the corpus luteum causes additional changes in the uterus in preparation for implantation of the embryo and is a crucial hormone for the maintenance of pregnancy.

### 4.3.4 The Uterus and Fallopian Tubes

The uterus (womb) is located in the pelvis and is the site of implantation of the fertilized egg after it develops into a blastocyst. The uterus is composed of an outer muscular layer (myometrium) and a lining (endometrium) that undergoes dramatic changes in response to estrogen and progesterone during the menstrual cycle. At the end of the menstrual cycle, the endometrium degenerates and is shed through the vagina as the menstrual flow. As the new cycle begins and the follicle begins to secrete estrogen, the endometrium begins to proliferate and thicken. Hence, the follicular phase of the cycle is also referred to as the proliferative phase. After ovulation, the endometrium becomes secretory in the final preparation for implantation and pregnancy. The luteal phase is sometimes called the secretory phase. If fertilization and implantation do not occur, progesterone and estrogen secretion diminish, and the endometrium degenerates and is shed. A new crop of follicles begins to develop and the cycle begins again.

### 4.3.5 Feedback Relationships

The hypothalamus, pituitary, and ovary all make and release chemical messengers (reproductive hormones) and communicate with each other with these hormones. The control of the ovarian cycle is complicated, but a few key points regarding the feedback relationships between the pituitary and ovarian hormones are important for understanding how the cycles are maintained.

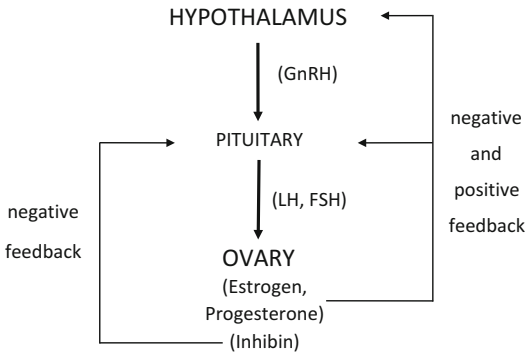



**Fig. 4.1** This figure shows the feedback relationships between the ovarian hormones and the hypothalamic-pituitary axis. Steroid (estrogen and progesterone) and protein (inhibin) hormones produced by the ovary regulate the secretion of GnRH from the hypothalamus and LH and FSH from the pituitary to provide the appropriate stimulation for follicular development and ovulation. These feedback relationships are the basis for cyclic ovarian function

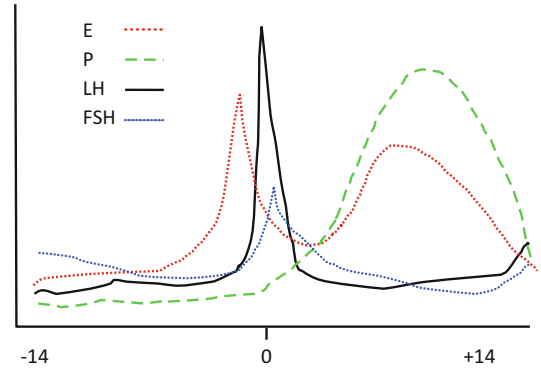



**Fig. 4.2** Changing levels of pituitary (LH, FSH) and ovarian hormones (E, P) throughout the menstrual cycle plotted in reference to the day of the ovulatory LH peak. *E* estradiol, *P* progesterone, *LH* luteinizing hormone, and *FSH* follicle-stimulating hormone

As we stated before, at the beginning of the cycle, a few follicles begin to develop under the stimulation of FSH. FSH is elevated at the beginning of the cycle because estrogen, progesterone, and inhibin are low. These hormones from the ovary control FSH secretion, and if the circulating levels of these hormones are low, FSH rises. As the ovarian follicle begins to mature, estrogen and inhibin gradually increase in the circulation and maintain the release of both FSH and LH from the pituitary at a level that will continue the process of follicular maturation. This relationship where LH and FSH are kept in check by the ovarian hormones they stimulate is called negative feedback (Fig. 4.1). Around day 12 of the cycle when the follicle matures and is ready to release the ovum, granulosa cells in the follicle produce levels of estrogen sufficient to trigger the mid-cycle or ovulatory release of LH. This ovulatory surge of LH causes the follicle to rupture and release the ovum. This release of LH triggered by high levels of estrogen is called positive feedback and only occurs at mid-cycle when estrogen levels have exceeded a threshold level for several hours. After ovulation, the corpus luteum (yellow body) makes progesterone, estrogen, and inhibin, and these hormones keep LH and FSH secretion in check. The corpus luteum has about a 2-week lifespan, and unless a pregnancy is established, the corpus luteum will die. At the end of the cycle, estrogen and progesterone fall to low levels, releasing the negative feedback inhibition so that FSH levels in the blood increase and a new group of follicles begin to grow (see Fig. 4.2 for hormonal changes throughout the cycle). These negative and positive feedback signals keep the reproductive hormones in balance and are the basis by which the ovarian cycle is controlled.

### 4.3.6 Pregnancy and Lactation

When a woman becomes pregnant, menstrual cycles cease because elevated levels of estrogen and progesterone suppress pituitary LH and FSH release and new follicles do not mature. Levels of progesterone and estrogen are initially supported by a hormone from the embryo called human chorionic gonadotropin or hCG. This is the hormone that is measured in pregnancy tests. After a woman delivers and begins to nurse her child, the hormone prolactin is elevated by the suckling stimulus, and this can also suppress follicular development and result in the absence of menstrual cycles. This is called lactational amenorrhea and is a common experience, particularly when the nutritional needs of the child are not supplemented and nursing is the only source of energy. In primitive societies where children

were nursed for several years, lactational amenorrhea served as an effective method of birth control [7]. However, it is not a reliable form of birth control and should not be relied on as such.

Regardless of self-reported sexual activity, pregnancy should always be considered when investigating the cause of amenorrhea in a woman of childbearing age because it is the most common cause of that condition.

### 4.3.7 Hormonal Birth Control

There are several different birth control methods that use different strategies for controlling fertility. One of these methods, hormonal birth control or oral contraceptives, uses synthetic steroid hormones and the inherent negative feedback system to suppress gonadotropin (LH and FSH) release. These hormonal methods inhibit growth and development of follicles so that ovulation and pregnancy do not occur. Birth control pills contain either a progestin (a compound that acts like progesterone) alone or a combination of an estrogen and a progestin. Progestin-alone pills do not block ovulation but affect the lining of the uterus so that there is a hostile environment for implantation and also retard sperm penetration into the uterus. The steroids in the combination pill suppress LH and FSH release and prevent follicular development and ovulation. The steroid regimen in the combination pill more closely mimics the changes in estrogen and progesterone that occur in the menstrual cycle than does the progestin-alone pill. These combination pills are given so that women who want to have regular menstrual cycles can do so. Modern birth control pills have a very low risk of side effects and are very effective.

## 4.4 Contemporary Understanding of the Issues

Although our basic understanding of the physiology of the ovarian and menstrual cycle is sufficient for management of many clinical problems, we have a long way to go before we have a complete understanding of the genetic and molecular details of the communication between the brain, pituitary, and ovary that result in regular cycles.

## 4.5 Future Directions

One of the most pressing health issues of our time is the epidemic of obesity in the western world. Overweight and obese individuals have significantly more health risks than do normal-weight individuals. Overweight individuals have an increased risk for type 2 diabetes, cardiovascular disease, and joint problems. However, even overweight individuals who are physically active are much healthier than those who do not exercise. The dramatic increase in obesity, particularly in young people, has stimulated new research into the physiology of fat. Fat produces many proteins, some of which can impact the reproductive system and alter the function of the metabolic and immune systems as well.

## 4.6 Concluding Remarks

Regular menstrual cycles are the result of predictable cyclic changes in reproductive hormones produced by the pituitary and ovary. A few neurons at the base of the brain in the hypothalamus provide the ongoing stimulus, GnRH pulses, for the synthesis and secretion of LH and FSH which stimulate the ovary to produce the ovarian steroids estrogen and progesterone and the small peptides inhibin and activin. It is the feedback relationships between the pituitary and ovarian hormones that provide the basis for the cyclic function of the reproductive system. If fertilization occurs, there is the possibility of a pregnancy which will suppress menstrual cycles for the duration of the pregnancy. Nursing or lactation can also suppress the ovarian cycles, but should not be relied on for birth control. The negative feedback relationship between the ovarian steroids and pituitary gonadotropins is the basis by which hormonal birth control works. Synthetic steroid hormones that can be administered orally suppress the secretion of LH and FSH and thus interrupt the growth and development of ovarian follicles.

## References


1. Bronson FH. Mammalian reproductive biology. Chicago: University of Chicago Press; 1989.
2. Loucks AB, Thuma JR. Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. J Clin Endocrinol Metab. 2003;88:297–311.
3. Loucks AB, Redman LM. The effect of stress on menstrual function. Trends Endocrinol Metab. 2004;15:466–71.
4. Whitacre FE, Barrera B. War amenorrhea. JAMA. 1944;124:399–403.
5. Levine JE, Pau KY, Ramirez VD, et al. Simultaneous measurement of luteinizing hormone-releasing hormone and luteinizing hormone release in unanesthetized, ovariectomized sheep. Endocrinology. 1982;111:1449–55.
6. Clarke IJ, Cummins JT. The temporal relationship between gonadotropin releasing hormone (GnRH) and luteinizing hormone (LH) secretion in ovariectomized ewe. Endocrinology. 1982;111:1737–9.
7. Short RV. Lactational infertility in family planning. Ann Med. 1993;25:175–80.

# Abnormal Menstrual Cycles

5

Reid L. Norman

**Abstract**

Relevant for female athletes in general and especially in adolescent female athletes are the long-term consequences of insufficient energy intake that results in menstrual cycle disturbances and low bone mineral density. Prolonged exercise as practiced by elite athletes can lead to serious, long-term health problems if diet is not adjusted to compensate for energy expenditure. A good barometer of how energy intake matches energy expenditure in female athletes is menstrual function. Eumenorrhea is the term used to describe normal length, regular menstrual cycles. Oligomenorrhea refers to infrequent menses or menstrual cycles that occur inconsistently at intervals of 39–90 days. Amenorrhea is the absence of menstrual cycles in a nonpregnant, sexually mature woman. When menstrual cycles become disordered as a result of prolonged or excessive exercise, there very well may be an issue related to insufficient energy intake, and consultation of a health care provider knowledgeable in sports medicine is strongly advised to correct the situation. The standard curriculum for high school and college coaches of female athletes should include the health consequences of exercise-induced menstrual function and specific methods of prevention and treatment.

**Keywords**

Eumenorrhea • Oligomenorrhea • Amenorrhea • PCOS • Hirsutism • Anorexia nervosa

## 5.1 Learning Objectives

After completing this chapter, you should have an understanding of:

- The terminology for normal and disrupted menstrual cycles
- Some of the causes of menstrual dysfunction

R.L. Norman (✉)
Department of Pharmacology and Neuroscience, Texas Tech University Health Sciences Center School of Medicine, 3601 4th St, Lubbock, TX 79430, USA
e-mail: reid.normna@ttuhsc.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_5, 

- The symptoms associated with the female athlete triad
- The consequences of prolonged menstrual cycle dysfunction

## 5.2 Introduction

Regular menstrual cycles in young women reflect cyclic ovarian activity and generally are associated with a healthy lifestyle. When a woman experiences a change from regular, cyclic menstrual function to irregular or acyclic function, that change is an indication of either a pregnancy or of some underlying pathology, and the cause for the change in cyclic function should be determined by a health care professional as soon as possible.

To communicate effectively with your peers and students regarding menstrual cycles, you should know and understand normal and abnormal menstrual function and the terminology describing that function. Although there is no complete agreement on the strict definition of all of the following terms, the list below is a reasonable approximation of these definitions (from [1]). These words describing menstrual function are used widely in the scientific literature, but you should assume that the average lay person will probably not be aware of the meaning of these words. Therefore, to avoid misunderstanding and confusion, you should define what you mean when you use these terms in discussion of menstrual problems.

### 5.2.1 Eumenorrhea (Regular)

This refers to menstrual cycles that occur regularly at intervals of 25–38 days. These cycle lengths are generally observed in sexually mature women except during pregnancy and during the pubertal and the perimenopausal (around the time of menopause; this can be several years before the actual cessation of menstrual function) transitions when menstrual cycles are more variable.

### 5.2.2 Oligomenorrhea (Irregular)

This term refers to infrequent menses or menstrual cycles that occur inconsistently at intervals of 39–90 days, typically with only a few periods a year.

### 5.2.3 Amenorrhea (Acyclic)

This is when menstrual cycles occur at intervals of greater than 90 days or when there is the complete absence of menstruation. There are two types of amenorrhea that are defined based on whether the woman has experienced previous menstrual cycles.

#### 5.2.3.1 Primary Amenorrhea

This is when a young woman has not experienced menarche by age 15. In other words, a girl has primary amenorrhea if she has not had her first period by the age of 15.

#### 5.2.3.2 Secondary Amenorrhea

This is the absence of a menstrual period for six consecutive times or months after menarche has occurred. It is estimated that as high as 5 % of the adult women in the United States experience secondary amenorrhea [2] sometime in their reproductive life. There are a number of adjectives to describe the causes of amenorrhea such as dietary, emotional, jogger's, postpartum, and lactational (nursing), but these will be avoided for the most part. However, the term used by clinicians to describe amenorrhea caused by exercise or stress is functional hypothalamic amenorrhea (FHA) and is used widely in the clinical and scientific literature.

## 5.3 Research Findings

### 5.3.1 Eumenorrhea

In Chap. 9 we discussed the normal menstrual cycle so we will not repeat that here.

### 5.3.2 Oligomenorrhea

This term actually means "few menses." Although very light menstruation is sometimes referred to as oligomenorrhea, medical professionals have a more narrow definition, applying this term only to the frequency of menstrual periods. There are many reasons for irregular menstruation, but the most frequent cause is what clinicians refer to as PCOS or polycystic ovary syndrome [3] which affects approximately 6 % of women [4]. Low energy availability is another cause of oligo- and amenorrhea [5] and is probably an important consideration for elite high school- and college-age female athletes, particularly in those sports where body image is considered important. Female athletes will often restrict their food intake to maintain a desired body image even though they are expending considerable energy training for their sport. This can result in an energy deficit (low energy availability) sufficient to inhibit menstrual function. Psychological stress is another factor to consider, although it is hard to separate from other causes.

#### 5.3.2.1 PCOS

PCOS syndrome was originally described by Stein and Leventhal [6] and its pathophysiology is still poorly understood. This syndrome is associated with irregular menstruation, increased secretion of androgens, and the lack of ovulation (anovulation). The diagnosis of PCOS, as defined by the Rotterdam workshop [7], requires two of the following three criteria: (1) infrequent or lack of ovulation, (2) clinical signs of excess androgen, and (3) polycystic ovaries with ultrasound. The typical symptoms that women who have this syndrome experience are irregular menstruation, infertility, and some evidence of increased androgen secretion or hyperandrogenism. Menstrual irregularity may be difficult to document without charting of the cycle for several months. This can easily be done by keeping track on a calendar of the days when bleeding occurs. Excessive bleeding can also be documented this way. Infertility is a complaint more associated with married women who wish to become pregnant and not generally a concern of single, college-age women. Physical symptoms of increased androgen secretion in women are acne and/or oily skin and increased hair growth on the face, back, between the breasts, upper arms, upper and lower abdomen, and inner thighs [8]. However, a woman's perception of hirsutism (increased facial and body hair) may be altered if other women in the family or in the cultural community are hirsute, and therefore, this may not be of particular concern to her. Another concern regarding this syndrome is that it is associated with a high frequency of insulin resistance and increased risk for type 2 diabetes [9], hypertension, and cardiovascular disease [10]. Because early diagnosis could result in amelioration or prevention of the serious consequences and financial burden of this disease [1], it is recommended that women with menstrual cycle disturbances seek medical advice promptly.

#### 5.3.2.2 Low Energy Availability

Low energy availability can result from several different behaviors, and often, there is more than one contributing factor to disrupted cycles resulting from reduced availability of energy. Disrupted menstrual function due to intense exercise, such as that found in elite or even highly competitive female athletes, coupled with restricted food intake is not uncommon in high school and college athletes. Disruption of pulsatile LH secretion in adolescent females is more sensitive to low energy availability than in females who are older [11]. In a recent review of the literature on energy availability and infertility by Loucks [12], this concept of a gradual decline in dependence of LH secretion on energy availability as a woman matures explains the greater prevalence of menstrual disorders in adolescent women and the lower success in restoring menstrual cycles in younger anorexia nervosa patients. The total amount of energy available is partitioned by the body for basic metabolic needs necessary for survival, for locomotion, and for growth and reproduction. If energy intake is restricted below the level needed to support all of these biological processes, those that are necessary for growth and reproduction slow or cease completely.

This is an oversimplification of the idea that reproduction in animals is initiated only when there is sufficient energy to support the successful completion of that process [13], but the basic idea is that reproduction requires a lot of energy and that natural selection favors those females with the ability to allocate energy resources so that survival is not compromised by unsuccessful attempts to reproduce. To accomplish this task of successfully reproducing and surviving at the same time, animals have developed two capabilities: (1) they have some internal way to measure how much energy they take in and how much energy they are using and (2) they use environmental cues to predict the availability of food. This latter capability is particularly important to mammals that have a long pregnancy. Change in day length is one of the most common environmental factors animals use to predict the availability of food, and most animals give birth in the spring when food will be available for several months. As will be discussed in Chap. 11, psychological stress is an important environmental factor that affects reproduction.

#### 5.3.2.3 Other Causes

Other causes of menstrual cycle disturbances are hypothyroidism, elevated levels of prolactin (hyperprolactinemia), Crohn's disease, and eating disorders, and sometimes, there is no known cause (idiopathic).

### 5.3.3 Amenorrhea

Amenorrhea literally means "the absence of menses." The complete absence of menstrual cycles in a nonpregnant, sexually mature woman is a symptom of something seriously wrong. Since the most common cause for the sudden onset of amenorrhea is pregnancy, that possibility should always be ruled out as the first cause. As many as 5 % of women of reproductive age experience amenorrhea sometime during their reproductive life [2, 14, 15]. However, girls and women engaged in athletics often welcome the absence of periods and do not consider this a serious medical issue. In addition to some of the aforementioned medical conditions that can cause disruption of menstrual cycles, there can also be serious long-term consequences of suppressed ovarian function.

High levels of prolactin or hyperprolactinemia can also cause amenorrhea. Lactational amenorrhea is common in women who supply most of the nutritional needs of their infant by breastfeeding. Pituitary tumors (prolactinomas) that secrete prolactin also suppress gonadotropin secretion which results in cessation of menstrual cycles. Approximately 50 % of the individuals that have these tumors also have galactorrhea [16], or secretion of milk from the breast, which often goes unnoticed. If the tumor is actively growing, it can progressively cause visual deficits, blurred vision, and headaches. If not treated, long-term consequences of hyperprolactinemia include osteoporosis due to suppression of ovarian steroid secretion. Medical management of prolactin-secreting tumors is effective using dopamine agonists that suppress the tumors and optimize prolactin levels.

Anorexia nervosa is probably the most recognizable cause of amenorrhea in young women who are not pregnant. Serious eating disorders, particularly anorexia nervosa and bulimia nervosa, are often associated with menstrual disorders. Both of these conditions have their highest incidence during adolescent years and can lead to death if not treated by appropriate professionals. In young female athletes, amenorrhea is often part of a syndrome called the female athlete triad. This term was first used in the literature in 1993 associated with sports medicine [17] to describe a condition in young female athletes who exhibited disordered eating, loss of bone (osteopenia), and amenorrhea. Women at greatest risk for this condition are those training in sports where low body weight is desired or required, but it can occur in women involved in a wide range of physical activities. Reversible amenorrhea where there is no physical cause for the condition is called functional amenorrhea. This condition is the result of diminished or suppressed GnRH release from the brain to the extent that there is not sufficient LH and FSH to stimulate ovarian function [18]. Because of low gonadotropin levels follicles in

the ovary do not develop, estrogen levels are low, and the lining of uterus (endometrium) does not proliferate. This endocrine situation is much like the prepubertal condition where menstruation does not occur.

The American College of Sports Medicine (ASCM) published a revised position stand on the female athlete triad in 2007 which does *not* include dietary restriction or disordered eating as a necessary component of the triad [19]. This group emphasizes that menstrual disorders and osteopenia observed in many female athletes who participate in prolonged exercise can result from energy deficiency if energy intake is not sufficient to balance the increased energy expenditure. In addition to the familiar disordered eating and the weight loss programs that put female athletes at risk for serious health problems, a less familiar and less well-understood phenomenon is suppression of appetite by prolonged exercise. A recent excellent review of the complexity of the effect of exercise on health issues in women, and the general lack of understanding of these issues by athletes, their coaches, and physicians, documents the need for careful diet management in female athletes to avoid serious health issues [20]. A recent survey of college coaches of female sports revealed that less than half of those responding could correctly identify the components of the female athlete triad and a third were not familiar with the term [21]. Furthermore, more than half of the respondents seldom or never queried their athletes regarding their menstrual function. This would suggest that there is an urgent need for additional education of college, and probably even more so for high school, coaches regarding the long-term consequences of prolonged or excessive exercise in female athletes.

## 5.4 Contemporary Understanding of the Issues

Relevant for female athletes in general and especially in adolescent female athletes are the long-term consequences of insufficient energy intake that results in menstrual cycle disturbances and low bone mineral density. Prolonged exercise as practiced by elite athletes can lead to serious, long-term health problems if diet is not adjusted to compensate for energy expenditure. A good barometer of how energy intake matches energy expenditure in female athletes is menstrual function. When menstrual cycles become disordered as a result of prolonged or excessive exercise, there very well may be an issue related to insufficient energy intake. When menstrual cycles are disrupted in athletes, consultation of a health care provider knowledgeable in sports medicine is strongly advised. The standard curriculum for high school and college coaches of female athletes should include the health consequences of exercise-induced menstrual function and specific methods of prevention and treatment.

## 5.5 Future Directions

Because there are so many potential interactions between fat, the digestive system, the immune system, and the brain, there is still much to learn about the chemical signals that control appetite and satiety. However, even if we knew most of these interactions, the health implications of insufficient energy to support all necessary biological processes will not be addressed until athletes and coaches can be educated about the consequences of winning at any cost.

## 5.6 Concluding Remarks

Menstrual cycles are a reflection of cyclic ovarian function and irregular or absent cycles may indicate some underlying pathology. Regardless of the cause, prolonged disruption of ovarian function can have serious health consequences, even if there is no serious medical condition causing the abnormal cycles. Therefore, both athletes and their coaches and trainers should be concerned when there are menstrual cycle disturbances and seek appropriate medical advice.

## References


1. Loucks AB, Horvath SM. Athletic amenorrhea: a review. Med Sci Sports Exerc. 1985;17:56–72.
2. Peterson F, Fries H, Nillius SJ. Epidemiology of secondary amenorrhea. Incidence and Prevalence rates. Am J Obstet Gynecol. 1973;117:80–6.
3. Hull MG. Ovulation failure and induction. Clin Obstet Gynaecol. 1981;8:753–85.
4. Azziz R, Woods KS, Reyna R, et al. The prevalence and features of the polycystic ovary syndrome in an unselected population. J Clin Endocrinol Metab. 2004;89:2745–9.
5. Loucks AB, Thuma JR. Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. J Clin Endocrinol Metab. 2003;88:297–311.
6. Stein I, Leventhal M. Amenorrhea associated with bilateral polycystic ovaries. Am J Obstet Gynecol. 1935;29:181–91.
7. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertil Steril. 2004; 81:19–25.
8. Hillard PJA, Deitch HR. Menstrual disorders in the college age female. Pediatr Clin North Am. 2005;52: 179–97.
9. Ovalle F, Azziz R. Insulin resistance, polycystic ovary syndrome, and type 2 diabetes mellitus. Fertil Steril. 2002;77:1095–105.
10. Legro RS. Polycystic ovary syndrome and cardiovascular disease: a premature association? Endocr Rev. 2002;24:302–12.
11. Loucks AB. The response of luteinizing hormone pulsatility to five days of low energy availability disappears by 14 years of gynecological age. J Clin Endocrinol Metab. 2006;91:3158–64.
12. Loucks AB. Energy availability and infertility. Curr Opin Endocrinol Diabetes Obes. 2007;14:470–4.
13. Bronson FH. Mammalian reproductive biology. Chicago, IL: The University of Chicago Press; 1989.
14. Bachman GA, Kemmann E. Prevalence of oligomenorrhea and amenorrhea in a college population. Am J Obstet Gynecol. 1982;144:98–102.
15. Singh KB. Menstrual disorders in college students. Am J Obstet Gynecol. 1981;140:299–302.
16. Thorner MO, Vance ML, Laws Jr ER, et al. The anterior pituitary. In: Wilson JD, Foster DW, editors. Williams textbook of endocrinology. 9th ed. Philadelphia: Saunders; 1998. p. 249–340.
17. Yeager KK, Agostine R, Nativ A, et al. The female athlete triad: disordered eating, amenorrhea, osteoporosis. Med Sci Sports Exerc. 1993;25:775–7.
18. Loucks AB, Vaitukaitis J, Cameron JL. The reproductive system and exercise in women. Med Sci Sports Exerc. 1992;24(Suppl):S288–93.
19. Nattiv A, Loucks AB, Manore MM, et al. American College of Sports Medicine Position Stand: The female athlete triad. Med Sci Sports Exerc. 2007;39:1867–82.
20. Loucks AB, Kiens B, Wright HH. Energy availability in athletes. J Sports Sci. 2011;29:S7–15.
21. Pantano KJ. Current knowledge, perceptions, and interventions used by collegiate coaches in the U.S. regarding the prevention and treatment of the female athlete triad. N Am J Sports Phys Ther. 2006;1: 195–207.

# Nutritional, Physical, and Psychological Stress and Functional Amenorrhea

6

Reid L. Norman and Melissa R. Iñigo

**Abstract**

The lack of regular, cyclic menstruation in reproductive-aged women in whom there is no organic or anatomic cause is called functional hypothalamic amenorrhea (FHA). The cause of this malfunction in the hypothalamic-pituitary-ovarian axis is generally attributed to psychological, physical, or nutritional stress, and the underlying deficit is the suppression of gonadotropin-releasing hormone release by the hypothalamus. The diagnosis of FHA is reached by the exclusion of all other factors and conditions that could cause amenorrhea. FHA, by definition, is a chronic condition and can have serious health consequences if not treated. Since the underlying causes of the FHA are thought to be either low energy availability or dysfunctional attitudes and behaviors that result in stimulation of the hypothalamic-pituitary-adrenal axis and/or suppression of thyroid function, cognitive behavioral therapy and hypnotherapy are the best potential approaches for ameliorating this condition. Finally, increasing food intake and reducing stressful activities can be very effective in restoring menstruation.

**Keywords**

Functional hypothalamic amenorrhea • Psychological stress • Cortisol • CRH • Endogenous opioids • Leptin

## 6.1 Learning Objectives

After completing this chapter, you should have an understanding of:

- The definition of functional hypothalamic amenorrhea (FHA)
- The causes of FHA
- The importance of seeking medical attention for this condition
- The long-term consequences of chronic low estrogen levels

R.L. Norman (✉)
Department of Pharmacology and Neuroscience, Texas Tech University Health Sciences Center School of Medicine, 3601 4th Street, Lubbock, TX, USA
e-mail: reid.normna@ttuhsc.edu

M.R. Iñigo
Department of Health, Exercise, and Sport Sciences, Texas Tech University, HESS, Box 43011 Texas Tech University, Lubbock, TX 79409-3011, USA
e-mail: minigo@umd.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_6, 

## 6.2 Introduction

FHA is the absence of menstrual cycles for more than 6 months without any anatomic or pathologic cause for the condition. The term "functional" indicates that the lack of menstrual cycles is due to improper functioning of the hypothalamic-pituitary-ovarian axis rather than due to an anatomic (organic) problem. The diagnosis of women of FHA requires the exclusion of all other possible metabolic, neurological, or organic causes for the absence of menstruation such as prolactin-secreting pituitary adenomas, thyroid dysfunction, and polycystic ovarian syndrome (PCOS) [1]. The underlying deficit in FHA is suppressed gonadotropin-releasing hormone (GnRH) release from the hypothalamus resulting in low gonadotropin (LH and FSH) and estrogen levels. When estrogen levels are low, the endometrium of the uterus does not develop and periodic menstruation is absent.

## 6.3 Research Findings

### 6.3.1 Potential Causes of FHA

Since primitive times, a common belief among women has been that regular menstrual bleeding is a sign of mental health and that emotional trauma results in disrupted menstruation [2]. Hans Selye [3] was the first to recognize that mental stress was among those changes in the environment that disturbed the sexual cycle in females. Refeinstein [4] was also among the first to link disrupted menstrual cycles with "overt or latent psychological disturbances." He recognized that psychogenic or functional amenorrhea was a problem associated with the brain even before it was accepted that reproductive function (i.e., release of LH and FSH) was governed by the hypothalamus (GnRH release). Recent studies suggest that FHA is triggered by a variety of stressors, including energy deprivation induced by dieting, excessive exercise, and psychosocial distress. Women experience one or all three factors at the same time, which results in the suppression of the GnRH drive [1, 5, 6]. Evidence of hypercortisolemia in amenorrheic women provides the link for the relationship between stress and FHA [7]. For instance, amenorrheic women were observed to have elevated cortisol levels while sleeping compared to healthy controls [8]. Women cope with and respond to these stressors differently, which makes it difficult to establish a threshold at which psychogenic stress interferes with the normal menstrual cycle [9, 10].

Low energy availability due to dieting coupled with excessive exercise suppresses the reproductive system in both women and nonhuman primates. When female rhesus monkeys were kept on a constant diet and increased their energy expenditure by increasing the time they exercised each day, all of the animals eventually became amenorrheic [11]. Menstrual cycles were re-established when additional calories were provided during a treatment period of constant training intensity and volume [12]. One study in women with FHA found that 46 and 39 % of amenorrheic participants were anorexic and bulimic, respectively [13]. When these amenorrheic women were compared to matched controls, calorie intake over a 24-h recall was not significantly different between groups; however, calories expended per day during high-intensity aerobic exercise were considerably greater in amenorrheic women. The greater energy expenditure coupled with the anorexic and bulimic behaviors of the amenorrheic group suggests that they had lower energy availability compared to their normal healthy counterparts. These women also experienced other endocrine alterations associated with FHA such as reduced leptin, thyroxine, and triiodothyronine levels [13]. It is the availability of energy not the stress of exercising that reduces both LH secretion [14, 15] and diurnal rhythm of leptin [16]. Together, these studies in women and nonhuman primates convincingly demonstrate that reproductive cycles are dependent on adequate energy availability and are quickly disrupted when energy is limited.

An acute psychological stressor can also inhibit gonadotropin secretion from the pituitary [17, 18], which may cause FHA. In nonhuman primates, ACTH and cortisol were elevated and

LH levels were decreased when the animals were moved from the home cage to a restraining chair. Within a few hours after returning to their cages, pulsatile LH secretion resumed and stress hormone levels returned to normal. These data are consistent with the general notion of how stress inhibits reproductive function (i.e., by suppressing the release of GnRH from the hypothalamus and, subsequently, the function of the pituitary and ovary). Some psychological stressors experienced by women and which have been associated with FHA are performance pressure, intellectual pursuits, aiming for perfection, traumatic experiences (e.g., sexual molestation), problems with social approval, having unrealistic goals, negative attitude towards eating, and other negative attributions [5, 6, 19].

### 6.3.2 Neuroendocrinology and Pathophysiology of FHA

Nutritional, physical, and psychological stressors affect the neuroendocrine control of the reproductive axis, which leads to the suppression of GnRH and, in turn, FHA. Specifically, neuropeptides such as corticotropin-releasing hormone (CRH), vasopressin, β-endorphin, leptin, ghrelin, allopregnanolone, and neuropeptide Y all may play a role in the pathophysiology of FHA. CRH and vasopressin are released by the paraventricular nucleus of the hypothalamus and can inhibit GnRH release. CRH causes the release of ACTH from the pituitary which then stimulates cortisol secretion by the adrenal. In vitro [20, 21] and in vivo [22] studies have shown that the release of GnRH can also be directly inhibited by CRH through its direct connections with GnRH neurons in the hypothalamus [23].

Cortisol is also coupled with a progesterone metabolite, allopregnanolone, in women with FHA as well as in healthy controls. However, women with FHA experience a blunted allopregnanolone response to CRH [24]. CRH stimulates the production of β-endorphin, an opioid peptide, and circulating levels of this peptide increase during intense exercise. In vitro studies have shown that β-endorphin inhibits GnRH release [25]. When β-endorphin release was blocked by naxolone, an opioid antagonist, GnRH levels increased [17, 26]. β-endorphin can also directly inhibit LH release [7]. These findings suggest that β-endorphin mediates the action of CRH in suppressing GnRH [27].

In women with FHA, leptin (a protein produced by fat and implicated in reproductive function) levels are lower than in normal healthy women and the normal diurnal rhythm in leptin levels is also absent [7, 28]. Low leptin levels are typical in amenorrheic women with low body fat, and leptin levels increase as body fat increases in women with FHA [29]. Leptin levels increase significantly with every 1 kg increase in weight and are associated with the return of pulsatile LH release. Moreover, hypoleptinemia may increase cortisol levels through activating the adrenergic pathway and CRH release [7]. Low leptin levels stimulate the release of neuropeptide Y in the hypothalamus. Neuropeptide Y stimulates GnRH production (if estradiol concentrations are high) as well as increased pulsatile LH release. However, in women with FHA, estradiol levels are low (<20 pg/ml) [19], and basal levels of neuropeptide Y are lower compared to healthy women [7]. Another hormone from the gut, ghrelin, opposes leptin function. Ghrelin inhibits GnRH secretion and is elevated in women with FHA, especially if these women have disrupted eating habits [30].

Other peptides such as orexin A and kisspeptin that control GnRH release [7] have not been studied in women with FHA.

## 6.4 Contemporary Understanding of the Issues

### 6.4.1 Possible Genetic Contribution to FHA

FHA is a condition that is similar to isolated hypogonadotropic hypogonadism and may have a genetic component as well [31]. Both conditions refer to the amenorrhea due to the suppression of GnRH; however, hypogonadotropic hypogonadism has a genetic basis. Mutations in

genes for fibroblast growth factor receptor, prokineticin receptor 2, GnRH receptor, and KAL-1 are among those found in the hypogonadotropic hypogonadism and are also found in some women with FHA. These genes are known to be loss-of-function mutants; for instance, expression of prokineticin receptor 2 on the cell surface and its signaling activity were significantly less in women with FHA than in healthy controls [31]. Furthermore, these genetic defects, particularly in the fibroblast growth factor receptor and prokineticin receptor 2, may contribute to the negative eating habits of women with FHA, as they alter eating behavior in animal models [32, 33]. Finally, the mutant GnRH receptor, KAL-1, and prokineticin receptor 2 are involved in the processes that lead to suppressed GnRH, altered LH activity, and absence of menstruation.

### 6.4.2 Psychiatric Contribution to FHA

Psychiatric histories of women with FHA have also been compared with amenorrheic (from organic causes) and eumenorrheic controls [34]. Women with FHA had more dysfunctional attitudes, did not cope as well with ordinary stresses, and showed more interpersonal dependence than did eumenorrheic women. These women also more often had a history of psychiatric mood disorders, particularly depression and anxiety, than did women with normal cycles [35]. Depression was also linked to sexual function problems experienced by women with FHA [35]. These psychiatric disorders are associated with elevation in hypothalamic-pituitary-adrenal axis activity. When combined with performance anxiety and dietary restriction, these disorders may contribute to significant endocrine dysfunction and result in FHA.

### 6.4.3 Medical Problems Associated with FHA

Significant weight loss may occur in women with FHA because of dieting and/or excessive exercise; however, not all women with this condition are underweight. Weight loss-induced amenorrhea significantly lowers bone mineral density such that women with FHA have osteopenia [36–39]. Indeed, an inverse relationship exists between the length of amenorrhea (in months) and bone mineral density [37, 39]. Low bone mineral density may affect cardiovascular factors as well. Bone mineral density is inversely correlated with total cholesterol, apolipoprotein A, and very-low-density lipoprotein levels [40]. Endothelial dysfunction, which increases the risk for atherosclerosis, may also occur in women with FHA [41]. Amenorrheic female athletes, who participate in high-intensity training and strict dieting, can develop a serious condition called the female athlete triad (discussed in Chap. 5).

### 6.4.4 Diagnosis of FHA

The diagnosis of FHA has gained a firmer basis over the past few years. A thorough review of the medical history, including exercise and eating habits (e.g., binging or purging), must be conducted to identify the specific nutritional, physical, and psychological stressors that are present. The physical examination must exclude all other possible medical reasons that would cause amenorrhea such as PCOS, adrenal hyperplasia, presence of an adrenal-secreting tumor, adnexal mass, an imperforated hymen, and Mullerian duct anomaly (i.e., absence of uterus) [1]. Signs of hirsutism, acne, male pattern baldness, clitoromegaly, voice changes, and vomiting (i.e., gingival abrasions, loss of dental enamel, parotid swelling) must also be examined [1]. Thus, levels of beta subunit of chorionic gonadotropin, thyrotropin, thyroxine, prolactin, follicle-stimulating hormone, free testosterone, and dehydroepiandrosterone sulfate must all be analyzed [1]. Magnetic resonance images of the brain may also be used to determine conditions that might contribute to FHA [1].

The long-term negative health consequences of FHA include increased risk of cardiovascular disease (e.g., coronary artery disease), osteoporosis, increased risk for fractures,

depression, dementia, and other psychiatric conditions [39, 42, 43]. Both pharmacologic and non-pharmacologic therapies have been suggested for treatment of FHA.

### 6.4.5 Potential Interventions in Women with FHA

Several forms of estrogen administration have been investigated, including estriol and 17-ß estradiol [36, 44]. In general, estrogen treatment increases plasma levels of LH and bone mineral density; however, bone mineral density does not return to levels observed in healthy women [36, 44]. Naltrexone cloridate, an opioid antagonist, administered at 50 mg/day for 3–6 months resulted in marked increases in estrogen and restoration of menses in some women [45]. Recently, leptin administration has also been shown to increase LH levels and pulse frequency as well as increase estradiol levels, number of dominant follicles, ovarian volume, follicular diameter, free triiodothyronine levels, free thyroxine levels, insulin-like growth factor 1 levels, insulin-like growth factor-binding protein 3, bone alkaline phosphatase, and osteocalcin and even restored menstruation in some women [46]. Although these pharmacologic therapies have provided positive results, scientists argue that drug use does not address the underlying causes of FHA [42]. Merely increasing food intake and decreasing exercise load are highly effective in increasing bone mineral density and restoring menstruation [43, 47]. However, non-pharmacologic treatment has its limitations. Regular menstrual periods may not return until after 1 year of increased food intake and decreased exercise [47], while pharmacologic treatments restore menstruation within 3–6 months [45]. Furthermore, no specific threshold for weight or percent body fat has been established for the restoration of menses [1]. Finally, the practicality of non-pharmacologic approaches is questionable, especially in serious athletes who maintain a strict diet [1]. Psychological approaches to reduce stress such as cognitive behavioral therapy [48] and hypnotherapy [49] are highly effective, and recovery of ovarian function and restoration of menses occurred in as high as 87.5 % of the women with FHA [48, 49].

## 6.5 Future Directions

Prolonged FHA increases the risk for potentially fatal outcomes. Future directions include the possible use of CRH antagonists to treat women with FHA. Some speculate that women with FHA experience blunted ACTH and cortisol response to CRH due to the reduced expression and sensitivity of CRH receptors [7]. Treatment with CRH antagonists may reduce cortisol levels and allow the return of pulsatile GnRH secretion. Moreover, women with FHA should increase food intake and reduce exercise load. The active guidance of a primary care physician, nutritionist, and psychotherapist may help these women recover and restore menstruation more successfully [1].

## 6.6 Concluding Remarks

The lack of regular, cyclic menstruation in reproductive-aged women in whom there is no organic or anatomic cause is called FHA. The cause of this malfunction in the hypothalamic-pituitary-ovarian axis is generally attributed to psychological, physical, or nutritional stress. The diagnosis of FHA is reached by the exclusion of all other factors and conditions that could cause amenorrhea. FHA, by definition, is a chronic condition, and thus it can have serious health consequences if not treated. Since the underlying causes of the FHA are thought to be dysfunctional attitudes and behaviors that result in stimulation of the hypothalamic-pituitary-adrenal axis and/or suppression of the thyroid function, cognitive behavioral therapy and hypnotherapy are the best potential approaches for ameliorating this condition [42]. Finally, increasing food intake and reducing stressful activities can be very effective in restoring menstruation.

## References


1. Gordon CM. Clinical practice. Functional hypothalamic amenorrhea. N Engl J Med. 2010;363:365–71.
2. Kroger W, Freed SL. Psychosomatic gynecology. Illinois: The Free Press; 1956.
3. Selye H. The effect of adaptation to various damaging agents on the female sex organs in the rat. Endocrinology. 1939;25:615–24.
4. Refeinstein Jr EC. Psychogenic or "hypothalamic" amenorrhea. Med Clin North Am. 1946;30:1103–15.
5. Marcus MD, Loucks TL, Berga SL. Psychological correlates of functional hypothalamic amenorrhea. Fertil Steril. 2001;76:310–6.
6. Young EA, Korszun A. The hypothalamic-pituitary-gonadal axis in mood disorders. Endocrinol Metab Clin North Am. 2002;31:63–78.
7. Meczekalski B, Podfigurna-Stopa A, Warenik-Szymankiewicz A, et al. Functional hypothalamic amenorrhea: current view on neuroendocrine aberrations. Gynecol Endocrinol. 2008;24:4–11.
8. Berga SL, Daniels TL, Giles DE. Women with functional hypothalamic amenorrhea but not other forms of anovulation display amplified cortisol concentrations. Fertil Steril. 1997;67:1024–30.
9. Drew FL. The epidemiology of secondary amenorrhea. J Chronic Dis. 1961;14:396–407.
10. Ferin M. Stress and the reproductive cycle. J Clin Endocrinol Metab. 1999;84:1768–74.
11. Williams NI, Caston-Balderrama AL, Helmerich DL, et al. Longitudinal changes in reproductive hormones and menstrual cyclicity in cynomolgus monkeys during strenuous exercise training: abrupt transition to exercise-induced amenorrhea. Endocrinology. 2001;142:2381–9.
12. Williams NI, Helmreich DL, Parfitt DB, et al. Evidence for a causal role of low energy availability in the induction of menstrual cycle disturbances during strenuous exercise training. J Clin Endocrinol Metab. 2001;86:5184–93.
13. Warren MP, Voussoughian F, Geer EB, et al. Functional hypothalamic amenorrhea: hypoleptinemia and disordered eating. J Clin Endocrinol Metab. 1999;84:873–7.
14. Loucks AB, Verdun M, Heath EM. Low energy availability, not stress of exercise, alters LH pulsatility in exercising women. J Appl Physiol. 1998;84:37–46.
15. Loucks AB, Thuma JR. Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. J Clin Endocrinol Metab. 2003;88:297–311.
16. Hilton LK, Loucks AB. Low energy availability, not exercise stress, suppresses the diurnal rhythm of leptin in healthy young women. Am J Physiol Endocrinol Metab. 2000;278:E43–9.
17. Norman RL, Smith CJ. Restraint inhibits LH and testosterone secretion in intact male rhesus macaques: effects of concurrent naloxone administration. Neuroendocrinology. 1992;55:405–15.
18. Norman RL, McGlone JJ, Smith CJ. Restraint inhibits LH secretion in the follicular phase of the menstrual cycle in female rhesus macaques. Biol Reprod. 1994;50:16–26.
19. Liu JH, Bill AH. Stress-associated or functional hypothalamic amenorrhea in the adolescent. Ann N Y Acad Sci. 2008;1135:179–84.
20. Gambacciani M, Yen SSC, Rasmussen D. GnRH release from the medial basal hypothalamus: in vitro inhibition by corticotropin-releasing factor. Neuroendocrinology. 1986;43:533–6.
21. Nikolarakis KE, Almeida OFX, Herz A. Corticotropin-releasing factor (CRF) inhibits gonadotropin-releasing hormone (GnRH) release from superfused rat hypothalami in vitro. Brain Res. 1986;377:388–90.
22. Petraglia F, Sutton S, Vale W, Plotsky P. Corticotropin-releasing factor decreases plasma luteinizing hormone levels in female rats by inhibiting gonadotropin-releasing hormone release into hypophyseal portal circulation. Endocrinology. 1987;120:1083–8.
23. MacLusky JN, Naftolin F, Leranth C. Immunocytochemical evidence for direct synaptic connections between corticotropin-releasing factor (CRF) and gonadotropin-releasing hormone (GnRH) containing neurons in the preoptic area of the rat. Brain Res. 1988;439:391–5.
24. Genazzani AD, Luisi M, Malavasi B, et al. Pulsatile secretory characteristics of allopregnanolone, a neuroactive steroid, during the menstrual cycle and in amenorrheic subjects. Eur J Endocrinol. 2002;146:347–56.
25. Rasmussen DD. New concepts in the regulation of hypothalamic gonadotropin releasing hormone (GnRH) secretion. J Endocrinol Invest. 1986;9:427–37.
26. Rasmussen DD, Liu JH, Wolf PL, Yen SS. Endogenous opioid regulation of gonadotropin-releasing hormone release from the human fetal hypothalamus in vitro. J Clin Endocrinol Metab. 1983;57:881–4.
27. Barbarino A, De Marinis L, Tofani A, et al. Corticotropin-releasing hormone inhibition of gonadotropin release and the effect of opioid blockade. J Clin Endocrinol Metab. 1989;68:523–8.
28. Laughlin GA, Yen SS. Hypoleptinemia in women athletes: absence of a diurnal rhythm with amenorrhea. J Clin Endocrinol Metab. 1997;82:318–21.
29. Ahima RS. Body fat, leptin, and hypothalamic amenorrhea. N Engl J Med. 2004;351:959–62.
30. Tolle V, Kadem M, Bluet-Pajot MT, et al. Balance in ghrelin and leptin plasma levels in anorexia nervosa patients and constitutionally thin women. J Clin Endocrinol Metab. 2003;88:109–16.
31. Caronia LM, Martin C, Welt CK, et al. A genetic basis for functional hypothalamic amenorrhea. N Engl J Med. 2011;364:215–25.
32. Gardiner JV, Bataveljic A, Patel NA, et al. Prokineticin 2 is a hypothalamic neuropeptide that potently inhibits food intake. Diabetes. 2010;59:397–406.
33. Sun HD, Malabunga M, Tonra JR, et al. Monoclonal antibody antagonists of hypothalamic FGFR1 cause potent but reversible hypophagia and weight loss in

rodents and monkeys. Am J Physiol Endocrinol Metab. 2007;292:E964–76.
34. Giles DE, Berga SL. Cognitive and psychiatric correlates of functional hypothalamic amenorrhea: a controlled comparison. Fertil Steril. 1993;60:486–92.
35. Dundon CM, Rellini AH, Tonani S, et al. Mood disorders and sexual functioning in women with functional hypothalamic amenorrhea. Fertil Steril. 2010;94: 2239–43.
36. Sowinska-Przepiera E, Andrysiak-Mamos E, Syrenicz J, et al. Polymorphism of the vitamin D3 receptor gene and bone mineral density in girls with functional hypothalamic amenorrhea subjected to oestroprogestagen treatment. Endokrynol Pol. 2011;62:492–8.
37. Podfigurna-Stopa A, Pludowski P, Jaworski M, et al. Skeletal status and body composition in young women with functional hypothalamic amenorrhea. Gynecol Endocrinol. 2012;28:299–304.
38. Warren MP, Brooks-Gunn J, Fox RP, et al. Osteopenia in exercise-associated amenorrhea using ballet dancers as a model: a longitudinal study. J Clin Endocrinol Metab. 2002;87:3162–8.
39. Grinspoon S, Miller K, Coyle C, et al. Severity of osteopenia in estrogen-deficient women with anorexia nervosa and hypothalamic amenorrhea. J Clin Endocrinol Metab. 1999;84:2049–55.
40. Soleimany G, Dadgostar H, Lotfian S, et al. Bone mineral changes and cardiovascular effects among female athletes with chronic menstrual dysfunction. Asian J Sports Med. 2012;3:53–8.
41. O'Donnell E, Goodman JM, Harvey PJ. Clinical review: cardiovascular consequences of ovarian disruption: a focus on functional hypothalamic amenorrhea in physically active women. J Clin Endocrinol Metab. 2011;6:3638–48.
42. Berga SL, Loucks TL. The diagnosis and treatment of stress-induced anovulation. Minerva Ginecol. 2005;57:45–54.
43. Vescovi JD, Jamal SA, De Souza MJ. Strategies to reverse bone loss in women with functional hypothalamic amenorrhea: a systematic review of the literature. Osteoporos Int. 2008;19:465–78.
44. Genazzani AD, Meczekalski B, Podfigurna-Stopa A, et al. Estriol administration modulates luteinizing hormone secretion in women with functional hypothalamic amenorrhea. Fertil Steril. 2012;97:483–8.
45. Genazzani AD, Gastaldi M, Petraglia F, et al. Naltrexone administration modulates the neuroendocrine control of luteinizing hormone secretion in hypothalamic amenorrhoea. Hum Reprod. 1995;10: 2868–71.
46. Welt CK, Chan JL, Bullen J, et al. Recombinant human leptin in women with hypothalamic amenorrhea. N Engl J Med. 2004;351:987–97.
47. Arends JC, Cheung MY, Barrack MT, et al. Restoration of menses with nonpharmacologic therapy in college athletes with menstrual disturbances: a 5-year retrospective study. Int J Sport Nutr Exerc Metab. 2012; 22:98–108.
48. Berga SL, Marcus MD, Loucks TL, et al. Recovery of ovarian activity in women with functional hypothalamic amenorrhea who were treated with cognitive behavior therapy. Fertil Steril. 2003;80:976–81.
49. Tschugguel W, Berga SL. Treatment of functional hypothalamic amenorrhea with hypnotherapy. Fertil Steril. 2003;80:982–5.

# Effects of the Menstrual Cycle on the Acquisition of Peak Bone Mass

7

Mimi Zumwalt and Brittany Dowling

**Abstract**

The menstrual cycle has huge implications on the building, maintenance, and break down of skeletal bone in women. Due to the fluctuating level of female hormones, the menstrual cycle plays a different role during various times of the month which in turn affects bone health. Estrogen is a crucial hormone for bone turnover/remodeling which, when released, provides a protective mechanism against the process of natural bone loss due to aging. Acquiring a high amount of peak bone mass during adolescence helps to protect the female athlete against rapid degradation of bone due to the decline of estrogen around menopause. Therefore, taking appropriate steps years before and after menopause is crucial in order to preserve bone mass in females.

**Keywords**

Menstrual cycle • Estrogen • Bone turnover/remodeling • Peak bone mass

## 7.1 Learning Objectives

At the completion of this chapter, you should have an understanding of the following:

- The pertinent female reproductive anatomy and physiology of a normal menstrual cycle, including the different phases and roles of various hormones
- The different components of bone along with its function, biochemistry, and metabolism
- The interaction between different ovarian hormones and various bone components
- The definition, importance, and effective methods of achieving peak bone mass

M. Zumwalt, M.D. (✉)
Department of Orthopaedic Surgery and Rehabilitation, Texas Tech University Health Sciences Center, 3601 4th Street, Lubbock, TX 79430-9436, USA
e-mail: mimi.zumwalt@ttuhsc.edu

B. Dowling
Department of Health, Exercise, and Sports Sciences, Texas Tech University, 1545 Shadowtree Court, Colorado Springs, CO 80921, USA
e-mail: dowlingb86@gmail.com

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_7, 

- The results of an abnormal menstrual cycle on attainment of peak bone mass, how to go about assessing/measuring bone density, and ways to address this issue further to help minimize bone loss and maintain bone density

## 7.2 Introduction

Bone tissue has various functions; however, its primary function is to provide structural and mechanical support for soft tissues. Because the skeleton provides structural support for body, having the maximal amount of strong bones will serve to protect against osteoporosis (decreased bone mass due to bone loss) later on in life [1]. Bone is far from static; in fact, this living tissue is quite dynamic. New bone continually remodels and repairs old bone depending on mechanical, physiological, and hormonal stimuli. The latter, systemic hormonal milieu, plays a very important role during puberty and in females is manifested by the menstrual cycle [2–4]. There is no evidence for gender difference in bone mineral density (BMD) at birth, and accrual rates stay the same until puberty, where sex differences become pronounced. Studies have found 40–50 % of adult peak bone mass is accumulated during puberty [5] and peak bone mass is achieved around ages 25–35 years [6]. During the teenage years 60–90 % of all skeletal bone is laid down [8–10]. Genetics predetermine 60–80 % of skeletal development; however, environmental factors (diet, energy availability, physical stress) account for 20–40 % of developing bone [3, 4, 8]. The amount of bone mass that is gained during this adolescent period equates to the quantity of bone that will be lost during the rest of one's adult life [10, 11]. Therefore, measures to maximize and protect the quantity of bone developed during the growing period is advantageous and will serve the body well in activities of daily living and other life endeavors beyond these early years [12].

This chapter will focus primarily on the female menstrual cycle and its influence on peak bone mass achieved during and after adolescence.

## 7.3 Research Findings and Contemporary Understanding of the Issues

### 7.3.1 Anatomy of the Female Sex Organs and Physiology of the Menstrual Cycle

The female reproductive system is a multiorgan system comprised of the hypothalamus, pituitary gland, ovaries, uterus, and vagina (Fig. 7.1).

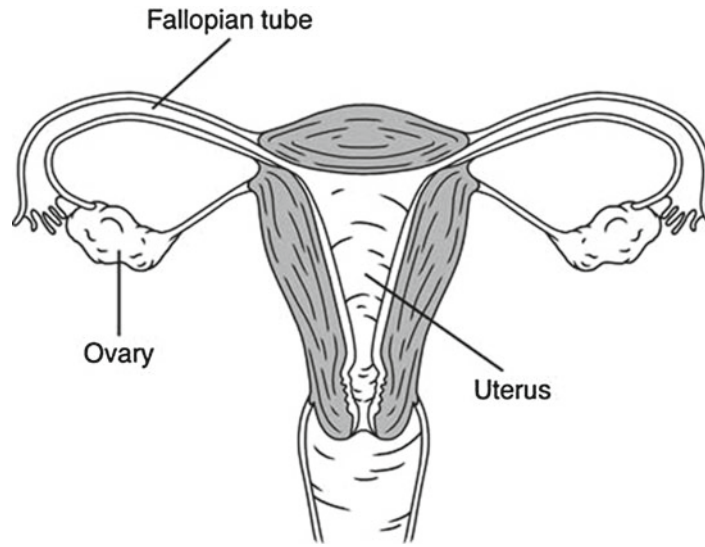



**Fig. 7.1** Female reproductive unit

Pertinent anatomy involved with the menstrual cycle includes the ovaries, a paired organ which has both a reproductive and an endocrine function. This is where eggs are formed and female sex hormones are produced [13, 14]. Three levels of hormones are normally secreted in a feedback loop termed the hypothalamic-pituitary-ovarian (HPO) axis [15]: (1) by the hypothalamus—gonadotropin-releasing hormone (GnRH); (2) by the anterior pituitary—follicle-stimulating hormone (FSH) and luteinizing hormone (LH) in response to GnRH; and (3) by the ovaries—two gonadal steroids, estrogen and progesterone, in response to FSH and LH. Regulation of the menstrual cycle is controlled by both LH and FSH from the anterior pituitary gland in response to GnRH from the hypothalamus [16]. As discussed in prior chapters, these hormones are secreted in different amounts during various phases of the monthly menstrual cycle [3, 14]. The onset of menarche in females signals the transition from childhood to the pubertal, reproductive state, correlating with both body height and bone maturation [15]. Menarche normally occurs about two years after the appearance of secondary sex characteristics (breast and pubic hair development) around age 11–13 years, but can begin as early as 7 years old [9, 14]. Menarche is relatively a late determinant of pubertal development [17]. If menstruation does not occur by age 16, this delay of menarche (primary amenorrhea) may be due to excessive exercise or insufficient intake of appropriate nutrition, among other factors [16, 17].

The menstrual cycle occurs on a regular basis between 20 and 45 days, on average approximately every 4 weeks (28 days), with menstrual flow lasting from 3 to 7 days [7, 9, 14]. Typically, three separate phases encompass the normal menstrual cycle: (1) follicular or proliferative phase (growth of the endometrium or uterine lining stimulated by estrogen), (2) ovulatory phase (ovulation or egg release in response to LH), and (3) luteal or secretory phase (transformation of the endometrium from a proliferating into secreting-type tissue under progesterone influence) [16, 18]. The duration of the follicular phase is more variable between different women, whereas the luteal phase is consistently 14 days [7]. The first day of the menstrual cycle is marked with the initial sign of vaginal bleeding. The onset of the follicular phase is stimulated by FSH more so than LH; the quantity of LH slowly increases during the follicular phase until peaking levels at mid-cycle. High amounts of FSH during the beginning of the luteal phase stimulate growth of several ovarian follicles then levels begin to decline until late luteal phase [7]. During this time both progesterone and estrogen serum levels are low. Estrogen then increases steadily, peaking just before ovulation, generally occurring on the same day of the LH surge [7]. This rise in estrogen occurs about the middle of the cycle, day 14, when one follicle is released in the ovulation phase. The luteal phase then takes over during the second half of the cycle under the continuing influence of LH. Progesterone then increases above estrogen levels, though the latter hormone concentration remains fairly high. These two hormones, estrogen and progesterone, are secreted by the corpus luteum which then degenerates, causing both hormonal levels to concurrently fall as long as the egg is neither fertilized nor implanted in the uterus. The monthly cycle then repeats itself by shedding of the endometrium, manifested by menstrual flow, to restart the follicular phase all over again under FSH influence [9, 14] (Fig. 7.2).

### 7.3.2 Effects of Female Sex Hormones on Bone

The female reproductive system plays a major role in the maintenance of the skeletal bone integrity from menarche to menopause. The skeleton is one of the main target tissues for estrogen, and to a lesser extent, progesterone. Bone cells contain estrogen and progesterone receptors that positively stimulate bone formation and suppress bone resorption. Thus, they are key regulators of bone turnover, adjusting the bone mass "set point" by keeping in balance the quantity of bone being formed with the amount that is reabsorbed (coupling effect) [10, 15]. Sex hormone receptors have been found in growth plate chondrocytes (cartilage cells) during puberty [17]. At the early stages

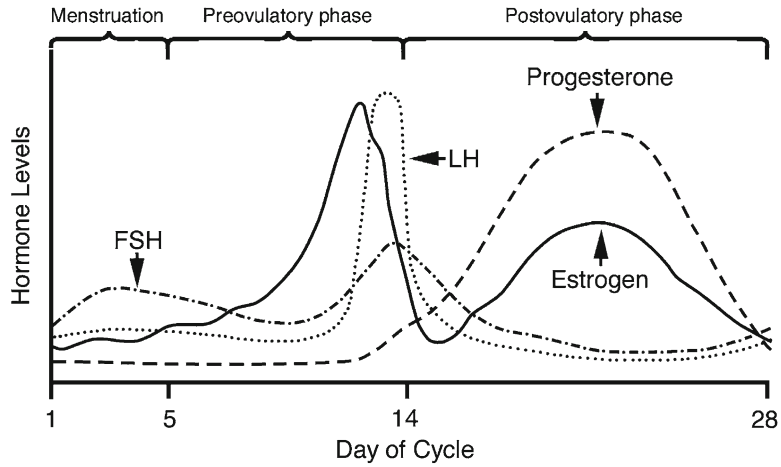



**Fig. 7.2** Menstrual cycle. FSH, follicle stimulating hormone; LH, lutenizing hormone

of puberty, sex hormones stimulate longitudinal bone lengthening of the diaphyses (shafts of long bones) resulting in the "pubertal growth spurt" [6]. Estrogen acts indirectly through inhibiting bone resorption by osteoclasts while increasing the activity of bone forming cells, osteoblasts (discussed further below in Sect. 7.3.3) [4, 14]. However, at the late stages of puberty the sex-specific hormones, primarily estrogen, play a large role in the closure of growth plates. Estrogen, therefore, highly influences the final height achieved in females [14].

Estrogen and progesterone not only play an essential role on homeostasis of the skeleton by exerting their direct effects through receptors on bone cells but also exert indirect effects on other systemic hormones. These sex hormones work in conjunction with other hormones and steroids to further facilitate bone development. For example, estrogen regulates circulating levels of growth hormone (GH) and insulin growth-like factor (IGF-1); these two hormones stimulate longitudinal bone growth [1, 17]. In a cross-sectional study conducted on children, serum IGF-1 levels were positively associated with bone growth [6]. Insulin growth-like factor receptors (IGF-1R) are also located in the kidneys, where they are associated with the production of the hormonal form of vitamin D [17]. Environmental factors can also stimulate bone formation. Mechanical loading of bone exerts a positive effect on bone formation at points of stress, in a process defined by Wolff's Law. A cross-sectional study found hip and spine BMD values 30–40 % greater in female gymnasts compared to long-distance runners. This implies that the higher impact forces generated in gymnastics (10–12 times body weight) as compared to running (3–5 times body weight) positively affect BMD [19]. Bone formation can also be altered by external agents such as certain metals, pesticides, and components in cigarette smoke [19].

To summarize, the normal, monthly occurrence of the female menstrual cycle represents an intricate interaction of three organs – hypothalamus, anterior pituitary, and ovaries – resulting in a feedback mechanism whereby several secreted hormones influence each other to effect maturation and ovulation of an egg (follicle). Next, the endometrium becomes prepared to receive the mature follicle and, if no implantation occurs, then sloughing of the uterine lining manifests itself by menstrual bleeding and the cycle repeats. Disturbances in any portion of this hormonal loop between the reproductive system and these two specific centers in the brain could, in turn, alter the quality/quantity of menstruation and secondarily affect bone deposition, especially during the critical time of initial peak bone mass acquisition involved with the pubertal/adolescent stage [15, 20, 21].

### 7.3.3 Bone Composition, Physiology, Function, and Interaction with Ovarian Hormones

Skeletal tissue is one of the hardest and strongest tissues in the body. Bone is mineralized connective tissue comprised of specialized cells, along with noncellular substances, organic matrix (35 % by weight), and inorganic mineral (65 %). Two key types of bone cells are osteoblasts, which are involved in bone formation, and osteoclasts, which are involved in bone resorption. Components making up the organic matrix include glycoproteins, collagen (Type I), elastin, and protein (90 % collagen), bathing in a sea of gelatin-like mucopolysaccharide (ground substance). Bone can be classified into two categories: cortical, representing 80 % of bone mass, and trabecular, representing only 20 % of bone mass but comprising 80 % of bone surface area [19].

Collagen fibers in the bony skeleton represent specific sites where inorganic calcium/phosphate hydroxyapatite crystals are deposited prior to the mineralization process of bone. Ninety-eight to ninety-nine percent of the body's total calcium is sequestered in the skeletal framework, which serves as a mineral bank and releases calcium into the bloodstream to keep serum calcium levels constant. Because the quantity of calcium is tightly regulated, the body will rob calcium from its main reservoir, bone, to maintain adequate blood levels in order to carry out essential, vital tasks such as blood clotting and muscle contraction [2, 4, 22]. Calcium, however, cannot be absorbed and incorporated into bone unless vitamin D is readily available [2, 11]. Ovarian hormones play an important role in the metabolism of calcium and vitamin D. Indeed, estrogen helps to regulate the absorption of calcium, subsequently contributing to bone formation, as already discussed above. In effect, any irregularities in the menstrual cycle will negatively affect bone deposition in the long run by negating estrogen's positive influence on calcium balance. Additionally, with the loss of protection from estrogen, osteoclasts will be affected to a greater extent than osteoblasts, resulting in an uncoupling effect. This dissociation between osteoblasts and osteoclasts causes undesirable bone loss, which could manifest itself as early osteoporosis [4, 9, 23, 24] (Fig. 7.3).

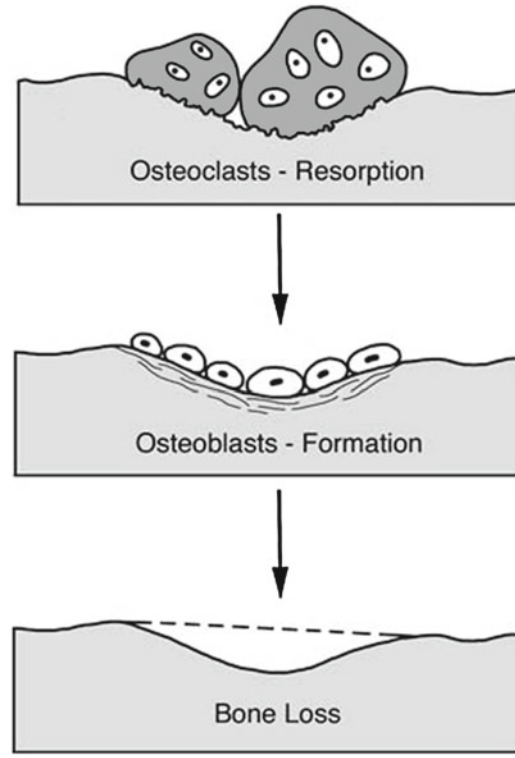



**Fig. 7.3** Bone remodeling

### 7.3.4 Peak Bone Mass

Peak bone mass (PBM) signifies the maximal quantity of bone which can be gained primarily during the adolescent years (with the critical window being between 9 and 20 years), while the skeleton is undergoing an accelerated growth in both size and density. By age 7, females have reached 80 % of their adult height but only 40 % of their PBM [2]. This "adolescent growth spurt" occurs 1–2 years preceding the rapid deposition of bone into the skeleton. In females, up to 90 % of PBM is accumulated by 18 years of age [3, 10]. After this prime period, bone can still continue to grow in terms of strength and density, up until about age 25–35, at which time true PBM is

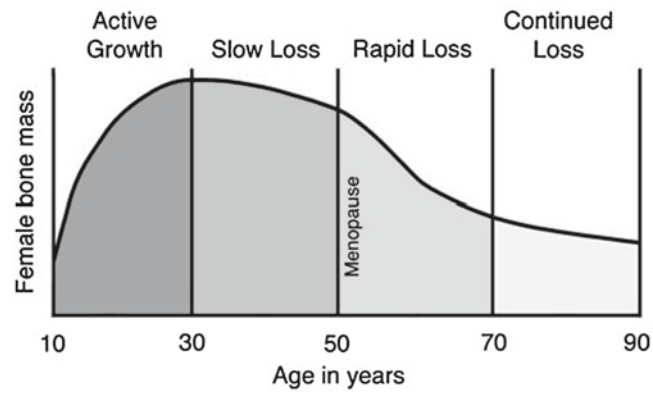



**Fig. 7.4** Rate of bone loss through a women's lifetime (Fig 14.2 in Ist ed)

reached [2, 3, 25]. However, some researchers have shown that this PBM acquisition may occur as young as the late teens [7]. After attainment of peak bone mass, measures to maintain bone density are of paramount importance since physiologic bone loss will inevitably occur gradually over time [2, 7, 25, 26]. In fact, prior to reaching menopause in their fourth or fifth decade, females lose about 0.3 % of their entire skeleton each year after the final acquisition of peak bone mass [2] (Fig. 7.4).

The quantity of bone deposited in the skeleton is influenced by both genetics (uncontrollable) and environmental (controllable) factors [3, 7]. Mechanical loading in the form of weight-bearing/resistance exercise; appropriate and adequate nutrition ( i.e., sufficient consumption of key bone building nutrients such as calcium, magnesium, zinc, vitamins C and D); and normal, regular menses all contribute to optimal bone health [2, 8–10, 12, 24]. Endocrine function is highly dependent on the amount of available energy. Energy availability is the quantity of energy remaining after physiological energy is utilized (i.e., energy for everyday movement and activity). As long as energy availability is maintained, the endocrine system will function optimally with normal hormone serum levels. However, without adequate energy the endocrine system is altered, primarily affecting the hypothalamic production of hormones [27]. Insufficient energy will adversely affect the pulsatile secretion of LH. This causes a decrease in hormones such as estrogen, testosterone, GH, and IGF-1. On the other end of the spectrum, an extreme excess of available energy, as seen in obesity, will tend to cause hormone level alteration as well [28]. In short, any aberrant serum levels of sex hormones will alter the menstrual cycle and ultimately affect bone balance [29].

A delay in menarche (initiation of menses), dysmenorrhea (irregular menstruation), oligomenorrhea (insufficient number of cycles per year), or amenorrhea (absence of menses) all will interfere with the final attainment of peak bone mass by causing more rapid bone loss. Bone mass density steadily declines, particularly in non-weight-bearing limbs, as the number of missed menses increases and this loss of BMD may not be completely reversible [25]. Irregular menses is primarily due to a deficiency of estrogen and, if not corrected promptly, ultimately will result in premature osteoporosis [2, 30]. Such menstrual irregularities/disorders could be caused by various factors: extremely low weight, rapid weight loss, excessively intense exercise, and disordered eating, along with associated extreme physical and/or psychological stress [6, 16, 31]. Specifically, women with menstrual disorders will lose as much as 2 % of their skeleton per year, almost tenfold more than the usual, natural rate. In other words, exercising excessively can negatively affect both the reproductive and the skeletal systems, altering the body's normal hormonal milieu and causing a reduction in bone mass [32]. One study has found that total, vigorous, intense training for more than 8 h per week could lead to amenorrhea and, subsequently, osteoporosis with its inherent risks [18].

Physical activity affects the skeleton differently during the various stages of puberty [7]. Studies have shown that estrogen increases skeletal sensitivity to mechanical loading, suggesting that early and mid-puberty are optimal times for skeletal benefit as far as bony deposition is concerned [33]. Female athletes, particularly in sports such as running, gymnastics, and swimming, reach menarche later than non-athletes. A study conducted on sisters found menarche occurred later in swimmers than their sedentary siblings, thus may potentially affect bone mass negatively [28].

### 7.3.5 Results of an Abnormal Menstrual Cycle on Peak Bone Mass

Even though genetic factors significantly influence bone mass and density, other factors such as nutrition, exercise, disease, drugs, and age at menarche can play a role as well. Abnormal menses can be classified into two different categories. Primary amenorrhea is defined as the absence of menstruation by age 15 or within 5 years after breast development. Secondary amenorrhea is termed as the absence of three or more consecutive menses following menarche. It has been estimated that 1 in 5 active women have some form of abnormal menses [34].

As discussed above, insufficient quantity of available energy will cause altered endocrine function resulting in abnormal hormone levels, which in turn affect the menstrual cycle. Studies have found menstrual disorders occurring in 24–26 % of runners [35]. Loucks found that in females, after 5 days of low energy availability, there is a decrease in LH pulse frequency [36]. Similarly, the prevalence of amenorrhea increases from 3 to 60 % in long-distance runners as the weekly distance ran is increased from less than eight miles to over 70 miles [37]. The presence of altered menstruation in adult female athletes has been reported to range from 12 to 79 %, with adolescent athletes having even higher rates [4]. When facing the problem of irregular menses occurring in an adolescent female, causes for secondary amenorrhea should be identified such as pregnancy, endocrine disorders, anatomic defects, or tumors of the involved organs, which can interfere with the hypothalamic-pituitary-ovarian axis [7, 16, 18, 25]. After these potential pathologic causative factors are excluded, then information regarding eating habits, weight control behavior, and exercise patterns should be obtained. This detailed history is used to evaluate whether the affected young patient is at risk for premature osteoporosis due to the female athlete triad (diagnosis of exclusion) – a constellation of amenorrhea, disordered eating, and osteoporosis. More importantly, it must be understood that the amount of bone which is lost may not be totally recovered, even upon resumption of normal menses [32].

Females at a higher risk for amenorrhea are usually involved in sports requiring an aesthetic/athletic look or those needing to weigh less for better performance (gymnasts, dancers, runners, divers, etc.) [6, 10, 29, 38]. The afflicted female athlete's young, fragile bones are at two- to threefold increased risk for stress and/or frank fractures, similar to older, perimenopausal women in their 40s and 50s, especially if the duration of absent menses is longer than 6 months [18, 30, 38]. Bone mass density further declines as the number of missed menses increases [39]. Stress fractures are more common in female athletes with amenorrhea, with a relative risk of fracture four to five times greater in amenorrheic athletes [30]. However, there is a positive effect of certain types of weight-bearing exercise on bone density. Zanker et al. studied a group of retired female gymnasts and found they had higher BMD compared to women who did not exercise or participate in sports [40]. Even though involvement in a regular program of exercising, especially under high loads such as gymnastics, may partially offset the osteopenic bone caused by being amenorrheic, this is still far from being enough if an inadequate amount of calories is consumed. In fact, research has shown that bone mass in females who are sedentary, eat well, and have normal monthly cycles is actually higher than those athletes who exercise to the point of losing their menses [2]. Similarly, another study has

**Table 7.1** Classification of bone mineral density according to the World Health Organization

| | | |
|---|---|---|
| Normal bone density | <1 | Standard deviation (SD) below the mean |
| Osteopenia | 1–2.5 | SD below the mean |
| Osteoporosis | >2.5 | SD below the mean |
| Severe osteoporosis | >2.5 | SD below the mean/ fragility fracture(s) |

shown that increases in circulating FSH levels above 20 mIU/L are linked to progressive bone loss in perimenopausal women, again stressing the importance of maintaining regular menstruation to preserve bone density [18].

Currently, dual energy x-ray absorptiometry (DEXA) is the "gold standard" for measurement of bone mineral density (BMD) to assess bone heath, and thus, it should be used to help diagnose osteoporosis and monitor progress of treatment [2, 10, 12, 16, 24–26, 38]. This technique uses emission of x-rays at two separate energy levels to distinguish bone from the surrounding soft tissue with very low radiation exposure. The time for scanning with this type of device is brief and BMD measurements are more accurate (within 1–2 %) as compared to other methods [2, 10]. If the DEXA scan result for BMD is between 1.0 and 2.5 standard deviations (SD) below the mean for young adults, then bone is considered osteopenic. Even at this level, there exists 2–2.5 times more risk of spine or hip fractures due to increased fragility of the skeleton. On the other hand, bone is deemed osteoporotic (with associated higher fracture risk) if the BMD is equal to or greater than 2.5 SD from the mean [2, 10, 16, 26] (see Table 7.1). One study demonstrated that reduced urinary sex steroid hormones are found during the luteal phase of the menstrual cycle in premenopausal women with lower BMD (10th vs. 50th–75th percentile), again linking monthly menses to bone mass acquisition [19]. As an adjunct, other methods to assess bone metabolism include serum osteocalcin and alkaline phosphatase (for bone formation) and urine collagen breakdown products (telopeptide crosslinks) as indicators of resorption and current rate of bone loss, respectively [2, 10, 12].

The next step in prevention of early onset of osteopenia/osteoporosis in an adolescent female is to correct any offending factor contributing to the rapid bone loss. First steps should include ensuring adequate and appropriate dietary intake of nutrition, reducing quantity of training (5–20 % less), increasing body weight slowly and gradually (generally 5 % of body weight increase or one-half to one pound per week), and, finally, conferring with her to confirm that she is maintaining the correct goal weight [6, 32, 38]. Proper nutrient intake can greatly aid in the prevention of bone loss. The benefits of calcium supplementation have been shown to be more beneficial before the onset of puberty [41]. A study conducted on 8-year-old prepubertal girls found that by increasing the daily calcium dose from 700 to 1,400 mg, this can raise the BMD by 58 % compared to the placebo group after 1 year of supplementation [42]. Regulation of menses can be obtained by hormonal replacement therapy (HRT) – the oral contraceptive pill is a popular form of this type of pharmacological treatment [32]. However, the ultimate effectiveness of hormonal replacement remains controversial. Oral estrogen leads to suppression of GH and IGF-1 concentrations as already discussed above; these two hormones are very vital in bone development [36]. Specific lifestyle changes are recommended as well: avoidance of inactivity, refraining from cigarette smoking, and minimizing drinking alcohol [1]. In terms of dietary intake, avoid drinking caffeinated beverages and ingesting excess protein is recommended because both of these substances contribute to calcium wasting and, thus, could secondarily affect bone formation. Adequate intake of calcium and vitamin D will also aid in bone building. Finally, appropriate exercise is recommended, especially the weight-bearing type (loading in the erect, standing position) with at least some impact such as walking or jogging. As discussed previously, bone will build at points of mechanical stress (Wolff's Law). However, the mode of exercise needs to be considered as not to overload the bone, causing injury and eventually skeletal failure [2, 3, 9–12, 16, 19, 25, 38]. Studies have shown that jumping is an activity sufficient to stimulate bone growth, because one jump produces two rapid strain energies (taking off and landing). The ground

reaction force from landing and muscle activation with this type of maneuver has been shown to be enough of a stimulus for adequate bone growth [43].

Evaluation and treatment for females with menstrual disturbances include conducting a detailed history and physical exam, plus possibly ordering certain laboratory tasks. It is also prudent to approach management of the female at risk by compiling a multidisciplinary team, involving an internist to rule out chronic disease, a psychiatrist/psychologist to address associated eating disorders and other psychological issues, a gynecologist to rule out anatomic abnormalities and for prescribing HRT, a dietician for nutritional counseling, and an endocrinologist to address other possible pathology interfering with the hormonal feedback loop [18, 29]. Additionally, it is very important to involve family members, teachers, trainers, other players, and coaches to solicit additional support for the involved patient, ultimately to help regulate and maintain normal bodily functions in the female athlete [29, 30] (Fig. 7.1).

## 7.4 Future Directions and Concluding Remarks

The role of the female menstrual cycle in terms of its contribution toward acquisition of peak bone mass has been fairly well elucidated. A regular, well-functioning monthly cycle is of paramount importance in bone deposition during the adolescent growth spurt and then in prevention of bone loss after peak bone mass is achieved in the second decade of life. The main hormone involved in bone regulation is estrogen which is secreted by the ovaries, affecting both the osteoclasts and the osteoblasts, keeping these two types of bone cells in balance in terms of their function on skeletal resorption and formation, respectively. Additionally, the organs involved with controlling the menses are the hypothalamus, anterior pituitary and, of course, the ovaries. Each organ secretes different hormones, which interact in a complex, loop-type feedback mechanism to regulate the monthly female cycle, in order to maximize bone deposition and minimize bone loss. If menarche is delayed or if menstrual dysfunction occurs or menses disappears entirely, then the protective mechanism of estrogen on bone is lost. If this menstrual disturbance is not corrected fairly promptly, it will eventually result in osteopenia or frank osteoporosis, ultimately increasing one's susceptibility to fractures. In fact, osteoporotic prevention starts as early as the initial onset of menarche during adolescence. Additionally, other factors, such as lifestyle habits (nutrition, training, etc.), also play a very essential part in contributing to bone building (formation) or bone loss [18, 25]. As such, measures taken to ensure that the monthly female menstrual cycle is functioning correctly and optimally are of utmost importance to positively influence the final attainment of peak bone mass [18].

## References

1. Elgan C, Dykes AK, Samsioe G. Bone mineral density changes in young women: a two year study. Gynecol Endocrinol. 2004;19(4):169–77.
2. Lane J, Russell L, Khan S. Osteoporosis. Clin Orthop Relat Res. 2000;March(372):139–50.
3. National Institutes of Health Osteoporosis and Related Bone Diseases-National Resource Center. Osteoporosis: peak bone mass in women, Mar 2005; URL: http://www.osteo.org/newfile.asp?doc=r701i&doctitle=Peak+Bone+Mass+in+Women&doc.
4. Resnick D, Manolagas S, Niwayama G. Histogenesis, anatomy, and physiology of bone. Anatomy and physiology. Philadelphia: Saunders; 1989. p. 16–28.
5. Perez-Lopez FR, Chedraui P, Lopez-Cuadros JL. Bone mass gain during puberty and adolescence: deconstructing gender characteristics. Curr Med Chem. 2010;17:453–66.
6. Clarke BL, Khosla S. Female reproductive system and bone. Arch Biochem Biophys. 2010;503:118–28.
7. Brown S. Better bones at every age. Lets Live Magazine, Oct 2000.
8. Lebrun C. Menstrual cycle dysfunction. American College of Sports Medicine, Oct 2000
9. Nichols D, Bonnick S, Sanborn C. Bone health and osteoporosis. The Athletic Woman. 2000;19(2):233–49.
10. United States Department of Health and Human Services. Bone health and osteoporosis: a report of the surgeon general. Chapter 6: Determinants of bone health, Oct 2004; URL: http://www.surgeongeneral.gov/library/bonehealth/chapter_6.html.
11. Lane JM, Nydick M. Osteoporosis: current modes of prevention and treatment. J Am Acad Orthop Surg. 1999;7(1):19–31.

12. Altruis Biomedical Network. Anatomy and physiology. E-Gynecologic.com 2000; URL: http://www.e--gynecologic.com/index.html.
13. Guyton A, Hall J. Female physiology before pregnancy; and the female hormones. In: Schmitt W, Gruliow R, Faber P, Norwitz A, Shaw P, editors. Textbook of medical physiology. 10th ed. Pennsylvania: Saunders; 2001. p. 929–43.
14. Berne R, Levy M, Koeppen B, Stanton B, editors. The reproductive glands. Physiology. 5th ed. St. Louis, MO: Mosby; 2004. p. 920–78.
15. Harmon K. Evaluating and treating exercise-related menstrual irregularities. Phys Sportsmed. 2002;30(3): 29–35.
16. Khan A, Syed Z. Bone densitometry in premenopausal women: synthesis and review. J Clin Densitom. 2004;7(1):85–92.
17. Nelson L. Amenorrhea. eMedicine, May 2005. URL: http://www.emedicine.com/med/topic117.htm.
18. Duncan CS, Blimki CJR, Cowell CT, Burke ST, Briody JN, Howman-Giles R. Bone mineral density in adolescent female athletes: a relationship to exercise type and muscle strength. Med Sci Sports Exerc. 2002;34:286–94.
19. Whiting WC, Zernicke RF. Biomechanics of musculoskeletal injury. Champaign, IL: Human kinetics; 1998.
20. Csermely T, Halvax L, Schmidt E, Zambo K, Vadon G, Szabo I, et al. Occurrence of osteopenia among adolescent girls with oligo/amenorrhea. Gynecol Endocrinol. 2002;16(2):99–105.
21. Galuska DA, Sowers MR. Menstrual history and bone density in young women. J Womens Health Gend Based Med. 1999;8(5):647–56.
22. Altruis Biomedical Network. Osteoporosis. E-Gynecologic.com 2000; URL: http://www.e- gynecologic.com/index.html.
23. Thys-Jacobs S. Micronutrients and the premenstrual syndrome: the case for calcium. J Am Coll Nutr. 2000;19(2):220–7.
24. Templeton K. Secondary osteoporosis. J Am Acad Orthop Surg. 2005;13(7):475–86.
25. Erickson S, Sevier T. Osteoporosis in active women: prevention, diagnosis, and treatment. Phys Sportsmed. 1997;25(11):61–72.
26. Theodorou S, Theodorou D, Sartoris D. Osteoporosis: a global assessment of clinical and imaging features. Orthopaedics. 2005;28(11):1346–53.
27. Jayasinghe Y, Grover SR, Zacharin M. Current concepts in bone and reproductive health in adolescents with anorexia nervosa. BJOG. 2008;115(3):304–15.
28. Brooks GA, Fahey TD, Baldwin KM. Exercise physiology: human bioenergetics and its applications. 4th ed. New York, NY: McGraw-Hill; 2005.
29. Women's Health Orthopaedic Edition. Assessing health in young female athletes. 2000 November–December;3(6):205–7.
30. Slemenda C, Longcope C, Peacock M, Hui S, Johnston C. Sex steroids, bone mass, and bone loss. J Clin Invest. 1996;97(1):14–21.
31. Balasch J. Sex steroids and bone: current perspectives. Human Reprod Update. 2003;9(3):207–22.
32. Warren MP, Stiehl AL. Exercise and female adolescents: effects on the reproductive and skeletal systems. J Am Med Womens Assoc. 1999;54(3):115–20.
33. Devlin MJ. Estrogen, exercise, and the skeleton. Evol Anthropol. 2011;20(11):54–61.
34. Beals KA, Hill AK. The prevalence of disordered eating, menstrual dysfunction, and low bone mineral density among US collegiate athletes. Int J Sport Nutr Exerc Metab. 2006;16(1):1–23.
35. Sanborn CF, Albrecht BH, Wagner Jr WW. Athletic amenorrhea: lack of association with body fat. Med Sci Sport Exerc. 1987;19:207–12.
36. Lissett CA, Shalet SM. The impact of dose and route of estrogen administration on the somatotropic axis in normal women. J Clin Endocrinol Metab. 2003; 88:4668–72.
37. Sanborn CF, Martin BJ, Wagner WW. Is athletic amenorrhea specific to runners? Am J Obstet Gynecol. 1982;143:859–61.
38. Hobart J, Smucker D. The female athlete triad. Am Fam Physician. 2000;61(11):3357–64.
39. Keen AD, Drinkwater BL. Irreversible bone loss in former amenorrheic athletes. Osteoporos Int. 1997; 7:311–5.
40. Zanker CL, Osborne C, Cooke CB, Oldroyd B, Truscott JG. Energy balance, bone turnover and menstrual history of sedentary female former gymnasts, aged 20–32. Osteoporos Int. 2004;15:145–54.
41. Johnston CC, Miller JZ, Slemenda CW, Resiter TK, Hui S, Christian JC, et al. Calcium supplementation and increases in bone mineral density in children. N Engl J Med. 1992;327:82–7.
42. Bonjour JP, Carrie AL, Ferrari S, Clavien H, Slosman D, Theintz G, et al. Calcium enriched foods and bone mass growth in prepubertal girls: a randomized, double-blind, placebo-controlled trial. J Clin Invest. 1997;99:1287–94.
43. Bailey CA, Brooke-Wavell K. Exercise for optimizing peak bone mass in women. Proc Nutr Soc. 2008; 67:9–18.

# Part II

# Disordered Eating Issues in the Active Female

# Menstrual Dysfunction Screening and Management for Active Females

8

Jacalyn J. Robert-McComb and Jennifer J. Mitchell

**Abstract**

There is a wide spectrum of attitudes among women concerning her menstrual cycle. Most athletes would prefer to not have to deal with the associated hygiene issues. This sets the stage for feeling that lack of menses (amenorrhea) is a good thing. Unfortunately, that is not necessarily the case. The menstrual cycle can be considered a vital sign related to the athlete's overall health. Those athletes not having regular menses are likely exhibiting functional hypothalamic amenorrhea (FHA), a diagnosis of exclusion. This is one component of the female athlete triad. The FHA comes from inadequate energy availability and resultant diminished sex hormones. This also causes changes in bone health and puts the athlete at risk for stress fractures, suboptimal peak bone mass density, and future osteoporosis. Through education of female athletes and those in her circle of influence, it is possible to allow realization that amenorrhea is a medical issue and needs further attention. Determination of optimal screening for this condition has yet to be accomplished, but this chapter describes who should be screened and provides examples of questionnaires, which may be useful in sorting out an athlete's needs. Management can be as simple as improving the athlete's nutrition and/or decreasing her exercise intensity, but it can often be difficult to convince the athlete and her coaches of these steps.

**Keywords**

Amenorrhea • Menstrual dysfunction • Oligomenorrhea • Functional hypothalamic amenorrhea

J.J. Robert-McComb, Ph.D., FACSM, ACSM P.D. (✉)
Department of Health, Exercise, and Sports Sciences, Lubbock, TX, USA
e-mail: jacalyn.mccomb@ttu.edu

J.J. Mitchell, M.D., F.A.A.F.P.
Department of Family and Community Medicine, Texas Tech University Health Sciences Center School of Medicine, Lubbock, TX, USA
e-mail: jennifer.mitchell@ttuhsc.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_8, © Springer Science+Business Media New York 2014

## 8.1 Learning Objectives

Upon completion of reading this chapter, the reader will:

1. Understand the importance of screening for menstrual dysfunction in the active female
2. Be able to describe how insufficient energy availability can impact menstrual function and bone health
3. Be able to list groups of athletes in whom screening for menstrual dysfunction is essential
4. Have a resource of potential screening questions for menstrual dysfunction
5. Understand the first step in management of functional hypothalamic amenorrhea in an athlete to increase energy intake and to improve overall energy availability, in order to restore cyclic menstruation

## 8.2 Introduction

Screening for menstrual disorders in athletes is more complex that it appears to be. Historically, the simple question was whether the individual is having normal menstrual cycles or periods. If yes, then that was the end of screening. If no, the athlete was referred to the physician. With improved understanding of the female athlete triad and the components involved, screening has become more complex. In addition to menstrual status, those responsible for the health of the female athlete must also evaluate the other two components of the triad; whether the athlete has optimal energy availability, or may have indications of disordered eating or a pathologic eating disorder, and the athlete's bone health. Evaluation of one component of the female athlete triad should not occur in isolation from the other two components.

Terms that are used in this chapter, when discussing menstrual screening or the routine screening of adolescent females by their primary health care provider, include the following: primary amenorrhea, secondary amenorrhea, and functional hypothalamic amenorrhea (FHA). Even though these terms have been described in previous chapters, we will briefly define these terms so that you can more fully understand the recommendations in this chapter.

Amenorrhea is the absence of menstruation or a woman's monthly period. Primary amenorrhea is when a girl has not yet started her monthly periods by age 15 and she has gone through other normal changes that occur during puberty. Secondary amenorrhea occurs when a woman who has been having normal menstrual cycles stops getting her periods for 6 or more months (some sources state 3 months, although 6 months is more common). Note, however, that women who are pregnant, breastfeeding, or in menopause are not considered to have secondary amenorrhea. Functional hypothalamic amenorrhea (FHA) is a reversible form of gonadotropin-releasing hormone (GnRH) deficiency commonly triggered by stressors such as excessive exercise, nutritional deficits, or psychological distress.

The Office on Women's Health, Department of Health and Human Services, suggests that the following physical and behavioral/emotional characteristics as listed in Table 8.1 be used during the routine screening of adolescent and preadolescent patients by their primary care provider for the detection of issues related to the triad.

As will be discussed in Chap. 13, there is not yet an agreed upon optimal timing or method of screening for female athlete triad disorders. An energy deficit in a female athlete may cause a spectrum of menstrual dysfunction, either subtle or obvious, which may then have an impact on bone health. This leads to the realization that a comprehensive menstrual history may be needed in all athletes.

## 8.3 Research Findings

### 8.3.1 Why Screen for Menstrual Dysfunction

It is important to screen female athletes for menstrual disorders since a normal menstrual cycle is a key vital sign to one's health. An individual, depending on her level of education and maturity, may not recognize an abnormality in the menstrual cycle. Young girls, just reaching maturity, may look

**Table 8.1** A primary care provider reference tool for screening female adolescent and preadolescent patients

| *Physical behavioral/emotional* | |
|---|---|
| • Primary or secondary amenorrhea | • Recurrent or excessive dieting when not overweight |
| • Weight loss greater than 10 % | • Eating in secret |
| • Failure to gain expected weight during the adolescent growth spurt | • Eating large quantities of food in a short period of time |
| • Overweight | • Excessive concerns about perceived body image that are incongruous with actual weight |
| • Lanugo hair | • Compulsive or overly rigid exercising |
| • Hypothermia | • Depression |
| • Dry hair or skin, dehydration | • Use of self-inducted vomiting, laxatives, starvation, diuretics, or other extreme measures to lose weight |
| • Weight fluctuation in a short period of time | |
| • Bloating and abdominal discomfort | |
| • Damaged teeth | |
| • Enlargement of lymph or salivary glands | |

Source: From Office on Women's Health. Screening for Eating Disorders-Tips for Health Care Providers

upon discussion of menstruation and sexuality as taboo. Others may view amenorrhea as a positive benefit of not having to deal with monthly hygiene issues [1].

An athlete with a menstrual disorder may be manifesting one or more components of the female athlete triad. It now appears that low energy availability is the key factor involved in the triad that then leads to menstrual dysfunction and issues with skeletal health [2]. When the deficit is extreme, the body attempts to reduce the deficit by suppressing physiologic functions essential for growth, development, and health [3].

Energy availability is the amount of energy remaining once exercise energy expenditure is subtracted from energy intake (EA=EI-EEE). It is the amount of energy remaining for all other metabolic processes [4].

The longer a menstrual abnormality is allowed to go on untreated, the greater the long-term consequences [5]. Also, athletes who begin sport-specific training before menarche have been shown to have significantly later menarche [6]. This may have implications related to both reproductive and skeletal health. Prevention and early detection of female athlete triad disorders are of utmost importance for the health of young female athletes [6, 7].

### 8.3.2 Who Should Be Screened for Menstrual Dysfunction

The main populations to screen include the following groups [1, 8–10]:

1. Adolescents involved in vigorous exercise with primary amenorrhea:
   (a) Normal secondary sexual development but no menarche by 15 years old
   (b) No menarche within 5 years after breast development that occurred less than 10 years old
   (c) Failure of the thelarche (breast development) by 13 years old
2. Athletes with previously regular cycles, at any age, with secondary amenorrhea or the lack of menses for 3 continuous cycles after beginning menses are good candidates for screening.
3. Athletes with oligomenorrhea, less than 9 cycles per year, are candidates for screening.
4. An intensively exercising, reproductively mature woman interested in conception is a viable candidate for screening.

### 8.3.3 Types of Menstrual Dysfunction

Aside from pregnancy and menopause, causes of secondary amenorrhea are most likely due to:

1. Thyroid dysfunction
2. Elevated prolactin
3. Ovarian failure

4. Polycystic ovarian syndrome (PCOS)
5. Hypothalamic amenorrhea

Thyroid dysfunction and elevated prolactin are easily sorted out by blood testing for thyroid-stimulating hormone (TSH) and prolactin (PRL) levels. An athlete with ovarian failure will have elevated follicle-stimulating hormone (FSH) levels and very low or absent estrogen. PCOS and hypothalamic amenorrhea are typically differentiated based on clinical presentation as they both are likely to have normal FSH levels. The athlete with PCOS will usually be at or above a normal Body Mass Index (BMI) and will likely be hirsute and may show signs of insulin resistance [9, 11, 12].

### 8.3.4 Functional Hypothalamic Amenorrhea

In athletes, amenorrhea is much more common than in non-exercising controls with prevalence reported from 3 to 69 % compared to 2–5 % in the general population [2, 6, 13].

Prevalence is typically an estimate as it is difficult to gain accuracy due to inconsistencies in studies including various definitions of amenorrhea, selection bias, underreporting, lack of education on what is normal versus abnormal, various competition levels, sports disciplines with varied intensity, and frequency of training [6, 14].

However, it is important to remember the series of events appears to be related to a deficit in energy availability causing menstrual dysfunction and subsequent issues with skeletal health. Exercise does not have an impact on LH pulsatility beyond the impact of its energy cost on energy availability [4, 15–17].

Loucks provides convincing evidence for the energy availability hypothesis related to menstrual dysfunction in athletes. Additionally, she provides evidence against the original theories concerning body composition and exercise stress. The energy availability hypothesis states if the brain energy requirements are not met, an alteration in brain function occurs which disrupts the GnRH pulse generator [15]. Regulation of puberty and reproductive function depends on interactions at specific levels of the hypothalamic-pituitary-ovarian (HPO) axis. The GnRH "pulse generator" neurons in the hypothalamus secrete GnRH every 60–90 min. This hormone causes release of gonadotropins (luteinizing hormone (LH) and follicle-stimulating hormone (FSH)) from the pituitary gland. These, in turn, cause release of progesterone and estrogen from the ovaries. These two end hormones are key to regular, ovulatory menstrual cycles [12, 18, 19].

Deficiency in GnRH pulsatile secretion leads to hypothalamic amenorrhea.

Since hypothalamic amenorrhea in the athlete becomes a diagnosis of exclusion, it is often termed functional hypothalamic amenorrhea (FHA) because it is a functional suppression of reproduction [12]. Because FHA is the typical menstrual abnormality seen in athletes, it will be the focus of discussion concerning treatment of menstrual disorders for this chapter.

### 8.3.5 When to Screen for Menstrual Dysfunction

The answers concerning when to screen athletes for menstrual disorders are similar for screening of disordered eating (DE) and eating disorders (ED) as discussed in Chap. 13. Evaluation of one component of the female athlete triad should not occur in isolation from the other two components. There is not yet an agreed upon optimal timing or method of screening for any component of female athlete triad disorders [2, 14]. Screening should also be economical and time efficient and should create an environment that will not cause an athlete to minimize or deny certain medical conditions [14].

Options for screening include during pre-participation examinations (PPEs) for competitive athletes, during clinical presentation of the athlete for routine health care (i.e., well woman examination) or for illness or injury, and incidental observation by an athletic trainer, parent, friend, coach, or administrator [2, 7]. Since menstrual dysfunction is often seen as related to sexuality, it can be a very sensitive topic and is not as likely to be incidentally discussed as energy availability might be. This leaves incidental observation the least likely

scenario. Because of this, it is probably best to have a short screening tool utilized by those who interact on a routine basis with the athletes, such as athletic trainers, personal trainers or other gym personnel, and coaches who do not have availability of athletic trainers. The questionnaire tool could be distributed to all female athletes at specific times during the athletic year and would act as a first step to identify a possible disorder in menstruation. It ideally would also contain questions concerning nutrition and bone health. It would be easy to score and if the athlete screens positively with the tool, she would then be referred to a team physician or other designated intervention team for the second stage of screening, an in-depth evaluation.

### 8.3.6 Screening Questions

No validated tools to screen for menstrual dysfunction exist. Several pre-participation examination forms have from one to six questions included on the form [20, 21]. Tools used to screen for disordered eating and eating disorders may include a few questions about menstrual health. It is likely best to have a supplemental form, apart from the PPE form, in order to effectively screen for menstrual dysfunction. Screening should also include questions related to energy availability and skeletal health. Appendices 1–4 [22] provide examples of tools used to screen female athletes for various components of the female athlete triad. The ideal will be to develop a standardized form to screen for the female athlete triad that is then validated. Any athlete who screens positively would need further evaluation by a physician.

### 8.3.7 Evaluation of Menstrual Dysfunction Beyond Screening

The in-depth evaluation with the physician or intervention team should include a routine health history, a comprehensive menstrual and obstetrical and gynecologic history, an appropriate examination, and an evaluation of bone mineral density. The physician could obtain the sexual history, in order to avoid an uncomfortable setting for the athlete and her athletic trainer and/or coaches [2, 7, 9, 11, 13, 14]. Some screening tools and PPE forms currently in existence already have a variety of questions concerning menstrual history. However this varies from one question to several to an entirely separate form [20, 21, 23–25].

The question still remains concerning what makes up an adequate screening tool compared to an extensive obstetrical and gynecologic history. None of the existing forms have been validated for menstrual dysfunction screening. In a study done of NCAA Division 1 schools in 2003, 138 of 170 schools responded and 79 % stated they did screen for menstrual disorders (MD). Only 24 % of those used a comprehensive menstrual history questionnaire. A menstrual disorder treatment protocol was used by 33 %. Of the responding schools, 60 % screened for eating disorders (ED). However, less than 6 % used a structured interview or a validated questionnaire. The conclusion from this study was that there exists a pressing need for more standardized ED and MD screening, prevention, and treatment programs among NCAA Division 1 schools. They further concluded, at the very least, that NCAA-member institutions should implement mandatory ED and MD education for all athletes and athletic personnel [5].

A study performed in 2012 involving menstrual irregularity in high school athletes showed a high incidence of menstrual irregularity and an increased number of musculoskeletal injuries than in athletes reporting normal menses. More than half of the athletes reported a change in menses during training or competition. The recommendation from this study was for improved education of high school athletes to improve caloric intake to better balance their energy availability to prevent or correct menstrual irregularity [26].

### 8.3.8 Management of Functional Hypothalamic Amenorrhea

Once an athlete is identified as having a menstrual disorder, management becomes the next issue. In functional hypothalamic amenorrhea,

there is insufficient energy availability. This then alters GnRH pulsatility in the hypothalamus and LH and FSH release. With limited pituitary secretion of LH and abnormal pulsatility, there is a lack of ovarian stimulation and thus an estrogen deficiency which impacts skeletal health from low sex hormones. There is also altered neuroendocrine function with low levels of insulin, glucose, leptin, triiodothyronine, and insulin-like growth factor-1 and elevated growth hormone and cortisol [18].

In adolescent girls, about 90 % of total body mineral content is accrued by 15½–18 years of age. Delayed puberty can compromise bone mass accumulation and low bone mineral density is a common finding in athletes with functional hypothalamic amenorrhea [18]. Twenty-five percent of bone mass accrual occurs in the 2 years surrounding menarche [13]. Due to this, the athlete becomes at risk for stress fractures, failure to achieve optimal peak bone mass density, and is thus at risk later in life for osteoporosis or delayed stress fractures [11]. Other risks from hypoestrogenism may include cardiovascular disease, dementia, depression, delayed post-exercise recovery, decreased immune function, and other neurodegenerative and psychiatric disorders [7, 13, 17, 19].

Physically active women with functional hypothalamic amenorrhea are able to prevent or reverse menstrual disorders by dietary modification in relation to their needed energy availability of approximately 30 kcal/kg lean body mass/day without any modification of their exercise regimen [4, 15, 17]. Therefore, the initial treatment of choice is improving the athlete's overall energy availability through improved nutrition [27, 28]. Increasing total daily energy intake in moderate increments may be the easiest approach, but the best method remains to be seen. Two small studies have shown that improving energy balance and energy availability in female athletes with amenorrhea resulted in the resumption of menstruation [27]. Treating the cause can lead to ovulatory cycles within 12 months, but up to 30 % may remain amenorrheic [11]. If modifying intake alone is not successful, the athlete may need to decrease her energy expenditure by modifying her training regimen [2]. A newer, interesting approach describes "eating by discipline." Since appetite is an unreliable indicator of energy requirements, the author advises eating specific amounts of particular foods at planned times rather than waiting for hunger and then eating until satisfied [4].

An initial bone mineral density (BMD) study is helpful in determining how long to attempt treatment with either diet alone or diet and exercise modification. If BMD testing is in the osteoporotic range, it is important to initiate hormone therapy immediately to stabilize bone density and prevent further loss [3]. If BMD results are near normal or in the osteopenic range, dietary modification is first line therapy.

If amenorrhea does not respond to dietary and exercise changes and is prolonged, greater than 3–6 months, hormone therapy should be considered in an attempt to protect BMD [7]. However, hormone therapy should not be used in adolescents younger than 16 years old [2, 29].

Hormone replacement therapy, both cyclic and continuous, has been described, but do not provide contraception [11, 12]. Since the majority of athletes with FHA may need to consider contraception, the use of oral contraceptive pills (OCPs) has been recommended. It is important to remember the use of OCPs will not normalize the metabolic factors impairing bone function, health and performance. They are unlikely to fully reverse the low BMD [2, 25, 30, 31]. Estrogen replacement without nutritional rehabilitation will not reverse bone loss [31].

Additionally, an OCP may have a masking effect in the amenorrheic athlete with the female athlete triad. This exogenous cycling can give the athlete a false sense of improvement without any enhancement of energy availability [13]. Transdermal estrogen may enhance bone mineral density more than oral, but may have a twofold risk of venous thromboembolic events related to its use. Another delivery option is etonogestrel/ethinyl estradiol vaginal (NuvaRing), which is preferred to OCPs by several patients, due to convenience and fewer systemic side effects. Some athletes are uncomfortable with the idea of being on a contraceptive. In those cases, it is better to discuss these medications as cyclic hormone regulation (CHR), rather than contraception.

Athletes who desire pregnancy can be treated with clomiphene citrate for ovulation induction. Results are varied with this approach [2, 30, 32]. Because of this, ovulation can be induced with pulsatile GnRH or injected gonadotropins [30]. Future treatment options may include daily subcutaneous injections of leptin analog [13], the use of subcutaneous IGF-1 combined with OCPs [15, 30], and opioid inhibitors such as naltrexone since endogenous opiates have been shown to inhibit GnRH [12, 30, 33]. Calcitonin nasal spray, Miacalcin, may be effective in preventing further decrease in BMD [3].

Bisphosphonates are not recommended in the age groups typically impacted by FHA due to prolonged activity in bones and potential teratogenic effects [2, 3, 13]. Also, selective estrogen receptor modulators (SERMs) have not been tested for safety in premenopausal women so are contraindicated [3]. It is very important to not forget basic therapeutic recommendations such as adequate calcium and vitamin D intake (adolescents: 1,300 mg calcium and 600 IU vitamin D per day; premenopausal: 1,000 mg calcium and 600 IU vitamin D daily), or supplemental, if needed [13]. With proper nutritional education and supervision, it is possible for the active female with functional hypothalamic amenorrhea, to resume normal menstrual cycles [15, 17, 30].

## 8.4 Contemporary Understanding of the Issues

Athletes are at higher risk than the general population for some type of menstrual dysfunction. The spectrum can range from long cycles, i.e., oligomenorrhea, to short cycles as seen in luteal phase defects, to absence of cycles, classified as either primary or secondary amenorrhea. Screening for primary amenorrhea is the most straightforward. If a female has had normal appearing sexual development, but no menses by age 15, the team physician, primary care provider or gynecologist should evaluate her. Another indicator of primary amenorrhea is no breast development by 13 years of age.

Screening for menstrual dysfunction should not occur in isolation and should include evaluation of the other two components of the female athlete triad, energy availability and skeletal health. Currently, there is no universally agreed upon timing or method to screen athletes for menstrual dysfunction or the other two components of the female athlete triad, energy availability and bone health.

When an athlete does screen positively for menstrual dysfunction, she should have a complete physician evaluation to include past medical history, past surgical history, current medications, social history, family history and a comprehensive obstetrical and gynecologic history. Comprehensive history taking will then guide the physician concerning appropriate physical examination, laboratory testing and radiology studies.

The most common type of secondary amenorrhea seen in athletes is functional hypothalamic amenorrhea. These athletes were having normal cycles at one time but now have not had cycles for 6 months (some sources state 3 months). The definition includes the distinction of absence of menstrual cycles with no other organic etiology found. It is thus a diagnosis of exclusion, requiring a full workup to exclude other causes, before recommendations for management can be formulated. The underlying defect is insufficient energy availability, which causes a deficiency in GnRH pulsatile secretion. This in turn leads to abnormal LH pulsatility and a decrease of sex hormones and other metabolic hormones. These situations lead not only to amenorrhea, but also to abnormalities in bone health. Women in this situation are at increased risk of stress fracture, inability to achieve optimum peak bone mass, future osteoporosis and delayed stress fractures.

When FHA is present, the first technique in management is increased energy intake. If this is not effective then recommendations should be made to decrease the amount of energy expended during exercise. After 3 to 6 months, when the combination of the two is not effective, management with hormone therapy may be considered. However, it is important to understand that causing an athlete to cycle regularly with exogenous

hormones is not addressing the underlying metabolic abnormality and will not do much to improve skeletal health. Additional treatment considerations include intake of adequate amounts of calcium and vitamin D. Education of the athlete and those who care for her is essential to be sure energy intake is increased during times of intense training and competition in order to maintain energy availability and attempt to prevent menstrual dysfunction.

## 8.5 Future Directions

Future research needs to be directed at refining prevalence numbers for menstrual disorders in active females. Current values are little more than estimates, given inconsistencies in studies to date. Additionally, sport-specific and ethnicity data need to be included to help focus on those athletes at highest risk.

Development of standardized screening procedures would be optimal for not only menstrual dysfunction, but for all three components of the female athlete triad. Rather than trying to incorporate screening questions into a pre-participation exam form, it appears best to develop a supplemental questionnaire with sections exploring each dimension of energy availability, menstrual status and bone health. This form could be used at the time of pre-participation exams or at other designated times during a competitive year or the athlete's career. It could also be used in the setting of health clubs and by personal trainers as part of the intake process for new clients.

Since alteration of GnRH pulsatility is the primary issue in functional hypothalamic amenorrhea, additional studies on factors influencing GnRH pulsatility may provide more strategies for effective prevention and treatment options to reverse menstrual dysfunction. Along with that is a need for applied research to confirm that athletes can and will prevent or reverse menstrual dysfunction by dietary reform without any moderation of the exercise regimen and to identify interventions acceptable to athletes and/or coaches.

Certain athletes will still not be able to increase their energy availability or decrease their energy expenditure to regain normal menstrual function. For those athletes, optimal medical treatment is essential. Studies concerning ideal hormone treatment regimens and use of other metabolic substances such as leptin analogs, IGF-1, and opioid antagonists are important to reverse menstrual dysfunction and improve bone health.

Improved education of the female athlete from early on in her sports involvement and those in her circle of influence (athletic trainers, coaches, family, etc.) is essential for primary prevention of female athlete triad disorders. One proposed model is for multidisciplinary treatment teams with a focus on proper nutrition related to each athlete's specific needs. Ultimately, health care providers working with athletes have a responsibility to become skilled at recognizing disorders associated with the female athlete triad in order to prevent them from occurring and to provide early intervention and treatment to minimize related illness and injury.

## 8.6 Concluding Remarks

Menstrual dysfunction in athletes should be considered a medical issue needing further evaluation. Amenorrhea in the active female should no longer be viewed as a good thing. In addition to being a medical problem, it can be a symptom related to abnormal skeletal health. At one extreme, it may be the first warning sign of a potentially lethal eating disorder. At the other extreme, it may be a sign of lack of proper nutritional education causing the athlete to exhibit disordered eating, i.e., not taking in enough calories for the level of training.

Prevention and early intervention are key components to minimizing morbidity and mortality. The ideal method and timing for screening have yet to be determined. The best method is likely using a separate questionnaire based screening tool during the pre-participation physical examination.

Screening for menstrual disorders can be a sensitive issue. Therefore, initial screening by athletic trainers or coaches should include basic questions concerning menses, bone health and energy availability. Once the athlete screens positively, she should be referred to medical personnel for a com-

**Table 8.2** Questions to ask the patient and/or the parents if an eating disorder is suspected

| |
|---|
| *Questions to ask the patient* |
| *Weight history* |
| Has there been any change in your weight? |
| What's the most you ever weight and when? The least and when? |
| Are you terrified about being overweight? |
| *History of dieting* |
| Have you ever tried to lose weight? |
| What kinds of diets have you tried? |
| Have you ever tried to lose weight or control your weight by vomiting, taking diet pills or laxatives, or excessive dieting? |
| Are you currently dieting or trying to lose weight? |
| *Emotions and eating* |
| Do you feel extremely guilty after eating? |
| Do you feel that food controls your life? |
| Have you gone on eating binges where you feel you may not be able to stop? |
| *Current eating/exercise habits* |
| Are you satisfied with the way you eat? |
| What did you eat yesterday? |
| Have you gone on eating binges where you feel you could not stop? |
| How much do you exercise in a typical week? |
| Have you ever fainted? |
| *Attitudes about weight and shape* |
| How do you feel about the shape and size of your body? |
| What do you think your ideal weight should be? |
| *Menstrual history* |
| Are your menstrual periods regular? |
| When was your last menstrual period? |
| *Questions to ask parents* |
| Does your child make negative remarks about his or her body? |
| Have you noticed any changes in his or her food-related habits? If so, what? |
| Are you concerned about your child's weight? Eating habits? Exercise habits? |

Source: Adapted from screening tools from the Office on Women's Health and the National Eating Disorders Screening Programs
Note: Confidential on-line screening tools are now available on-line at http://www.mentalhealthscreening.org/programs/workplace/nedsp.aspx)

prehensive evaluation. The sports medicine physician can begin by asking subtle questions related to energy availability and the link to menstrual disorders resulting from unhealthy eating patterns. Table 8.2 offers guidance for questioning. On-line screening tools are also available from the National Eating Disorder Screening Program free of charge.

## 8.7 Questions

1. Athletes who desire pregnancy but are not ovulating can be treated with:
   (a) Clomiphene citrate for ovulation induction
   (b) Pulsatile GnRH or injected gonadotropins
   (c) Endogenous opiates
   (*d) Both a and b can be used to induce ovulation
2. Which of the following statements is *false* about oral contraceptive pills (OCP)?
   (a) The use of OCPs will not normalize the metabolic factors impairing bone function, health and performance
   (b) OCP are unlikely to fully reverse the low BMD
   (*c) Estrogen replacement without nutritional rehabilitation will reverse bone loss
   (d) All statements are true
3. Hormone therapy should not be used in adolescents younger than _____years old
   (a) 15
   (*b) 16
   (c) 17
   (d) 18
4. Which of the following statements are true with regard to appetite in the female athlete?
   (*a) Appetite is an unreliable indicator of energy requirements
   (b) Athletes should just eat when they are hungry and this will prevent low energy availability
   (c) Athletes should wait for hunger and then eat until satisfied in order to increase energy availability
   (d) All of the statements are true
5. Treating the cause of menstrual dysfunction can lead to ovulatory cycles within 12 months, but up to _____of athletes may remain amenorrheic.
   (a) 50 %
   (b) 70 %
   (c) 40 %
   (*d) 30 %

6. _____ percent of bone mass accrual occurs in the _____ years surrounding menarche.
   (a) 45.4
   (b) 35.3
   (c) 30.1
   (*d) 25.2
7. In adolescent girls, about _____ of total body mineral content is accrued by 15½–18 years of age.
   (a) 60 %
   (b) 70 %
   (c) 80 %
   (*d) 90 %
8. Target groups for menstrual screening should include which group(s) of women?
   (a) A normal secondary sexual development but no menarche by 15 years of age
   (b) Failure of the thelarche (breast development) by 13 years old
   (c) No menarche within 5 years after breast development that occurred less than 10 years old
   (*d) All of the above mentioned groups should be targeted
9. In 2006, National Collegiate Athletic Association Division 1 Schools adopted a standardized eating disorder and menstrual dysfunction screening tool to be used for all female athletes.
   (a) True
   (*b) False
10. Which of the following characteristics is (are) true for an athlete with polycystic ovarian syndrome (PCOS)?
    (a) She will usually be at or above a normal Body Mass Index (BMI)
    (b) She will likely be hirsute
    (c) She may show signs of insulin resistance
    (*d) All of the above characteristics could be possible with PCOS

## Appendix 1: Women's Health History Questionnaire

**Name**: _____

Age: _____

Birth date: _____

Height: _____ Weight: _____

How many years have you been at your present weight? _____

Have you lost _____ or gained _____weight recently?

If you have lost or gained weight recently, how many pounds? _____

Ethnic background: (Check one)

| | | | |
|---|---|---|---|
| Caucasian, non-Hispanic | _____ | Hispanic | _____ |
| American Indian | _____ | Black, non- Hispanic | _____ |
| Asian | _____ | Other/specify | _____ |

If you are an intercollegiate athlete, please check the sport/activity, in which you currently participate, if you are not presently an intercollegiate athlete, please skip to the next question (Check one):

| | | | | | |
|---|---|---|---|---|---|
| Basketball | _____ | Golf | _____ | Soccer | _____ |
| Tennis | _____ | Track | _____ | Volleyball | _____ |
| Softball | _____ | Other, please specify | _____ | | |

If you play intramural sports, please check the sport/activity in which you currently participate, if you do not presently participate in intramural sports, please skip to the next question (Check one):

Basketball _____ Golf _____ Soccer _____
Tennis _____ Track _____ Volleyball_____
Softball _____ Other, please specify _____

If you belong to a club sport/activity, please check the sport/activity in which you currently participate, if you do not presently participate in club sports/activities, please skip to the next question (Check one):

Dance _____ Theater _____ Soccer _____
Volleyball _____ Running _____ Golf _____
Water-skiing _____ Cycling _____ Other, please specify_____

Please check the type and amount of exercise you usually engage in each week. Be as specific as possible, for example, weight training 1.5 h per week; jogging 4 mph for 1 h two times a week or aerobics 30 min two time a week, or walking 3 mph 4 h each week.

| Activity | #Times per week | Hours per week | Miles per week (if applicable) |
|---|---|---|---|
| Aerobics | | | |
| Basketball | | | |
| Calisthenics | | | |
| Cycling | | | |
| Dance | | | |
| Ballet | | | |
| Jazz | | | |
| Tap dance | | | |
| Modern | | | |
| Golf | | | |
| Handball | | | |
| Jogging | | | |
| Racquetball | | | |
| Running | | | |
| Soccer | | | |
| Softball | | | |
| Swimming | | | |
| Water-skiing | | | |
| Track (specify type of event) | | | |
| Volleyball | | | |
| Weight-lifting | | | |
| Other (list) | | | |

How many years (or months) have you been involved in the above activity schedule?
Years _____ months _____
List any injuries you have had or presently have that are related to your participation in these activities.

_____
_____
_____

How is your health? Good _____ Fair _____ Poor_____

Please explain: _____

Has a health care professional ever told you that you have problems with your thyroid glands?
Yes _____ No _____

Has a health care professional every told you that you had problems with your ovaries?
Yes _____ No _____

Has a health care professional ever told you that you had problems with your uterus?
Yes _____ No _____

Has a health care professional ever told you that you had problems with your endocrine system?
Yes _____ No _____

Are you now using or have you ever used birth control pills or other hormones (these are used to prevent pregnancy or regulate menstrual periods)?
Yes _____ No _____

If you have used birth control pills or other hormones, for what reason did you use these?
Regulate menstrual periods _____
Prevent pregnancy _____
Other reason. Please explain _____
_____

If you have used birth control pills or other hormones, how long has it been since you used them?
_____

Within the past year, have you taken steroids for any reason (these are occasionally used to reduce inflammation/swelling, etc. during illness or injury)?
Yes _____ No _____

If you have taken steroids, how long has it been since you took them?
_____

Are you pregnant? Yes _____ No _____
Is there a possibility you could be pregnant? Yes _____ No _____
Have you recently had a pregnancy test? Yes _____ No _____
If you answered yes, when was the test? Date _____
Do you currently smoke? Yes _____ No _____
Would you classify your menstrual cycle as regular? _____ Or irregular?_____
Please explain why? _____
_____
Approximately how many menstrual periods do you have in a year?_____
Do you experience menstrual bleeding every month? _____
Do you have a menstrual period every month that is about the same number of days apart from month to month?
Yes _____ No _____
What was the first day of your last menstrual period? (If you are currently on your menstrual period, please list the first day of your current period). _____
How many days long was your last menstrual period? _____
How many days ago was your last menstrual period? _____
What is the average duration (length in number of days) from the beginning of one menstrual cycle to the beginning of your next menstrual cycle? _____
How old were you when you first began menses? _____

Have you ever had or presently have anorexia nervosa or bulimia that you are aware of? _____

_____

How did you become aware of this? _____

## Appendix 2: Menstrual History Log Sheet

Name _____

Directions: Please mark the first day of your menstrual cycle with an X. Continue to mark any days that you experience bleeding

| Month | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Jan | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Feb | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Mar | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| April | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| May | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| June | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| July | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Aug | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Sep | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Oct | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Nov | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Dec | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |

## Appendix 3: Student-Athlete Nutritional Health Questionnaire

### Stress Fracture History

1. Have you ever had a stress fracture or a stress reaction? Yes No
2. Have you ever had x-rays to rule out a stress fracture or a stress reaction? Yes No
3. Have you ever had a bone scan or a bone density test? Yes No
4. Do you talk calcium? Yes No
5. Are you a vegetarian?

### Eating/Weight History

1. What is your highest and lowest weight in the past year? Highest weight _____ lbs.
   Lowest weight _____ lbs.
2. What is had any recent changes in weight? Yes No
3. What is your desired weight? Yes No
4. Do you weigh yourself often? Yes No
5. Do you consciously watch your weight? Yes No
6. Would your weight be different if you were not exercising vigorously? Yes No

7. How many times a year do you lose weight intentionally?
8. When your season is over and you stop or reduce training, do you gain or lose weight?
   If "gain," how much weight? _____ lbs.
   If "lose," how much weight? _____ lbs.
   What is your weight in season at the peak of training? _____ lbs.
9. Do you have to restrict your food intake more or less than in the past to be at your competitive weight? (*please circle the appropriate answer*)
   Much less than before  Somewhat less than before  No change  Somewhat more than before  Much more than before
10. Are you preoccupied with weight? Yes No
11. Does worrying about weight take up a significant amount of your time? Yes No

### Menstrual History (Females Only)

1. At what age did you have your first period? Month _____ Year _____
2. When was your last period? Month _____ Year _____
3. How many periods have you had in the last 12 months. _____
4. Are you on any form of estrogen/birth control? Yes No
   If yes, what form? _____
   How long?_____
   Why? (Control of period, medical prescription, other) _____
   If it has been recommended and you are not taking it, why? _____
5. Have you ever been diagnosed with anemia? Yes No
6. Do you eat red meat? Yes No
7. Do you have heavy menses? Yes No
8. Were you aware of any effect of regular training for sport on the occurrence of your first menstrual period? Yes No
   If yes, briefly explain _____
9. Are menstrual problems such as cramps and irregularity common in your family? Yes No

## Appendix 4: Female Athlete Screening Tool

Please answer as completely as possible.
The key is used to quantify and define activity level for further clarification of the questions.

| Key[a]: | Exercise = Physical activity ≥20 min |
|---|---|
| | Practice = Scheduled time allotted by coach to work as a team or individually in order to improve performance |
| | Training = Intense physical activity. The goal is to improve fitness level in order to perform optimally |

1. I participate in additional physical activity ≥20 min in length on days that I have practice or competition.
   (1) Frequently (2) Sometimes (3) Rarely (4) Never

2. If I cannot exercise, I find myself worrying that I will gain weight.
   (1) Frequently (2) Sometimes (3) Rarely (4) Never

3. I believe that most female athletes have some form of disordered eating habits.
   (1) Strongly Agree (2) Agree (3) Disagree (4) Strongly Disagree

4. During training, I control my fat and calorie intake carefully.
   (1) Frequently (2) Sometimes (3) Rarely (4) Never

5. I do not eat foods that have more than 3 g of fat.
   (1) Strongly Agree (2) Agree (3) Disagree (4) Strongly Disagree

6. My performance would improve if I lost weight.
   (1) Strongly Agree (2) Agree (3) Disagree (4) Strongly Disagree

7. If I got on the scale tomorrow and gained 2 pounds, I would practice or exercise harder or longer than usual.
   (1) Frequently (2) Sometimes (3) Rarely (4) Never

8. I weigh myself_____.
   (1) Daily (2) 2 or more times a week (3) Weekly (4) Monthly or less

9. If I chose to exercise on the day of competition (game/meet), I exercise for
   (1) 2 or more hours (2) 45 min to 1 h (3) 30–45 min (4) Less than 30 min

10. If I know that I will be consuming alcoholic beverages, I will skip meals on that day or the following day.
    (1) Frequently (2) Sometimes (3) Rarely (4) Never

11. I feel guilty if I choose fried foods for a meal.
    (1) Frequently (2) Sometimes (3) Rarely (4) Never

12. If I were to be injured, I would still exercise even if I was instructed not to do so by my athletic trainer or physician.
    (1) Strongly Agree (2) Agree (3) Disagree (4) Strongly Disagree

13. I take dietary or herbal supplements in order to increase my metabolism and/or to assist in burning fat.
    (1) Frequently (2) Sometimes (3) Rarely (4) Never

14. I am concerned about my percent body fat.
    (1) Frequently (2) Sometimes (3) Rarely (4) Never

15. Being an athlete, I am very conscious about consuming adequate calories and nutrients on a daily basis.
    (1) Frequently (2) Sometimes (3) Rarely (4) Never

16. I am worried that if I were to gain weight, my performance would decrease.
    (1) Strongly Agree (2) Agree (3) Disagree (4) Strongly Disagree

17. I think that being thin is associated with winning.
    (1) Strongly Agree (2) Agree (3) Disagree (4) Strongly Disagree

18. I train intensely for my sport so I will not gain weight.
    (1) Frequently (2) Sometimes (3) Rarely (4) Never

19. During season, I choose to exercise on my one day off from practice or competition.
    (1) Frequently (2) Sometimes (3) Rarely (4) Never

20. My friends tell me that I am thin but I feel fat.
    (1) Frequently (2) Sometimes (3) Rarely (4) Never

21. I feel uncomfortable eating around others.
    (1) Frequently (2) Sometimes (3) Rarely (4) Never

22. I limit the amount of carbohydrates that I eat.
    (1) Frequently (2) Sometimes (3) Rarely (4) Never

23. I try to lose weight to please others.
    (1) Frequently (2) Sometimes (3) Rarely (4) Never

24. If I were unable to compete in my sport, I would not feel good about myself.
    (1) Strongly Agree (2) Agree (3) Disagree (4) Strongly Disagree

25. If I were injured and unable to exercise, I would restrict my calorie intake.
    (1) Strongly Agree (2) Agree (3) Disagree (4) Strongly Disagree

26. In the past 2 years I have been unable to compete due to an injury
    (1) 7 or more times (2) 4–6 times (3) 1–3 times (4) No significant injuries

27. During practice I have trouble concentrating due to feelings of guilt about what I have eaten that day.
    (1) Frequently (2) Sometimes (3) Rarely (4) Never

28. I feel that I have a lot of good qualities.
    (1) Strongly Agree (2) Agree (3) Disagree (4) Strongly Disagree

29. At times I feel that I am no good at all.
    (1) Strongly Agree (2) Agree (3) Disagree (4) Strongly Disagree

30. I strive for perfection in all aspects of my life.
    (1) Strongly Agree (2) Agree (3) Disagree (4) Strongly Disagree

31. I avoid eating meat in order to stay thin.
    (1) Strongly Agree (2) Agree (3) Disagree (4) Strongly Disagree

32. I am happy with my present weight.
    (1) Yes (2) No

33. I have done things to keep my weight down that I believe are unhealthy.

    (1) Frequently (2) Sometimes (3) Rarely (4) Never

Source: From McNultyKY, Adams CH, AndersonJM, AffenitoSG. Development and validation of a screening tool to identify eating disorders in female athletes. *J Am Diet Assoc*2001;101(8):886-892. With permission.

**Scoring Instructions**: The FAST is scored on a 4 point Likert Scale. The higher the number the higher the probability (i.e., 4 points = frequently; 3 points = sometimes; 2 points = rarely; and 1 point = never).

Questions 15, 28, and 32 are reversed scored. For questions 32, a response of yes receives 1point and a response of no receives 2 points. In a small group of female athletes (N = 41), sub-clinical scores ranged from 77 to 94 and clinical scores were >94. **Note this is a screening tool**, **not a diagnostic tool**.

## References


1. Klein JD, Barratt MS, Blythe M, et al. Menstruation in girls and adolescents: using the menstrual cycle as a vital sign. Pediatrics. 2006;118(5):2245–50.
2. Nattiv A, Loucks AB, Manore MM, et al. The female athlete triad. Med Sci Sports Exerc. 2007;39(10): 1867–82.
3. Sangenis P, Drinkwater BL, Loucks A et al. International Olympic Committee medical commission working group on women in sport. Position stand on the female athlete triad. Revised August 2006. Available at: http://www.olympic.org/Documents/Reports/EN/en_report_917.pdf Accessed June 12, 2013.
4. Loucks AB, Kiens B, Wright HW. Energy availability in athletes. J Sports Sci. 2011;29(S1):S7–S15.
5. Beals KA. Eating disorder and menstrual dysfunction screening, education, and treatment programs. Phys Sportsmed. 2003;31(7):33–8.
6. Torstveit MK, Sundgot-Borgen J. Participation in leanness sports but not training volume is associated with menstrual dysfunction: a national survey of 1276 elite athletes and controls. Br J Sports Med. 2005;39:141–7.
7. Lebrun CM. The female athlete triad: what's a doctor to do? Curr Sports Med Rep. 2007;6:397–404.
8. Roupas ND, Georgopolous NA. Menstrual function in sports. Hormones. 2011;10(2):104–16.
9. Practice Committee of the American Society for Reproductive Medicine. Current evaluation of amenorrhea. FertilSteril. 2008;90 Suppl 3:S219–25.
10. Chen EC, Brzyski RG. Exercise and reproductive dysfunction. FertilSteril. 1999;71(1):1–6.
11. Bieber EJ, Sanfilippo JS, Horowitz IR. Clinical gynecology. Philadelphia, PA: Churchill Livingstone Elsevier; 2006.
12. Speroff L, Fritz MA. Clinical gynecologic endocrinology and infertility. 7th ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2005.
13. Nazem TG, Ackerman KE. The female athlete triad. Sports Health. 2012;4(4):302–11.
14. Rumball JS, Le Brun CM. Preparticipation physical examination. Selected issues for the female athlete. Clin J Sport Med. 2004;14(3):153–60.
15. Loucks AB. Energy availability, not body fatness, regulates reproductive function in women. Exerc Sport Sci Rev. 2003;31(3):144–8.
16. Loucks AB. Introduction to menstrual disturbances in athletes. Med Sci Sports Exerc. 2003;35(9):1551–2.
17. Redman LM, Loucks AB. Menstrual disorders in athletes. Sports Med. 2005;35(9):747–55.
18. Stafford DE. Altered hypothalamic-pituitary-ovarian axis function in young female athletes. Treat Endocrinol. 2005;4(3):147–54.
19. Pauli SA, Berga SL. Athletic amenorrhea: energy deficit or psychogenic challenge? Ann NY Acad Sci. 2010;1205:33–8.
20. Bernhardt DT, Roberts WO (eds). PPE preparticipation physical evaluation. 4th edn., American Academy of Pediatrics; 2010:20–130, 154–156.
21. Ljungqvist A, Jenoure P, Engebretsen L, et al. The International Olympic Committee (IOC) consensus statement on periodic health evaluation of elite athletes. J Ath Tr. 2009;44(5): 538–57.
22. McNulty KY, Adams CH, Anderson JM, Affenito SG. Development and validation of a screening tool to identify eating disorders in female athletes. J Am Diet Assoc. 2001;101(8):886–92.
23. De La Torre DM, Snell BJ. Use of the Preparticipation Physical Exam in Screening for the Female Athlete Triad Among High School Athletes. J School Nurs. 2005;21(6):340–345.
24. Beals KA, Hill AK. The prevalence of disordered eating, menstrual dysfunction, and low bone mineral density among US collegiate athletes. Int J Sport Nutr Exerc Metab. 2006;16(1):1–23.
25. Bonci CM, Bonci LJ, Granger LR, et al. National athletic trainers' association position statement: preventing, detecting, and managing disordered eating in athletes. J Athl Train. 2008;43(1): 80–108.
26. Thein-Nissenbaum JM, Rauh MJ, Carr KE, et al. Menstrual irregularity and musculoskeletal injury in female high school athletes. J Athl Train. 2012; 47(1): 74–82.
27. Manore MM, Kam LC, Loucks AB. The female athlete triad: components, nutrition issues, and health consequences. J Sports Sci. 2007;25(S1): S61–71.
28. Loucks AB. Menstrual disorders in athletes. Curr Sports Med Reports. 2006;5:273–5.
29. Warren MP, Perlroth NE. The effects of intense exercise on the female reproductive system. J Endocrinol. 2001;170:3–11.
30. Gordon CM. Functional hypothalamic amenorrhea. N Engl J Med. 2010;363(4):365–71.
31. Klein DA, Poth MA. Amenorrhea: an approach to diagnosis and management. Am Fam Physician. 2013;87(11):781–8.
32. Shangold M, Rebar RW, Wentz AC, Schiff I. Evaluation and management of menstrual dysfunction in athletes. JAMA. 1990;263(12):1665–9.
33. Gibbs RS, Karlan BY, Haney AF, Nygaard IE. Danforth's obstetrics and gynecology. 10th ed. Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins; 2003.

# Eating Disorders and Disordered Eating: A Global Perspective

9

Ascensión Blanco Fernández,
MariaFernandez-del-Valle, Marta Montil Jiménez,
and Maesy Indigo

**Abstract**

The continuous progression of the prevalence of eating disorders (ED) is a cause of concern during the last decades, and epidemiologic research has shown different increases between countries. Food is as well a psychological need that presents interindividual differences such as preferences/rejections to foods, healthy/unhealthy dietary habits, and worries about the shape, which result in "body dissatisfaction." Thus, dieting and compensatory behaviors to control weight are acquired as normal behaviors by the general population increasing the risk to suffer from ED. In addition, healthy people and other atypical disorders are strongly influenced by emotions in their eating behaviors, not only EDs.

**Keywords**

Eating disorders • Disordered eating • Atypical disorders • Emotions • Eating behaviors • Comorbidity

A.B. Fernández, Ph.D. (✉)
Especialidades Médicas, Psicología y Pedagogía,
Universidad Europea de Madrid (UEM), C/ Tajo S/N,
Villaviciosa de Odón, Madrid 28670, Spain
e-mail: ascension.blanco@uem.es

M. Fernandez-del-Valle, Ph.D.
Department of Health, Exercise, and Sport Sciences,
Texas Tech University, Exercise and Sport Sciences
Building, 3204 Main, Lubbock, TX 49423, USA
e-mail: maria.fernandez-del-valle@ttu.edu

M.Montil Jiménez, Ph.D.
Department of Kinesiology and Athletic Training,
Universidad Europea de Madrid, Calle Tajo s/n,
Villaviciosa de Odón, Madrid 28670, Spain
e-mail: marta.montil@uem.es

M. Indigo, M.S.
Department of Health, Exercise & Sport Sciences,
Texas Tech University, HESS, Box 43011, Lubbock,
TX 79409-3011, USA
e-mail: minigo@umd.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_9, 

## 9.1 Learning Objectives

After completing this chapter, you should be able to:

- Describe the main characteristics of eating disorders (ED).
- Describe the differences between ED and disordered eating (DE).
- Identify the risks and protective factors.
- Discuss the importance of the psychological perspective of ED and nutrition.

## 9.2 Introduction

Eating disorders (ED) are mental disorders that are considered by the World Health Organization (WHO) as severe diseases affecting Health Systems worldwide. Although EDs are mainly developed by females during adolescence, cases have been diagnosed and described in infants, prepubescent children, adults, and elders as well. The continued progression of the prevalence of ED is a cause of concern during the last decades. In this way, data show prevalence percentages from 6.4 to 4 % in young females and 0.3 to 1 % in males (12–21 years), which indicates a gender ratio 1:9 [1]. In eastern countries such as Spain, research of prevalence rates reported a 20.8 % risk of developing ED in college females and 14.9 % in males [2], while in adolescents data show 13 % for females and 1 % in males [3].

In the USA, the prevalence of anorexia is between 0.5 and 3.7 %, whereas bulimia nervosa is 1.1–4.2 % [4]. Generally when the estimations are based upon large populations, the rate of anorexia for developed countries is 8.1 per 100,000 habitants per year, whereas bulimia nervosa shows prevalence rates of 1 % in adolescents and young adults [5].

To date, no protocol treatment has been considered as the most successful. For example, there is a lack of consensus among professionals about which is the best way to recover weight in anorexic patients [6, 7].

Disordered eating (DE) or eating disorders not otherwise specified (EDNOS) also exist, and these describe a wide range of irregular patterns of eating behaviors that do not warrant a diagnosis of a specific eating disorder. DE is not the result of a specific ED. Often, these are caused, for example, by bad nutrition, bad health habits, mental disorders (e.g., clinical depression), or by unrelated mental disorders (e.g., homesickness, mourning, breakthrough). However, recognizing DE is very important because these may involve seriously disturbing perceptions of body image and food habit problems or may include only a few signs or symptoms from classic types of ED.

Eating disorders (ED) are considered much more serious than DE, and they are defined as psychiatric disorders, as it is shown in the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) diagnostic criteria. ED criteria are based on psychological, behavioral, and physiological characteristics [6]. Nevertheless, epidemiologic research reported that there is also a perturbing increase of the prevalence of DE [8], defined as eating disorders nonspecified or incomplete [4, 9].

There is a dearth of research in disordered eating among children and preadolescent girls, for the research has been focused on subsequent maturation phases such as pubertal and postpubertal years, adulthood, as well as in middle-aged and aging women [9–12].

Physical active women of all ages can develop EDs or DE because risk factors are present throughout the life span. For instance, a common risk factor is fasting or calorie restriction to participate in activities that promote thinness such as endurance sports (e.g., long distance, triathlons), sports with weight categories (e.g., martial arts, wrestling, boxing), and slim appearance sports/dances (e.g., ballet, gymnastics) [10–15]. Some affect mostly children to young adults, whereas other affect middle-aged to older women [16].

## 9.3 Research Findings

### 9.3.1 States of Food and Food Psychology and Associated Pathologies

Nutritional requirements change throughout life-span, and research has shown that poor nourishment is a limitant for adequate development

and growth and is associated with increased risk for future chronic pathologies [17].

The "nutritional stages" refer to the different situations that occur prior to, during, and after the feeding process. Interestingly, the stages are not the same in all individuals because these are dependent on eating behavior (determined by one's stage of life, choice of food, nourishment learning, motivation, and attitude towards the food). Eating behaviors are individual because it functions to satisfy specific physical needs; to feel better, as a socialization agent; to satisfy craving from attractive characteristics of food; etc. It seems valid to use these reasons to begin or end the eating process. Therefore, it is necessary to understand the transcendence of the physical, psychological, and social factors affecting this process during life.

#### 9.3.1.1 The Food's Psychology

Food is as well a psychological need that presents interindividual differences such as preferences/rejections to foods, healthy/unhealthy dietary habits, and worries about the shape, which result in "body dissatisfaction." Thus, dieting and compensatory behaviors to control weight are acquired as normal behaviors by the general population increasing the risk to suffer from ED. Different psychological approaches have determined that diet selection is based on the individual factors (learning process, beliefs, and the psychological features of the individual) and not merely on the nutritional content of the food [18, 19].

At least three different models try to explain the food selection:

- Evolutionary models for food selection (learning)
- Cognitive models for food selection (beliefs)
- Psychophysiological models for food selection (psychophysiology of the individual)

*The evolutionary models*. Different research groups have reported various factors that can be affecting food preference. Thus, the food/drink selection:

– Is innate, and therefore specific flavors are preferred such as sweet [20] and salty [21] and rejected such as bitter flavors [22].
– In individuals who were habituated to sweetened water when they were babies are going to drink greater amounts compared to those who were not [23].
– Depends on the environment (observational and social learning). For example, parents' attitudes towards food selection [24].
– Depends on the associative learning such as reward for food selection (i.e., "if you eat veggies I will feel really happy" [25]).
– Depends on the media and advertisement influence [26].

*The cognitive models*. This concept encloses different models that try to explain and predict the food selection such as risk perception, costs or benefits of a particular behavior, self-efficacy, past behavior, and social norms [27]. This provides evidence for the maintenance of positive and negative attitudes with respect to the food, which reduces the probability to eat a specific food.

*The psychophysiological models*. These models are focused in the regulation of hunger and satiation. On the one hand, "hunger" is the discomfort associated with the absence of food and is a motivation to eat [27]. On the other hand, "satiation" is an opposed concept where the consumption of food reduces the motivation to eat. There are parts of the brain such as the hypothalamus, with underlying mechanisms, that regulate the hunger–satiation state. Researchers have reported the importance of two central systems in this regulation: excitatory (serotonin) and inhibitory (catecholamine) [17, 28].

#### 9.3.1.2 Feeding and Emotions ("Emotional Feeding")

Eating behaviors are related to emotions, and some eating disorders reflect those feelings. For example, anorexic patients exhibit the "fear to eat" [29]. However, healthy people are strongly influenced by emotions in their eating behaviors as well. For example, healthy individuals reduce their food intake when they experience negative emotions [28–30].

The attachment bond is regulated by the feeding process during infanthood. This basic

nourishment–feeder relationship is maintained throughout the life span—binding food to affection and hunger to dissatisfaction. This association will interfere the differentiation of nourishment and emotional needs in both clinical and general population [31, 32]. Hence, educating children in identifying and expressing emotions can prevent ED and promote healthy nutrition [32].

Silva [30] has shown that ED patients try to remove their emotions and affections showing lack of emotions, assertiveness, submissiveness, shyness, and self-futility [33]. The inability to identify and describe emotions (alexithymia) characterizes ED patients [34–40] and contributes to ED development [39, 41, 42]. Indeed, one study [40] reported that alexithymia in patients that suffered from anorexia nervosa (AN) and bulimia nervosa (BN) directly influenced their food intake. Therefore, AN patients refused to eat in dysphonic situations (sadness, anxiousness, or irritability), while BN patients increased food intake.

In spite of the recognized influence of the emotions in eating behavior, there are few studies that include the role of the emotional variables in an individual's development. Research has shown association between alexithymia and the degree of anxiety, food acceptance, weight gain, body image, and self-esteem [43–47]. ED has been described as a way to regulate self-negative emotions [37, 48–54], to further food restriction function as a way to reduce negative emotions (using the food as a negative reinforcement) [48, 51, 54].

#### 9.3.1.3 Food: Culture and Socialization

Food preferences are culturally learned and socially regulated [55, 56]. Therefore, eating behavior is influenced by different factors such as race, religion, age, gender, health, social class, and ultimately by the environment.

A deeper knowledge and study of socialization processes in eating behavior can provide meaningful information to understand the uncertain causes of ED [57]. This can help develop strategies to detect factors affecting the acquisition of healthy behavior related to food intake [29] and, therefore, understand the differences between gender, races, etc. susceptible to suffer from one or other type of ED.

#### 9.3.1.4 The Food Craving: Feeding, Overactivation, and Stress

*The food craving* is the intense desire to consume food, and it is stronger than a normal hunger. Scientists have proposed four theories to explain this behavior:

1. Homeostatic theories: that the intense desire to eat appears as a consequence of a previous negative reinforcement (eating behavior associated to negative feelings). The individual eats to overcome those feelings not because they are hungry [58].
2. Theories of incentive: that the delectable properties of the food (smell, taste, texture) have a motivational effect on intake behavior [51]. These theories consider that the craving influences the eating behavior by pursuing the body well-being [58].
3. Cognitive theories: that food craving can be triggered in two situations, (a) when environmental conditions impede the food intake producing frustration and leading the individual to search for food and (b) when the individual is restricting the food intake and feels successful accomplishing it [59].
4. Psychological theories: that the most recent theories postulate that food, like drugs, is addictive and has the ability to produce durable changes in structures of the nervous system related to the appetite (i.e., the insula, the caudate, the amygdala, the nucleus accumbens, the anterior cingulate, and orbitofrontal cortex) [60]. These changes in the brain are responsible for the maintenance of food craving [61].

*Food, Overactivation, and Stress*: There is growing research on the relationship between eating patterns and stress. In spite of the common notion that stress causes a reduction in food intake, several studies suggest that eating behavior is related to the psycho-emotional characteristics of the individual and the type of stress. This has been crucial to explain the implications of the food restriction, overeating, and stress in the development of ED (i.e., anorexia nervosa and bulimia nervosa) and obesity. For instance, studies showed that emotion (i.e., positive or negative valence and low or high intensity) has a stronger influence in overeaters and restrained

**Table 9.1** Evaluation criteria of AN and BN

| Criteria | Indicators |
|---|---|
| Body weight | Current and historical |
| Eating behavior | Dieting, fasting, uncontrolled eating (binging: craving), purging, food ban (forbidden eating), anxiety at intake and after intake, distorted body image, body dissatisfaction, emotional issues influenced by intake, associated diseases (comorbidity: depression, anxiety, obsessive–compulsive disorders, sexual abuse, compulsive overeating) |
| Biomedical aspects | AN and BN: underweight. BMI < 17.5 in AN and normal weight BN, anthropometry, analytical |

eaters. This line of research has shown that the level of affective arousal is the key to trigger an episode of overeating in restrained eaters, rather than the valence of the emotion [62]. Nevertheless, anxiety and alexithymia are features present in ED patients even after recovery [63, 64], so that has been postulated as a risk factor to suffer ED [65–69].

### 9.3.1.5 Eating Disorders: Personality Characteristics and Profiles

#### Symptoms in AN, BN, and EDNOS Patients

- The principal characteristic of AN (Table 9.1) is the rejection to maintain body weight in the normal minimum values (IMC > 17.5 according to the WHO). The characteristics are drastic diets and obsessive physical exercise (restrictive AN) or in the form of binge eating and purging (compulsive/purgative AN).
- BN is characterized by frequent binge eating and compensatory behaviors, in order to control or lose weight, such as self-induced vomiting or the continuous use of laxatives or diuretics (purgative BN), or fasting and obsessive physical exercise (non-purgative BN) (see Table 9.1).
- EDNOS (eating disorder not otherwise specified), such as compulsive overeating or certain types of morbid obesity (Table 9.2).

#### ED and Comorbidity

*Personality disorders* (Table 9.3) [70–74]:

Personality is considered a habitual pattern of behavior that is manifested in different contexts and situations (i.e., cognition, impulse

**Table 9.2** EDNOS characteristics

| EDNOS | Characteristics |
|---|---|
| Binge eating disorders | A. Recurrent episodes of binge eating<br>1. Intake in a short period of time of uncontrolled amounts of food<br>2. Feeling of loss of control overeating during the episode (cannot stop eating)<br>B. Episodes of binging associated with three or more of the following:<br>1. Intake much faster than normal<br>2. Eating until feeling uncomfortably full<br>3. Ingestion of large amounts of food despite not being hungry<br>4. Eating alone to conceal their voracious appetite<br>5. Feeling disgusted with oneself, depressed, or great guilt after binge eating<br>C. Deep distress after remembering binging<br>D. Binging takes place at least two days a week for 6 months<br>E. The binging is not associated with inadequate compensatory strategies (e.g., purging, fasting, excessive exercise) |
| Do not meet criteria for any ED | 1. AN criteria but the individual still has regular menstruation<br>2. AN criteria except significant weight loss<br>3. Individual with normal weight performs inappropriate compensatory behavior after eating small amounts of food<br>4. Chewing and expel, but not swallowing large amounts of food<br>5. Compulsive disorder: recurrent binge eating in the absence of inappropriate compensatory behavior typical of BN |

**Table 9.3** Alteration: eating behavior and personality

| Table or personality profile | Indicators |
|---|---|
| AN | Obsessive |
| | Introverted |
| | Socially insecure |
| | Dependent |
| BN | Multi-impulsivity |
| | Low capacity control |
| AN and BN with personality disorders | >Frequency of binging |
| | Anxiety–depression |
| | ≥Difficulty with social integration |
| | ≥Frequently. Attempted suicide |

control, emotional stability, and interpersonal relationships), which is stable and continuous.

Personality disorders are atypical behavioral patterns that generate serious limitations in everyday life and increase distress. Unlike mental disorders, personality disorders are temporarily stable [75–77].

Despite the high rate of comorbidity among EDs and personality disorders (ranges between 51 and 84 %), the interest for this field is fairly recent [74, 78–80].

The first baseline research was conducted by Gartner [70] and consisted of 35 hospitalized patients diagnosed with AN, BN, or both AN and BN. The main results were that 57 % of the sample had at least one, AN or BN ED, plus personality disorders. Personality disorders were relatively common. The most frequent were borderline, avoidant, and self-destructive which are distributed equally amongst the various types of eating disorders. In a study done by Wonderlich [71], with a total of 46 patients, 72 % of the sample met diagnostic criteria for at least one personality disorder. For example, the "obsessive personality" was the most common feature of restrictive anorexia nervosa and "borderline and histrionic personality" for bulimia. Another study done by Grilo [80], with 136 hospitalized patients diagnosed with various EDs, 84 % had one or more personality disorders such as "borderline, avoidant, and dependent." According to the study done by Matsunaga [74], of the 108 patients with various EDs, 51 % of the sample had personality disorders where the "borderline and antisocial" were prevalent in BN and no clear predominance in the case of AN. Furthermore, according to the work of Striegel-Moore et al. [81], with a sample of 161 veterans (98 men and 63 women) with EDs, personality disorder comorbidity was significantly greater in women (49 %) compared to men (18 %).

### Affective Disorders: Depression and Anxiety

*Depression and EDs*. Different studies have provided compelling evidence for the relationship between affective disorders, especially between "EDs and depression." From a clinical standpoint, the distinction may be difficult, since they share signs and symptoms, family trends, natural history, neuroendocrine alterations, and the response to drug treatment [82]. The prevalence of affective disorders in AN indicates that the presence of depression ranges from 20 to 100 % of the cases according to various studies [83]. Luka et al. noted that 73.3 % of children with AN suffered depressive syndromes (33 % moderate, 20 % severe, and 20 % mild depression), primarily in the "binge-purging" (88.8 %) and "restrictive" (72.2 %) subtypes. However, there has been no uniformity in quality and methodology used in data collection [84].

In some cases the depressive disorders are prior to the ED. Indeed, it was observed that the affective disorder may precede the ED from 26 to 49 % of cases [85, 86, 87]. Retrospective studies suggest that in about one third of cases major depression was already present before the onset of BN [88–90]. Other researchers found that the main features of depression in patients with AN of different ages are the problems of self-concept, where patients with restrictive AN show less depressive symptoms than the compulsive purgative patients [91–93]. In BN patients, the incidence of mood alteration ranges between 52 and 83 %. Higher rates of family history of depression were found in patients with BN (46 %) versus healthy population (13 %) [94, 95]. Hatsukami et al. found that 43 % of bulimic women had some symptoms of depressive disorder [96], and the 56 % reached a score of 20 on the Beck Depression Inventory (BDI) (cutoff: 10) [93].

*Anxiety and ED*. Anxiety is an emotional state that is particularly relevant in ED. Becker et al. [97] recruited 257 women with anxiety disorder and found ED symptoms (12 %) using the Eating Disorder Examination Questionnaire. Other study analyzed the presence of anxiety disorders in people diagnosed with ED; the data showed higher percentages (63.5 %) [98]. Godart et al. conducted a study with 271 people diagnosed with ED and 271 controls; results were that 47 % of women had anxiety prior to the ED [99].

*Anxiety, Body Dissatisfaction, and Perfectionism*. In this case, anxiety has been associated with the dissatisfaction with body image. New concepts were created like social physical anxiety, which refers to the discomfort experienced by people affected when they perceive that their physical appearance is being evaluated by others. For instance, the appearance of anxiety in eating disorders occurs primarily at social events where people interpret their body is being evaluated [49]. In this sense, Fairburn et al. pointed out that social anxiety situations may be a risk factor for developing an eating disorder [100]. Diehl et al. [101] studied the relationship between social physical anxiety (assessed with the Social Physique Anxiety Scale) and high scores in the evaluation of ED signs measured by the Eating Attitudes Test instrument (EAT) in 160 university students. Results reported high correlation between social physical anxiety and concern for weight loss, which indicates that these variables may be predictors of ED [101]. In addition, Rodriguez-Campayo [102] found a significant relationship between perfectionism and anxiety in a sample of 356 university students, which points to a significant relationship between the socially prescribed "perfectionism" (belief that others accept you if you are perfect) and high levels of anxiety ($r=0.35$, $p<0.05$). Therefore according to these studies, anxiety in social situations (evaluation anxiety) and social phobia are risk factors for developing an ED. Also, it seems that obsessive perfectionism associated with the desire for social acceptance is related to anxiety.

### Body Image and ED

The alteration of body image has been considered as one of the diagnostic criteria for AN in ED [4]. Given its important role in the initiation and maintenance of the disease, it is crucial in the intervention for a successful recovery [103]. However, one problem regarding this diagnosis criterion is that this is also apparent in the general population due to a significant increase of concern for physical appearance and weight.

The concept of body image consists of two components: the perception of body size and the attitude or feelings towards the body. As a result, you can define two different modes of body image dysfunction: the distortion of body size perception and cognitive-evaluative dissatisfaction (an aspect of attitude) [104]. The concept of body image disorder consists of an abnormal perception of body size. Although there is always an expected margin of error in the feelings towards the body, the presence of systematic biases in patients with ED has led to a generalization on the concept of body image distortion. Body image is composed of perceptual components, cognitive–affective and behavior. There are primarily two ways to assess body image:

(a) *The accuracy in estimating the body size* of the person based purely on perceptual judgments. There are two ways to evaluate: Depending on the object being estimated, measuring the width of specific body parts to obtain an index of body image or Body Perception Index (BPI), as proposed by Slade and Russell [105], which relate the perceived size of the person with the real size measured by an anthropometer [BPI = (perceived size/real size) × 100]. Slade index can also be applied to the Image-Marking Procedure (IMP) and the Silhouettes technique [106–109].

(b) *The attitude and feelings of an individual towards their own body*, which reflects variables of attitudes, emotion, and cognition. With this approach, using different questionnaires the Body Dissatisfaction Index was created. It measures the attitude towards weight and body shape and the attitude towards food, binge eating, and dieting. Those specific questionnaires are focused on the body image, such as the BSQ [110], and other general questionnaires, such as the EAT [111].

Numerous studies have attempted to measure the relationship between the distortion of and image perception of the body and ED. However, we are still far from knowing the relationship of this measurement as a predictor of ED.

### Family and ED

Traditionally, family has been proposed as the "cause" of ED. Conversely, the family has been proposed to "suffer" the consequences, the victims, and has been proposed as a resource for treatment as well [112]. Research about the role of family in the development and maintenance of eating disorders dates back to 1873 with the

description of AN [113]. More recent familiar variables have been studied as etiologic factors for ED [112, 113].

Early conceptualizations of AN in adolescents proposed family "psychosomatic" [114] or "anorexigenic" [114] models suggesting that specific family patterns contributed to the development, maintenance, and perpetuation of the disorder. Based almost exclusively on clinical observations, several of these theories dominated in the 1970s and 1980s, converging on a description of the anorexic family as typically fused, rigid, and dedicated to self-sacrifice and loyalty [114]. Some studies have identified certain styles of family functions with individuals with eating disorder and indicate that interactions in these families differ from those without members with eating disorders [115]. One of the most influential authors of the theory, Salvador Minuchin, developed a structural model of family [112, 114, 116], which identifies five dominant features of interactions that maintain "somatization": (a) bonding, an extreme form of proximity and intensity in family interactions; (b) overprotection, reflected in the high degree of concern that family members have over the welfare of others; (c) stiffness, characterized by a need to maintain the status quo; (d) avoidance of conflict; and (e) involvement of the child in parental conflict (marital) through triangulation. Within the community of scientists who have studied the association between type of family functioning and the presence of eating disorders, there are controversial views. Despite inconsistent results, two major findings have emerged from the accumulation of data: (a) families with eating disorders often tend to be more dysfunctional and controlling, and (b) the quality of family dynamics plays an important role in the course and outcome of the disorder [114].

## 9.3.2 Disordered Eating

### 9.3.2.1 EDNOS or DE: Profile and Diagnostic Criteria

*DSM-IV-TR Criteria* [4]: EDNOS or DE integrate partial or incomplete syndromes. Atypical disorders have a close resemblance to the AN and BN, and many of these cases may be severe and persistent as complete frames, and some may even be almost identical but do not meet diagnostic criteria for full frames. In other cases, patients have had AN or BN in the past. The overestimation of figure and weight is also present in most of these patients as well as strict control overeating.

Table 9.4 describes the "emotional eater" with binge eating and compensatory behaviors developed such as bulimic patients. It incorporates different clinical forms, all occurring with obesity, and the key feature is the lack of awareness. These patients have a strong association to deficiencies in social and occupational relations,

**Table 9.4** Binge eating disorder and other atypical syndromes

| EDNOS | Syndromes |
|---|---|
| Binge eating disorders | *Full syndrome*: (those who meet all the requirements listed in the Diagnostic International Classifications) partial or incomplete syndromes, atypical tables, residual and chronic. All can be restrictive or purgative. They can even move from one category to another. Up to 50 % of AN patients in their recovery present bulimic symptoms, a lower percentage results in BN |
| | *Partial syndrome*: is most common in teenagers. In less than 6 months, this can evolve to full symptoms and lead to a more difficult course. They should be treated early since the prognosis is very good |
| | *Syndrome ongoing*: evolution is partial in quick weight loss |
| | *Among atypical syndromes*: stand alexithymic AN or psychosomatic AN, occurring without anorexic ideation sometimes after a typical disorder (or history) |
| Other atypical disorders | *Bigorexia* or misuse of the gyms |
| | *Orthorexia* or obsession on healthy and medicinal food |
| | Night eaters |
| | *Waste syndrome*: up to one third of the AN and BN do not recover completely. Residual food symptoms, being defensive about food remains. This does not necessarily limit their quality of life |
| | *Chronic syndrome*: a small percentage of those who start with AN or BN in adolescence will become chronic and resistant to treatment. Increasing after 18 years, after relapse or late appearances |

excessive preoccupation with shape and weight, general psychopathology, significant amount of time spent on diets, emotional problems, and history of depression.

#### 9.3.2.2 Disordered Eating: Disordered Lifestyle

DE is actually a way of projecting disorderly situations of daily life such as lack of control at certain times, which can lead to lack of control on the intake of food. Our objective should not be the patient gaining or losing weight; instead it should be normalizing their relationship with food.

In DE, a necessity to resolve daily problems and lifestyles that have not been resolved may be done through self-control with food. When self-control is lost, what follows is a binge phase that also extends to the "physical appearance." Therefore, in eating disorders there is a real alteration in large parts of daily life. This explains why patients only show isolated symptoms, even in extreme cases. Thus, the behaviors associated with "eating disorders" may include compulsive dieting of specific foods, skipping meals, or avoiding food groups in order to lose weight. Individuals who eventually develop ED are more likely to have had abnormal eating patterns or habits, such as skipping meals or eating excessive amounts of unhealthy foods, even before the age of 12 [117].

#### 9.3.2.3 DE or Lack of Control: Binge Eating and DE

Binging can be commonly present in patients with bulimia nervosa. Thus, in the case of EDNOS, we are talking about an atypical bulimic disorder, where the lack of control is the foundation of the disorder. However, it can also present other disorders. In addition to binging, purging, or vomiting, alcohol abuse, substance abuse, self-mutilation, and sexual promiscuity also exist [118].

Binging is related to uncontrolled behavior with respect to intake. Sometimes this uncontrolled behavior may arise from a difficulty of impulsive control to eat or because the individual experiences anxiety to eat [118].

Binging can also be considered an addictive condition, conditioned by emotional states of mind. In certain circumstances (loneliness, stress, sadness, etc.) or when the patient comes in contact with certain food-related stimuli (especially smell, vision, and taste), binging "automatically" forages without thinking. Indeed, the susceptibility to these stimuli can increase during the start of a weight loss diet plan. This explains why binging is more likely to occur after a period of fasting or dieting [119].

There are several theories explaining this uncontrolled intake:

- *Model of binging eating*: the internalization of body ideals and body dissatisfaction with binge eating through food restriction to obtain the ideal body [120]. Binging is the result of physical and psychological susceptibility following periods of food restriction. When on a diet the body tries to defend itself from weight loss (present or future) producing an increase in hunger and desire [121].
- *Affect regulation model*: Other authors have studied the relationship between binge eating and negative emotional states. It is common for patients to binge eat to reduce negative affect (NA). These patients have a very low state of mind, dissatisfaction, and emptiness without feeling hungry [122–124]. Patients exhibit "attentional bias" (when a person focuses attention on a specific stimulus) towards self-esteem and threat avoiding food [125, 126].
- *Emotional reinforcement model*: This model emphasizes the role of reinforcement on the motivation of intake. although the relationship between neuroendocrine mediators, stress, and food is still being studied [127].

#### 9.3.2.4 Other DE: Bigorexia and Orthorexia

Currently, besides the EDNOS there are other sets of conditions most closely linked to specific areas such as physical activity (PA) or healthy eating. These have not yet been classified as ED (they are not included in the DSM) and are actually appearing more frequently in the general population.

*Bigorexia* (see Table 9.4): Bigorexia is more common among athletes or people who go to the gym on a daily basis and who are obsessed with

exercise. It is ‘‘a muscle dysmorphic disorder”, the attempt of a person to have a completely muscular, developed, and bulky body for fear of appearing weak. Symptoms include constantly looking in the mirror, feeling thin even when not, weighing several times a day, and spending more than 6 h a day exercising. Pope et al. [128] discovered a disorder related to body image, while studying the secondary effects of steroids use in bodybuilders, who despite having a body with highly developed muscles, expressed an intense fear of having a small and weak body. The self-perception of their bodies appeared distorted, with a high tendency to “underestimate the shape” or perceived it to be incredibly less bulky than it really was. This intense fear of appearing small or having small volume, together with body image distortion, caused these people to spend long periods in the gym, following protein diets and a high percentage use of anabolic hormones [129].

*Orthorexia* (see Table 9.4): It is the “appetite for the right/healthy food,” an obsession for healthy food that is considered pathological. Symptoms include dedicating more than 3 h to think about food and taking a survival kit when traveling when the food does not meet their dietary requirements.

### 9.3.3 New Models of Psychological Intervention

Traditionally, ED has been addressed from multidisciplinary approaches and is seen as a biopsychosocial disease (see Chap. 10). Until now, we can only say that few protocols have proven to be effective in the treatment of the disorder:

- Psychoanalytic models
- Cognitive-behavioral therapies
- Family therapies
- Medical treatments and hospitalization

All of these approaches are focused on three steps. The first instance centered on the weight recovery and body sizes. The second focuses on maintaining these gains. Finally, the third is about the reintegration of patients in their family and social network.

In some studies, work has begun in the development of skills to improve the patients’ adaptation to the environment, social skills, and interpersonal communication such as assertiveness. However, studies on assertiveness in ED are almost anecdotic. On the other hand, there are numerous references in the specialized literature regarding the lack of self-assertion in patients with ED. AN and BN perceive themselves to be controlled by their family and society; however, they do not act assertively with their controllers [82].

Psychological intervention models have prioritized the study and development of numerous strategies to correct deficiencies and disorders [130]. For example, one strategy used, when we find ourselves in a negative emotional state such as sadness, is finding a tool or a mode of intervention (from a psychological point of view) that minimizes the impact of sadness on the individual’s life. It is known that many of the techniques and psychological intervention strategies generate positive emotional states [131]. For example, *relaxation techniques* are effective in the treatment of anxiety disorders because they produce either direct or indirect states of consciousness suitable for contentedness. However, it is important to note that experiencing negative emotions is something inevitable and useful from an evolutionary standpoint, but such emotions are at the core of many psychological disorders [132, 133]. *The visualization, simulation, and role-playing* of pleasurable situations are strategies that motivate patients to focus on the present moment and to take positive awareness of their selves.

Cognitive therapies focus on learned optimism, explaining that it is not the negative events themselves that lead to depression [32, 134], so researchers have tried to replace these explanations by other unstable and specific explanations. In the words of Vazquez [130], their efficacy is supposed to lie in the attempt to cancel the effect of negative meanings, but this also does not mean that it needs to be replaced with positive ones.

Currently, we found different therapeutic approaches that are more focused on trying to restore the state of psychological and physical health of the patient in self-managing strategies

and/or external strategies. Some of these models try to work on enabling and equipping the individual with self-care strategies to better address emotional, social, and familial conflicts. This helps in restoring physical and emotional balance (Empowerment Model) with the objective to create patient independence and ability to take care of themselves [135].

Other models have focused on solving the problem, looking at it from every angle and trying to find "resilience" in the patients. Resilient people are those able to bounce back up over and over again in situations of crisis or conflict. From this point of view, a resilient person is able to generate positive emotions [30] or psychological health at critical moments. They are able to analyze situations from an objective point of view and take advantage of it.

Another approach in the treatment of ED and family is the development of emotional intelligence and the use of communication tools for emotional management, as well as improving social skills in patients and families of patients with ED.

Some of the newest models that focus on emotional regulation form intervention strategies based on the concept of emotional intelligence [136]. Emotional intelligence (EI) may be defined as the capacity for recognizing our own feelings and those of others and ability to handle them. In Goleman's book, "Emotional Intelligence" first published in 1995, he estimated that EI can be organized around five capabilities: auto-body awareness, empathy, listening, self-motivation, and social skills. Working with ED from this approach assumes intervening in these five areas. In this sense, the foci are both within the self and emotional awareness through the detection of their own emotions, having empathy, understanding the ability to take the place of others and, therefore, understanding them (understanding and interpersonal communication), intrinsic motivation and social skills, or interaction with the environment [137].

#### 9.3.3.1 Coaching with ED/DE Patients

Before proceeding to develop the basis for intervention of this model, it is important to describe the concept. The concept of coaching refers to the action of "trainer or guide." A coach can be considered a person who guides or trains another to learn on their own and who has the ability to work independently in their learning and in managing difficult situations [9].

Among the actions expected of a coach, one role is supporting and guiding the search for solutions. Under certain conditions such as ED, we have seen that one of the most damaged elements in the personal sphere is self-esteem. The coach helps in finding the patients' own strategies to improve self-concept and to develop the capacity for empathy (one of the most damaged skills for example in AN, in addition to alexithymia). Therefore, coaching that includes asking questions to guide the self-learning and personal skills development (i.e., interpersonal communication skills, empathy, and listening) can be a good tool to intervene ED. It is especially useful when one of the crucial elements that is affected is communication in the "household" [9], where working on the development of family communication skills becomes a primary goal of treatment. The focus is on the following areas:

Work to develop interpersonal communication skills.

- Initial interview—baseline on communication skills, "learning gaps," and areas for improvement:
  - Empathy
  - Active listening
  - Dexterity in conversation with others (social abilities)
- Analysis and evaluation of predominant communication styles in the individual "passive–aggressive communication styles"
- Analysis and evaluation of family communication patterns: comparison with patient and refocus
- Effective communication styles: in social interactions and assertive styles
- Communication tools—affective interpersonal skills and training:
  - Positive feedback
  - Empathy
  - Listening
  - Motivation

  - Analysis of misleading communication and refocus model
- Coaching process—training:
  - Make and receive criticism.
  - Coping with situations
  - Coping with ED
  - Decision-making
  - Auto-learning and auto-tools
  - Training for the acceptance of physical and mental health

The coach centers the intervention on empowering the sick person to be able to successfully tackle their health with success and responsibility.

## 9.4 Contemporary Understanding of the Issues

Although the studies and research conducted so far indicate a breakthrough in the investigation of ED, we can still find a need for further research in the general population. As we have indicated in the last section of this chapter, there are currently emerging new forms of disordered behavior in relation to nutrition and eating on one side (the case of orthorexia and other obsessive feeding patterns) and related patterns as in the case of bigorexia and other forms of behavior where this activity is done compulsively. There are also increasing cases of ED associated with a distorted body image which also deserve a place in clinical research.

## 9.5 Future Directions

Despite the current limited research on DE behavior, patterns show increases in prevalence and knowledge about its epidemiology. Future research lines should be addressed to deepen the ways of interdisciplinary intervention, where there is a high cooperation between different professionals, various areas of study, and a plethora of research on these pathologies.

Furthermore, there is increasing scientific evidence that shows the relationship between emotional variables and the development of ED and the role of stress either caused by everyday life events, traumatic situations, or high psychosocial impact.

## 9.6 Concluding Remarks

The key points on which we have worked in this chapter are:

- Psychosocial variables related to the origin, development, and maintenance of ED such as emotional patterns, patterns of eating behavior, the role of stress, the relationship between emotional variables and states of mind, the role of family, and body image
- The different diagnostic classifications according to American and European scales of these disorders.
- The new pathologies in relation to food such as orthorexia and in relation to physical activity and body image such as bigorexia
- Finally, new models of psychological intervention based on aspects of communication and emotional intelligence

## References

1. Cataluña Gd. Guía de Práctica Clínica de los TCA. In: Salut Dd, editor. Barcelona; 2009.
2. Sepúlveda A, Carrobles J, Gandarillas A. Factores asociados a la población universitaria de riesgo de trastornos del comportamiento alimentario; 2005.
3. Rivas García-Soto A, Blanco A. Eating disorders in university population. Nutr Hosp. 1998; XIII(5):282.
4. APA. American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-IV-TR. Arlington, VA: American Psychiatric Publishing; 2000.
5. Lewis M, Adnopoz J. Child and adolescent psychiatry: a comprehensive textbook. Philadelphia, PA: Lippincott Williams & Wilkins; 2002.
6. Ozier AD, Henry BW. Position of the American Dietetic Association: nutrition intervention in the treatment of eating disorders. J Am Diet Assoc. 2011;111(8):1236–41.
7. Espeset EM, Nordbø RH, Gulliksen KS, Skårderud F, Geller J, Holte A. The concept of body image disturbance in anorexia nervosa: an empirical inquiry utilizing patients' subjective experiences. Eat Disord. 2011;19(2):175–93.
8. Blanco A. Epidemiología de los Trastornos Alimentarios. Diferencias Individuales y Perfiles de

Riesgo en Población Universitaria. Diseño de una prueba diagnóstica, Madrid; 2007.
9. Blanco A. Nuevos enfoques de intervención; coaching terapéutico, empowerment y psicología positiva. In: Grau T, Martinez J, Perote A, Sánchez M, editors. Controversias en el estudio de los Trastornos del Comportamiento Alimentario (TCA). Madrid: Instituto Tomás Pascual Sanz & Fundación Instituto Transtorno Alimentario; 2012. p. 213–35.
10. Clark N. Nutrition education for elite female runners. Physician Sports Med. 1988;16(2):124–36.
11. Blasco MP, García ML, Balaguer I. Transtornos alimentarios en la actividad física: una revisión. Rev Activ Física Deporte. 1993;3:41–53.
12. Sykora C, Grilo CM, Wilfley DE, Brownell KD. Eating, weight, and dieting disturbances in male and female lightweight and heavyweight rowers. Int J Eat Disord. 1993;14(2):203–11.
13. Beals KA, Manore MM. The prevalence and consequences of subclinical eating disorders in female athletes. Int J Sport Nutr. 1994;4(2):175–95.
14. Sundgot-Borgen J. Risk and trigger factors for the development of eating disorders in female elite athletes. Med Sci Sports Exerc. 1994;26(4):414–9.
15. Miguel-Tobal F, Martin Diaz MD, Legido Arce JC. Trastornos de la conducta alimentaria en el deporte: anorexia y bulimia nerviosas. REME Rev electrónica de motivación y emoción. 2003;5:11–12. http://reme.uji.es/articulos/amiguf3982201103/texto.html. Accessed 1 Mar 2009.
16. Torstveit M, Sundgot-Borgen J. Participation in leanness sports but not training volume is associated with menstrual dysfunction: a national survey of 1276 elite athletes and controls. Br J Sports Med. 2005;39(3):141–7.
17. Aranzeta J, Pérez Rodrigo C. Evolución del niño y necesidades de alimentación. In: Aranceta J, Serra L, Rodríguez-Santos F, editors. Psicología y Nutrición. Barcelona: Masson; 2008.
18. Douglas J, Bryon M. Interview data on severe behavioural eating difficulties in young children. Arch Dis Child. 1996;75(4):304–8.
19. Martin C, Dovey T, Arcelus J. Trastornos del la ingestión alimentaria de la infancia o la niñez: Una revisión. Mex J Eat Dis. 2012;3:89–98.
20. Desor J, Maller O, Turner R. Taste in acceptance of sugars by human infants. J Comp Physiol Psychol. 1973;84(3):496.
21. Denton DA. The hunger for salt: an anthropological, physiological, and medical analysis. New York: Springer; 1982.
22. Geldard FA, O'Hehir R, Gavens D. The human senses. New York: Wiley; 1972.
23. Beauchamp GK, Moran M. Dietary experience and sweet taste preference in human infants. Appetite. 1982;3(2):139–52.
24. Birch LL, Fisher JO. Mothers' child-feeding practices influence daughters' eating and weight. Am J Clin Nutr. 2000;71(5):1054–61.
25. Murcott A, Anderson AS. The nation's diet: the social science of food choice. London: Longman; 1998.
26. Mintel. Industry Intelligence Inc, vol. 2013. Londres: Mintel; 1990.
27. Ogden J. The psychology of eating: from healthy to disordered behavior. Chichester: Wiley-Blackwell; 2011.
28. Rodríguez-Santos F, Castillo R, Gómez-Candeleda C. Variables relacionadas con la alimentación y la nutrición: variables biológicas, psicológicas y socioculturales. In: Aranzeta J, Serra L, Rodríguez-Santos F, editors. Psicología y Nutrición. Barcelona: Masson; 2008. p. 17–34.
29. Zafra E. Miedo a comer: relaciones entre alimentación, emociones y cuerpo. Paper presented at II Congreso Español de Sociología de la Alimentación, Vitoria; 2011.
30. Silva J. Regulación emocional y psicopatología: el modelo de vulnerabilidad/resiliencia. Rev Chil Neuropsiquiatr. 2005;43(3):201–9.
31. España Gd. Guía de Nutrición saludable y Prevención de los TCA: Ministerio de Sanidad y ConsumoMinisterio de Educación Ministerio del Interior; 2008.
32. Abramson L, Seligman M. Teasdale JD. Learned helplessness in humans. Critique and reformulation. J Abnorm Psychol. 1978;87:32–48.
33. Williams GJ, Chamove AS, Millar HR. Eating disorders, perceived control, assertiveness and hostility. Br J Clin Psychol. 1990;29(3):327–35.
34. Sifneos PE. The prevalence of 'alexithymic' characteristics in psychosomatic patients. Psychother Psychosomat. 2010;22(2–6):255–62.
35. Bruch H. The golden cage: the enigma of anorexia nervosa. Cambridge, MA: Harvard University Press; 2001.
36. Way K. Anorexia nervosa and recovery: a hunger for meaning. London: Routledge; 1993.
37. Arnow B, Kenardy J, Agras WS. Binge eating among the obese: a descriptive study. J Behav Med. 1992;15(2):155–70.
38. Overton A, Selway S, Strongman K, Houston M. Eating disorders—the regulation of positive as well as negative emotion experience. J Clin Psychol Med Settings. 2005;12(1):39–56.
39. Schmidt U, Treasure J. Anorexia nervosa: valued and visible. A cognitive-interpersonal maintenance model and its implications for research and practice. Br J Clin Psychol. 2006;45(3):343–66.
40. Lemberg R, Cohn L. Eating disorders: a reference sourcebook. New York: Greenwood Reference C; 1998.
41. Casper RC. Personality features of women with good outcome from restricting anorexia nervosa. Psychosom Med. 1990;52(2):156–70.
42. Casper RC, Hedeker D, McClough J. Personality dimensions in eating disorders and their relevance for subtyping. J Am Acad Child Adolesc Psychiatry. 1992;31(5):830–40.
43. Montero García-Celay I, León O. A guide for naming research studies in psychology. Int J Clin Health Psychol. 2007;7(3):847–62.
44. Ramos-Alvarez MM, Moreno-Fernández MM, Valdés-Conroy B, Catena Martínez A. Criteria of the

peer review process for publication of experimental and quasi-experimental research in psychology: a guide for creating research papers. Int J Clin Health Psychol. 2008;8(3):751–64.
45. Beato-Fernández L, Rodríguez-Cano T, Belmonte-Llario A, Martínez-Delgado C. Risk factors for eating disorders in adolescents. Eur Child Adolesc Psychiatry. 2004;13(5):287–94.
46. Ghaderi A, Scott B. Prevalence, incidence and prospective risk factors for eating disorders. Acta Psychiatr Scand. 2001;104(2):122–30.
47. Kim J-H, Lennon SJ. Mass media and self-esteem, body image, and eating disorder tendencies. Cloth Textiles Res J. 2007;25(1):3–23.
48. Alpers GW, Tuschen-Caffier B. Negative feelings and the desire to eat in bulimia nervosa. Eat Behav. 2002;2(4):339–52.
49. Corstorphine E. Cognitive–emotional–behavioural therapy for the eating disorders: working with beliefs about emotions. Eur Eat Disord Rev. 2006;14(6):448–61.
50. Heatherton T, Baumeister RF. Binge eating as escape from self-awareness. Psychol Bull. 1991;110(1): 86–108.
51. Moreno S. Ansia por la comida y transtornos de la conducta alimentaria. Granada: Universidad de Granada; 2003.
52. Rodríguez S, Mata J, Moreno S. Psicofisiología del ansia por la comida y la bulimia nerviosa. Clín Salud. 2007;18(1):99–118.
53. Stice E, Nemeroff C, Shaw HE. Test of the dual pathway model of bulimia nervosa: evidence for dietary restraint and affect regulation mechanisms. J Soc Clin Psychol. 1996;15(3):340–63.
54. Waters A, Hill A, Waller G. Bulimics' responses to food cravings: is binge-eating a product of hunger or emotional state? Behav Res Ther. 2001;39(8):877–86.
55. Franco A. De Cazador a gourmet. Sao Paolo: SENAC; 2001.
56. Sloan D. Gastronomia, restaurantes e comportamento do consumidor. Manole: Tradução Sonia Bidutte. Barueri; 2005.
57. Del Valle T. Mujer y nuevas socializaciones: su relación con el poder y el cambio *Kobie*. Antropol Cult. 1992;6:5–15.
58. Cepeda-Benito A, Gleaves DH. A critique of food cravings research: theory, measurement, food intake. In: Hetherington MM, editor. Food cravings and addition. Leatherhead: Food Research Association; 2001. p. 1–12.
59. Tiffany ST. A cognitive model of drug urges and drug-use behavior: role of automatic and nonautomatic processes. Psychol Rev. 1990;97(2):147.
60. Robinson TE, Berridge KC. Addiction. Annu Rev Psychol. 2003;54:25–53.
61. Kalivas PW, Volkow ND. The neural basis of addiction: a pathology of motivation and choice. Am J Psychiatry. 2005;162(8):1403–13.
62. Silva J. Sobrealimentación inducida por la ansiedad. Parte II: Un marco de referencia neurocientífico para el desarrollo de técnicas psicoterapéuticas. Ter Psicol. 2008;26:99–115.
63. Davey G, Chapman L. Disgust and eating disorder symptomatology in a non-clinical population: the role of trait anxiety and anxiety sensitivity. Clin Psychol Psychother. 2009;16(4):268–75.
64. Holtkamp K, Müller B, Heussen N, Remschmidt H, Herpertz-Dahlmann B. Depression, anxiety, and obsessionality in long-term recovered patients with adolescent-onset anorexia nervosa. Eur Child Adolesc Psychiatry. 2005;14:106–10.
65. Buckner JD, Silgado J, Lewinsohn PM. Delineation of differential temporal relations between specific eating and anxiety disorders. J Psychiatr Res. 2010;44(12):781–7.
66. Kiyotaki Y, Yokoyama K. Relationships of eating disturbances to alexithymia, need for social approval, and gender identity among Japanese female undergraduate students. Pers Indiv Diff. 2006;41(4):609–18.
67. Pascual A, Etxebarria I, Cruz MS. Emotional differences between women with different types of eating disorders. Int J Psychol Psychol Ther. 2011;11(1):1–11.
68. Iancu I, Cohen E, Yehuda YB, Kotler M. Treatment of eating disorders improves eating symptoms but not alexithymia and dissociation proneness. Compr Psychiatry. 2006;47(3):189–93.
69. Ridout N, Thom C, Wallis DJ. Emotion recognition and alexithymia in females with non-clinical disordered eating. Eat Behav. 2010;11(1):1–5.
70. Gartner AF, Marcus RN, Halmi K, Loranger AW. DSM-III—R personality disorders in patients with eating disorders. Am J Psychiatry. 1989;146:1585–92.
71. Wonderlich SA, Swift WJ, Slotnick HB, Goodman S. DSM-III-R personality disorders in eating-disorder subtypes. Int J Eat Dis. 1990;9(6):607–16.
72. Braun DL, Sunday S, Halmi K. Psychiatric comorbidity in patients with eating disorders. Psychol Med. 1994;24(04):859–67.
73. Steiger H, Stotland S. Prospective study of outcome in bulimics as a function of axis-II comorbidity: long-term responses on eating and psychiatric symptoms. Int J Eat Dis. 1996;20(2):149–61.
74. Matsunaga H, Kiriike N, Nagata T, Yamagami S. Personality disorders in patients with eating disorders in Japan. Int J Eat Dis. 1998;23(4):399–408.
75. Echeburúa E, Marañón I. Comorbilidad de las alteraciones de la conducta alimentaria con los trastornos de personalidad. Psicol Conduct. 2001;9(3):513–25.
76. Sarason IG, Pierce GR. Cognitive interference: theories, methods, and findings. Mahwah, NJ: Psychology Press; 1996.
77. Vázquez C, Ring JM. Altered cognitions in depression: are dysfunctional attitudes stable? Pers Indiv Diff. 1993;15(4):475–9.
78. Dolan B, Evans C, Norton K. Multiple axis-II diagnoses of personality disorder. Br J Psychiatry. 1995;166(1):107–12.
79. Garner DM, Myerholtz LE. Eating disorders. In: Bellack AS, Hersen M, editors. Comprehensive clinical psychology. Oxford: Pergamon; 1998.

80. Grilo CM, Levy KN, Becker DF, Edell WS, McGlashan TH. Comorbidity of DSM-III-R axis I and II disorders among female inpatients with eating disorders. Psychiatr Serv (Washington, DC). 1996;47(4):426.
81. Striegel-Moore RH, Garvin V, Dohm FA, Rosenheck RA. Psychiatric comorbidity of eating disorders in men: a national study of hospitalized veterans. Int J Eat Dis. 1999;25(4):399–404.
82. Behar R. Relación entre los trastornos de la conducta alimentaria y los trastornos afectivos: Una revisión de la evidencia. Transtor Animo. 2006;2(1):44.53.
83. Herzog DB, Nussbaum KM, Marmor AK. Comorbidity and outcome in eating disorders. Psychiatr Clin North Am. 1996;19(4):843–59.
84. Lucka I. Depression syndromes in patients suffering from anorexia nervosa. Psychiatr Pol. 2004;38(4): 843–59.
85. Piran N, Kennedy S, Garfinkel P, Owens M. Affective disturbance in eating disorders. J Nerv Mental Dis. 1985;173(7):395–400.
86. Hudson JI, Pope HG, Jonas JM, Yurgelun-Todd D. Phenomenologic relationship of eating disorders to major affective disorder. Psychiatry Res. 1983;9(4): 345–54.
87. Walsh BT, Roose SP, Glassman AH, Gladis M, Sadik C. Bulimia and depression. Psychosom Med. 1985;47(2):123–31.
88. Casper RC, Offer D, Ostrov E. The self-image of adolescents with acute anorexia nervosa. J Pediatr. 1981;98(4):656–61.
89. Laessle RG, Kittl S, Fichter MM, Wittchen H-U, Pirke KM. Major affective disorder in anorexia nervosa and bulimia. A descriptive diagnostic study. Br J Psychiatry. 1987;151(6):785–9.
90. Fairburn CG, Welch SL, Doll HA, Davies BA, O'Connor ME. Risk factors for bulimia nervosa: a community-based case-control study. Arch Gen Psychiatry. 1997;54(6):509.
91. Casper RC, Eckert ED, Halmi KA, Goldberg SC, Davis JM. Bulimia: its incidence and clinical importance in patients with anorexia nervosa. Arch Gen Psychiatry. 1980;37(9):1030.
92. Eckert ED, Goldberg SC, Halmi KA, Casper RC, Davis JM. Depression in anorexia nervosa. Psychol Med. 1982;12(01):115–22.
93. Rosen AM, Murkofsky CA, Steckler NM, Skolnick NJ. A comparison of psychological and depressive symptoms among restricting anorexic, bulimic anorexic, and normal-weight bulimic patients. Int J Eat Dis. 1989;8(6):657–63.
94. Herzog DB. Are anorexic and bulimic patients depressed. Am J Psychiatry. 1984;141(12):1594–7.
95. Laessle RG, Wittchen HU, Fichter MM, Pirke KM. The significance of subgroups of bulimia and anorexia nervosa: lifetime frequency of psychiatric disorders. Int J Eat Dis. 1989;8(5):569–74.
96. Hatsukami D, Eckert E, Mitchell JE, Pyle R. Affective disorder and substance abuse in women with bulimia. Psychol Med. 1984;14(3):701–4.
97. Becker CB, DeViva JC, Zayfert C. Eating disorder symptoms among female anxiety disorder patients in clinical practice: the importance of anxiety comorbidity assessment. J Anxiety Dis. 2004;18(3): 255–74.
98. Kaye WH, Bulik CM, Thornton L, Barbarich N, Masters K. Comorbidity of anxiety disorders with anorexia and bulimia nervosa. Am J Psychiatry. 2004;161(12):2215–21.
99. Godart NT, Flament MF, Curt F, et al. Anxiety disorders in subjects seeking treatment for eating disorders: a DSM-IV controlled study. Psychiatry Res. 2003;117(3):245–58.
100. Fairburn CG, Harrison PJ. Eating disorders. Lancet. 2003;361(9355):407–16.
101. Diehl NS, Johnson CE, Rogers RL, Petrie TA. Social physique anxiety and disordered eating: what's the connection? Addict Behav. 1998;23(1):1.
102. Rodríguez-Campayo MA, Rojo-Moreno L, Ortega-Toro E, Sepúlveda-García AR. Adaptación de la escala multidimensional del perfeccionismo a estudiantes universitarios españoles. Ansiedad Estrés. 2009;15:13–27.
103. Bruch H. Perceptual and conceptual disturbances in anorexia nervosa. Psychosom Med. 1962;24: 187–94.
104. Skrzypek S, Wehmeier P, Remschmidt H. Body image assessment using body size estimation in recent studies on anorexia nervosa. A brief review. Eur Child Adolesc Psychiatry. 2001;10(4):215–21.
105. Slade P, Russell G. Awareness of body dimensions in anorexia nervosa: cross-sectional and longitudinal studies. Psychol Med. 1973;3(02):188–99.
106. Askevold F. Image Marking Procedure. Sex and Gender Issues: A Handbook of Tests and Measures. 1990;(4):72.
107. Williamson DA, Kelley ML, Davis CJ, Ruggiero L, Blouin DC. Psychopathology of eating disorders: a controlled comparison of bulimic, obese, and normal subjects. J Consult Clin Psychol. 1985;53(2): 161–6.
108. Olson JM, Zanna MP, Herman CP. Self-inference processes: the Ontario symposium, vol. 6. Mahwah, NJ: Psychology Press; 2013.
109. Askevold F. Measuring body image. Psychother Psychosomat. 2010;26(2):71–7.
110. Cooper PJ, Taylor MJ, Cooper Z, Fairbum CG. The development and validation of the Body Shape Questionnaire. Int J Eat Dis. 1987;6(4):485–94.
111. Garner DM, Garfinkel PE. The Eating Attitudes Test: an index of the symptoms of anorexia nervosa. Psychol Med. 1979;9(2):273–9.
112. Carrasco E. Perspectiva familiar en el tratamiento de los trastornos de la conducta alimentaria. In: Behar R, Figueroa G, editors. Anorexia nerviosa y bulimia: clínica y terapéutica. Mediterráneo: Santiago; 2004. p. 235–57.
113. Fornari V, Wlodarczyk-Bisaga K, Matthews M, Sandberg D, Mandel FS, Katz JL. Perception of family functioning and depressive symptomatology in

individuals with anorexia nervosa or bulimia nervosa. Compr Psychiatry. 1999;40(6):434–41.
114. Cook-Darzens S, Doyen C, Falissard B, Mouren MC. Self-perceived family functioning in 40 French families of anorexic adolescents: implications for therapy. Eur Eat Disord Rev. 2005;13(4):223–36.
115. Kog E, Vandereycken W. Family interaction in eating disorder patients and normal controls. Int J Eat Dis. 1989;8(1):11–23.
116. Vidović V, Jureša V, Begovac I, Mahnik M, Tocilj G. Perceived family cohesion, adaptability and communication in eating disorders. Eur Eat Disord Rev. 2005;13(1):19–28.
117. Fernández-Aranda F, Krug I, Granero R, et al. Individual and family eating patterns during childhood and early adolescence: an analysis of associated eating disorder factors. Appetite. 2007; 49(2):476–85.
118. Lacey JH, Evans CDH. The Impulsivist: a multi-impulsive personality disorder. Br J Addict. 1986; 81(5):641–9.
119. Vanderlinden J, Vandereycken W. Trauma, disociación y descontrol de los impulsos en los trastornos alimentarios. Ediciones Granica SA; 2002.
120. Cooper PJ, Fairburn CG. Confusion over the core psychopathology of bulimia nervosa. Int J Eat Dis. 1993;13(4):385–9.
121. Calvo R. El espectro del atracón como síntoma. In: Grau T, Martinez J, Perote A, Sánchez M, editors. Controversias en el estudio de los Transtornos del Comportamiento Alimentario (TCA). Madrid: Instituto Tomás Pascual Sanz & Fundación Instituto Transtorno Alimentario; 2012. p. 47–59.
122. Powell AL, Thelen MH. Emotions and cognitions associated with bingeing and weight control behavior in bulimia. J Psychosomat Res. 1996;40(3): 317–28.
123. Lynch WC, Everingham A, Dubitzky J, Hartman M, Kasser T. Does binge eating play a role in the self-regulation of moods? Integr Physiol Behav Sci. 2000;35(4):298–313.
124. Hilbert A, Tuschen-Caffier B. Maintenance of binge eating through negative mood: a naturalistic comparison of binge eating disorder and bulimia nervosa. Int J Eat Dis. 2007;40(6):521–30.
125. Medina-Pradas C, Navarro JB, López SR, Grau A, Obiols JE. Further development of a scale of perceived expressed emotion and its evaluation in a sample of patients with eating disorders. Psychiatry Res. 2011;190(2):291–6.
126. Waller G, Barter G. The impact of subliminal abandonment and unification cues on eating behavior. Int J Eat Dis. 2005;37(2):156–60.
127. Tomiyama AJ, Dallman MF, Epel ES. Comfort food is comforting to those most stressed: evidence of the chronic stress response network in high stress women. Psychoneuroendocrinology. 2011;36(10):1513–9.
128. Pope HG, Katz DL, Hudson JI. Anorexia nervosa and "reverse anorexia" among 108 male bodybuilders. Compr Psychiatry. 1993;34(6):406–9.
129. Grau A. Controversias sobre los trastornos alimentarios: Vigorexia y otros trastornos de la imagen. Instituto Tomás Pascual y Fita; 2012.
130. Vázquez C. La Psicología Positiva en perspectiva. Papeles Psicól. 2006;27:1–2.
131. Fredrickson BL. Cultivating positive emotions to optimize health and well-being. Prevention and Treatment, 2000;3(0001a). Retrieved from content. apa.org/journals/pre/3/1/1a.html.
132. O'Leary A. Stress, emotion, and human immune function. Psychol Bull. 1990;108(3):363.
133. Watts FN. Applications of current cognitive theories of the emotions to the conceptualization of emotional disorders*. Br J Clin Psychol. 1992;31(2): 153–67.
134. Peterson C, Seligman EP, Vaillant GE. Pessimistic explanatory style is a risk factor for physical illness: a thirty-five-year longitudinal study. J Pers Soc Psychol. 1988;55(1):23–7.
135. Vazquez C, Hervás G, Samuel M. Intervenciones clínicas basadas en la psicología positiva: fundamentos y aplicaciones. Psicol Conduct. 2006;14(3): 401–32.
136. Goleman D. Inteligencia emocional. Editorial Kairós SA; 2012.
137. Jáuregui Lobera I. Autoayuda y participación de la familia en el tratamiento de los trastornos del comportamiento alimentario. Trastornos de la Conducta alimentaria. 2005;2:158–98.

# Disordered Eating Issues in Active Children and Adolescence

10

Maria Fernandez-del-Valle, Marta Montil Jiménez, and Lesley Carraway

**Abstract**

The development of a child's body image is strongly influenced by peers, school, family, society, and the media. While it is important for children to learn healthy eating habits and attitudes toward physical activity early in life, it is also essential that children learn to value themselves and develop a strong self-esteem. Without such positive influences, children and adolescents may develop disordered eating habits, which include the use of diet pills, laxatives, and excessive exercise. These disordered eating habits may manifest themselves as clinical eating disorders, such as anorexia and bulimia. It is of great importance that children receive support and positive influences from family, peers, and school to develop a healthy self-image and prevent the formation of disordered eating habits and eating disorders.

**Keywords**

Disordered eating • Risk and protective factors • Body image • Eating disturbances

## 10.1 Learning Objectives

After completing this chapter, you should have an understanding of:

- Body image disturbances and eating disturbances and prevalence.
- The connection between eating disorders, risk behaviors, and psychological disorders.
- Risk and protective factors for body image and eating disturbances.
- Promoting healthy body image and preventing eating disturbances among youth.
- The application of Social Development Strategy in the health promotion and prevention.

M. Fernandez-del-Valle, Ph.D. (✉)
Department of Health, Exercise, and Sport Sciences, Texas Tech University, Exercise and Sport Sciences Building, 3204 Main, Lubbock, TX, USA
e-mail: maria.fernandez-del-valle@ttu.edu

M.Montil Jiménez, Ph.D.
Department of Kinesiology and Athletic Training, Universidad Europea de Madrid, Calle Tajo s/n, Villaviciosa de Odón, Madrid 28670, Spain
e-mail: marta.montil@uem.es

L. Carraway, M.S.
Department of Health, Exercise, and Sports Sciences, Texas Tech University HESS, Box 43011, Lubbock, TX 79404, USA
e-mail: lesley.arbona@ttu.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_10, 

## 10.2 Introduction

The importance of healthy habits (i.e., physical activity and diet) and their contribution to improve the quality of life and health is well known [1, 2]. Both quality of life and health are linked to environmental influences and lifestyle of individuals. Physical Activity (PA), as well as a healthy dietary intake, is highly important during childhood and adolescence, playing a main role in the acquisition of healthy habits and behaviors through the lifespan [3–9]. From the environmental or social factors, family, in general, particularly parents, is the main socializing agent that stimulates healthy practices in children and adolescents [8–13].

Moreover, friends are important socializing agents that increase its effect during adolescence, determine the practice of PA, and promote healthy habits in the future. PA performed in school and high school determines the acquisition of adequate behavioral patterns for health and well-being [14]. Research has reported that children and adolescents show a higher PA participation, and it is during early adulthood when practice starts to decrease. This progressive reduction on PA is greater in females and depends on multiple factors related to psychological and environmental variables [15–19], reflecting higher activity levels and participation in male practitioners.

The Center for Disease Control and Prevention (CDC) has identified six categories of health-risk behaviors that contribute to the leading causes of morbidity and mortality among youth and adults—alcohol and other drug use, injury and violence, tobacco use, sexual behaviors, physical inactivity, and unhealthy dietary behaviors. Not only are these behaviors linked to health problems, they also contribute to numerous educational and social problems that negatively impact our nation. These interrelated health-risk behaviors are preventable, and usually begin in childhood and persist into adulthood [20].

*The Biopsychosocial (BPS) model*: This model was first postulated by Gorge L. Engel at the University of Rochester [21] as, "the need for a new medical model." The biopsychosocial model (BPS model) was termed to enclose the essential factors that trigger illnesses and disabilities in human beings, postulating three main factors: biological, psychological, and social; however, this model varies across cultures [22]. The biological component analyzes how the cause of the disease derives from the body's functioning (physiology, genetics, etc.); the psychological component analyzes the potential psychological causes (self-control, emotional turmoil, negative thinking, self-image, self-esteem, etc.); and the social component investigates how the environment can influence health (culture, family, friends, technology, religion, or socioeconomic status) [22].

*Applicability of the model*: Since WHO defined Health as, "A state of complete physical, mental and social well-being and not merely the absence of disease or infirmity" [23], health has to be analyzed from a BPS perspective. Given that, physical, mental, and social components are vital to support health, and an open-minded point of view is required to explore the key components influencing healthy habits in a physically active population (environmental influences, physical functioning, and mental health).

Historically, scientists have tried to explain the behavioral process regarding specific topics, such physical inactivity, dietary habits, substance abuse, or sexual behaviors, by explanatory models or theories [24–27]. Interestingly, they were focused on the psychological factors contributing to the development of unhealthy behaviors. Notwithstanding, BPS-based theories have gained ground due to the multifactorial factors involved in the origin of unhealthy behaviors [21, 22].

## 10.3 Research Findings

### 10.3.1 Risk and Protective Factors for a Healthy Lifestyle

There are unmodifiable and modifiable factors affecting human beings' behaviors, and both are summarized in Fig. 10.1 [28].

1. Unmodifiable: Factors that are noncontrollable or non-changeable are inherited by the

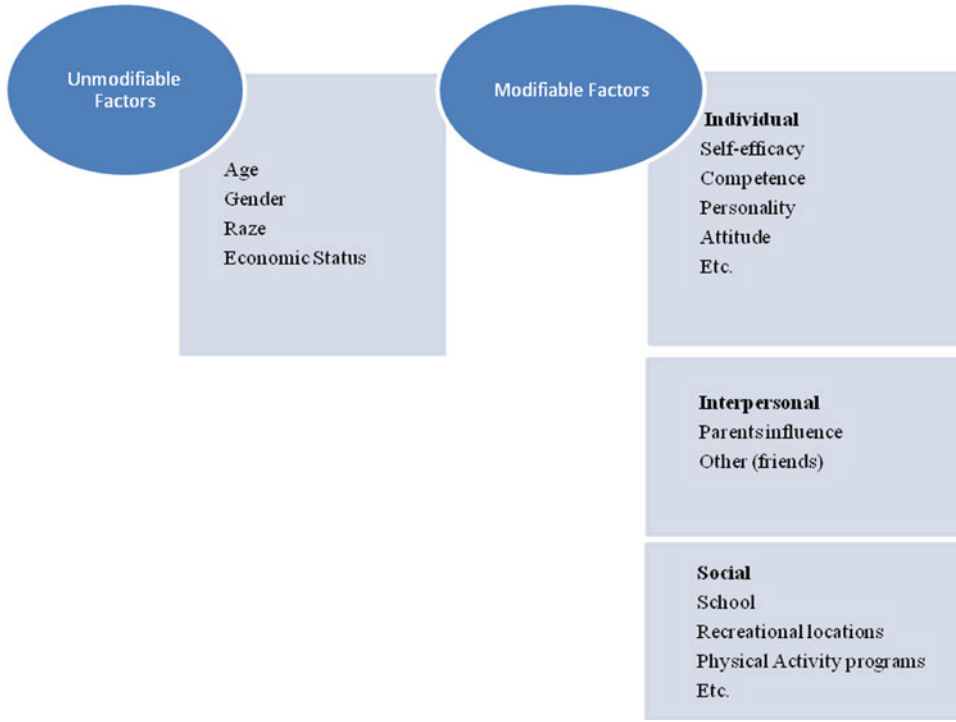



**Fig. 10.1** Unmodifiable and modifiable factors affecting the human behavior

individual or are beyond his/her control, such as age, sex, race, genes, etc.

2. Modifiable: Factors that are controllable or changeable, such as lifestyle, substance abuse, or poor dietary habits.

These categories have a combined potential for harm or benefit when they act together. Therefore, growing research has focused in detecting risk and protective modifiable factors and changing those by presenting preventive-targeted interventions.

Numerous research studies have identified multiple risk and protective factors for body image and eating disturbances that lead to disordered eating behaviors. These can be divided into three primary categories: biological, psychological, and social (familial and cultural).

Generally speaking, if risk factors are those conditions that increase the likelihood that an individual will develop an eating problem, the protective factors are those conditions that mitigate the risk (i.e., decrease the chances that disordered eating will occur). The following discussion is not inclusive, but represents an overview of some of the key evidence-based risk and protective factors within biological, psychological, and social categories. Body image and eating disturbances are extremely complicated and appear to result from a complex interplay among a myriad of risk factors and deficient protective factors for healthy behaviors.

#### 10.3.1.1 Risk Factors

The CDC's Youth Risk Behavior Surveillance System (YRBSS) monitors these six categories of health-risk behaviors among youth through national, state, and local data collection [29]. Data collected during September 2008–December 2009 revealed the following selected information about dietary-related behaviors among US students in grades 9–12: 45.6 % of students were trying to lose weight during the 30 days preceding the survey; 10.6 % of students had gone without eating for 24 h or more to lose weight or to keep from gaining weight during the 30 days preceding the survey; 5.0 % of students

had taken diet pills, powders, or liquids without a physician's advice to lose weight or to keep from gaining weight; and 4.0 % of students had vomited or taken laxatives to lose weight or to keep from gaining weight during the 30 days preceding the survey [29]. Those risk factors can be classified by the three components of the Biopsychosocial model:

(a) Biological risk factors: Biological risk factors include genetic predisposition to eating disorders, mood disorders [30], neurochemical (e.g., serotonin) imbalances [31, 32], and early puberty [33–35].
(b) Psychological risk factors: Psychological risk factors encompass negative body image [36] and body dissatisfaction [37], temperament (e.g., negative emotionality) [38, 39], personality characteristics (e.g., perfectionism) [40], low self-esteem [35, 41, 42], inadequate coping skills [43], substance abuse [44], and overweight/obesity [43, 45–47].
(c) Social risk factors: Familial risk factors include eating disorders in first-degree biological relatives [43, 48–50], maladaptive parental behaviors and dysfunctional family relations [34, 36, 44, 51–54], family pressure to adhere to the thin ideal [55], alcohol misuse [37], and physical or sexual abuse [54, 56, 57], although the latter aspect is controversial.

It is important to point out that familial influences are often difficult to separate from broader sociocultural influences because most (if not all) families are influenced to some degree by current societal standards [58]. With that in mind, other cultural risk factors include societal glamorization of the thin ideal [59], media exposure promoting thinness [60, 61], and peer influences promoting dieting and adherence to the thin standard [55, 62]. It is important to note that some of these risk factors were identified through cross-sectional studies; therefore, they cannot completely meet the definition of "risk factor" until they are found to be significant in longitudinal studies [43, 63]. Moreover, there may be critical developmental periods during which exposure to risk factors may have greater influence on the development of eating disturbances (e.g., puberty) [44].

#### 10.3.1.2 Protective Factors

In contrast, there is little research about protective factors and how they may buffer individuals against developing eating disturbances and clinical eating disorders (EDs) [43, 44]. Individual protective factors that have been suggested include the following:

(a) Biological protective factors: A genetic predisposition for slimness [64, 65]. Research has reported that those individuals who maintain their body mass index (BMI) between a normal to low range ( 18 kg/m$^2$) [44, 66] are associated with lower incidence of developing ED's or suffering from eating and body image disturbances [44].
(b) Psychological protective factors: Self-directedness and assertiveness [64], the ability to effectively cope with life stressors [64, 67], and high self-esteem [44, 67].
(c) Social protective factors: The primary protective factor that has received the most empirical support is positive family relationships [44, 68]. Protective factors that emerged from a qualitative research study with high school girls were self-acceptance, family acceptance, positive peer influences, and knowledge about the hazards of dieting [69]. Healthy habits during adulthood are associated with those developed during childhood [4, 70, 71]. Active children develop abilities, habits, and enjoyment of PA that increases the probability of continuing with its practice in the future. Those females who were involved in regular PA activity showed greater healthy behaviors later in adulthood [72]. The World Organization for Mental Health has postulated regular exercise practice as a protective factor to those suffering from mental disorders [18].

In addition to positive relationships with parents [34, 51, 68], other familial protective factors that have been considered include: living in a family that does not overemphasize body weight and physical attributes [64, 65], living in a family where parents do not misuse alcohol [73], and social support from the family [74].

Sociocultural factors that may be protective are: cultural messages that embrace different body shapes and sizes [64], participation in sports

that do not emphasize thinness for successful performance [42, 64, 75], close relationships with friends who do not overstress body weight [64, 76], and social support from peers [67, 68, 74].

### 10.3.2 Eating Disturbances and Body Image Issues in Active Children and Adolescents

There is a broad evidence base that encompasses numerous facets of body image and eating disturbances that negatively impact the whole child. Researchers and clinicians now recognize the triad of body image difficulties, eating disorders, and obesity as an interrelated set of body weight and shape disturbances that cause substantial problems for children and adolescents [77]. In addition, many researchers have viewed eating problems on a continuum, beginning with body dissatisfaction and weight concerns and ending with clinical eating disorders [78, 79]. In this chapter, the term "eating disorders" (EDs) refers to anorexia nervosa (AN) and bulimia nervosa (BN). The terms "eating disturbances" and "disordered eating" are broader and refer to a range of unhealthy diet-related behaviors, such as obsession with body weight and shape, excessive restrictive eating, skipping meals, laxative and diet pill use, cycles of binge eating and dieting, self-induced vomiting, and excessive exercise with the sole purpose of "purging" calories obtained from dietary intake. The purpose of this chapter is to present an overview of some of the key findings concerning body image and eating disturbances in children and adolescents, and discuss viable avenues for promoting healthy body image and preventing eating disturbances among this population.

#### 10.3.2.1 Eating Disturbances

Clinically diagnosable EDs [48] are atypical among prepubescent children [77, 80]; yet they rank as the third most common chronic illness among adolescent females, with an incidence of up to 5 % [81–83]. EDs are related to other risk behaviors, such as tobacco use, alcohol and other drug abuse, sexual activity, and suicide attempts [48]. EDs often lead to multiple negative outcomes that affect the whole child. These outcomes may range from a preoccupation with eating that can significantly hinder healthy growth and development, to deleterious medical complications, such as severe malnutrition, osteoporosis, acute psychiatric emergencies, heart and other organ damage, and even death [84]. In fact, death rates from eating disorders are among the highest for any mental illness [85]. More specifically, the mortality rate among those with AN is approximately 12 times higher than the death rate among US females ages 15–24 from all causes of death [86].

The average age of onset for AN is 14–18 years, and younger than 13 years if early-onset of eating disorders (EOED) [87], and late adolescence or early adulthood for BN [48]. Younger children may have significant problems related to body image, eating, and weight management that do not meet the diagnostic criteria for an ED, but can increase the risk for developing an ED later. The American Academy of Pediatrics [88], and Society for Adolescent Medicine [84], assert that an ED can still exist in the absence of established diagnostic criteria. Patients who do not fully meet the Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV) criteria for anorexia or bulimia, but experience the same medical and psychological consequences of these disorders, usually are diagnosed with an "eating disorder not otherwise specified" (EDNOS) [89]. The majority of adolescents in ED treatment centers meet the EDNOS criteria [84].

Warning signs include obsessive thinking about food, weight, shape, or exercise; unhealthy weight management practices; and failure to maintain a healthy body weight/composition for gender and age. Similarly, along the spectrum of eating disturbances, researchers often use the terms "partial syndrome" or "sub-clinical" to indicate similar characteristics. Although most children may not meet DSM criteria for an ED, those who exhibit symptoms of EDNOS or a sub-clinical eating disturbance can still suffer considerable social and educational impairment that may require clinical intervention [77]. In fact, about the 5 % of the population of adolescents in the USA suffer from sub-clinical ED or EDNOS [90]. Even in the absence of clinical intervention, obsessive inner dialogues about body weight and

shape, and the consequences of disordered eating significantly interfere with the developmental needs and resilience of children and adolescents [91, 92].

#### Eating Disturbances Comorbidity

Eating disorders often coexist with other psychological disorders, such as depression, obsessive-compulsive disorder, anxiety, bipolar disorder, substance abuse (including prescription drugs), and personality disorders (e.g., borderline personality) [48, 84, 93]. In some cases, the ED is a secondary symptom to an underlying psychological disorder; and in other cases, the psychological disorder may be secondary to the eating disorder. Adding to this complexity, a young person with an ED may present other self-destructive behaviors, such as self-injury (e.g., cutting) [94].

#### Eating Disturbances Approach

ED are comprised of a complex array of biopsychosocial issues that should be addressed by a multidisciplinary team of medical, nutritional, mental health, and nursing professionals who have expertise in child–adolescent health and are experienced in treating Body Image disturbances (BID) and EDs [84].

Although early interdisciplinary treatment increases the likelihood of successful recovery, there are numerous barriers to this type of health care, including time, cost, and inadequate insurance benefits. In addition, patients and their families often exhibit ambivalence or resistance to the treatment process [84]. Failure to detect an ED in its early stages can exacerbate the illness and make it much more difficult to treat [84, 88].

### 10.3.2.2 Body Image Disturbances

BID, eating concerns, and weight issues clearly present major challenges for children and youth. Data reveal that, among American high school students, 25 % of girls and 11 % of boys engage in disordered eating behaviors, including bingeing, vomiting, fasting, laxative and diet pill use, and compulsive exercise [95]. Furthermore, childhood obesity in the USA has increased at an alarming rate, with approximately 12.5 million American children ages 2 through 19 classified as obese [96]. Research has indicated that American children, some as young as 6 years of age [97, 98], are dissatisfied with their body shapes or weights [80, 99, 100]. Up to 70 % of normal-weight adolescent girls report feeling fat and engaging in unhealthy eating practices for weight loss purposes [101]. Studies also have shown that children as young as 8 and 9 engage in dieting practices [41, 102–104]. Moreover, girls who frequently diet are 12 times as likely to engage in binge eating as girls who do not diet [105].

Numerous research studies show that high numbers of Caucasian American children experience body dissatisfaction [80]. However, there is evidence that body dissatisfaction may be increasing among girls in minority ethnic groups. For example, Neumark-Sztainer and colleagues [106], found high levels of dieting and disordered eating among all ethnic groups. They also discovered that, although dieting was more prevalent among adolescents from higher socioeconomic families, disordered eating was widespread in adolescents from lower socioeconomic families as well. Other research has shown that African American adolescent females have reported being more comfortable with their bodies than those among other ethnic groups, but they also have exhibited a high drive for thinness [99]. In addition, there are indications that Mexican-American female adolescents desire to be thinner [107].

There also are developmental trends in body image and weight concerns, and these trends vary by gender and across ethnic groups. For example, research suggests that body dissatisfaction and weight concerns increase with age, particularly among females [68, 80, 108, 109]. These trends are important because there is evidence that body dissatisfaction in young girls can lead to eating problems [36, 80, 110], and early-onset depression [111, 112], later.

In terms of assessing body image, most researchers focus on two separate components of BID—perceptual body-size distortion and the affective (attitudinal) aspect. Perceptual body-size distortion is comprised of inaccurate perceptions of one's body size (e.g., individuals with eating disorders often overestimate their actual body size). The affective element relates to dissatisfaction

with one's body size, shape, or some other aspect of physical appearance [113]. Although most studies have focused on the distortion component, greater consistency has been found by using attitudinal measures. Moreover, these two components appear to function independently [114]. Therefore, body image is considered multidimensional, and the assessment of BID requires a variety of methods and techniques [115]. There are a variety of assessment instruments, most of which have been developed with adult samples [116]. Banasiak and colleagues [116] examined reliability in numerous assessments that measured dietary restraint and body concerns. Their findings suggest that many of the instruments developed on adults can be used with middle adolescent girls (ninth grade) when proper steps have been taken to ensure that girls understand the terminology used in the instruments [117]. For example, glossaries can be developed for assessment instruments to improve their reliability for use with adolescents. Reliable and valid measures of BID and ED symptoms in preadolescents are also needed. To help fill the gap in terms of measuring body image in children and adolescents, Veron-Guidry and Williamson [117] conducted a study in which they extended the Body Image Assessment (BIA) procedure for adults [118], to children and adolescents, developed norms, and evaluated reliability and validity of the adapted BIA procedure. Their data supported the validity of the BIA-C (children) and BIA-P (preadolescents) procedures and confirmed results from the adult BIA procedure [118]. Generally speaking, instruments used to measure body image and BID in children and adolescents should have sound psychometric properties (e.g., a test–retest reliability of at least 0.70) and be evidence-based. In addition, researchers have supported the use of video distortion methods, which have been successfully implemented with children as young as 5 and 6, as well as custom computer software for measuring body size estimations [113].

### Body Image Comorbidity

The term *body image* is the subjective depiction of physical appearance [119], and is comprised of behavioral, perceptual, cognitive, and affective experiences [120]. Numerous studies have shown a connection between BID and low self-esteem, psychosocial distress, and early-onset depression [80]. In addition, the relationship between body image dissatisfaction and BID has been strongly linked to eating disorders, such as anorexia nervosa and bulimia nervosa [80]. As mentioned earlier, body dissatisfaction has been seen in children as young as 6. As age increases, ideal body size generally becomes progressively thinner [121, 122]. Therefore, body image and BID in children and adolescents command attention in both research and practice [80, 113].

### Body Image Approach

Although the research literature concerning body image in children and adolescents has provided an enlightening knowledge base, Smolak [123], points out that there are salient research questions that still need to be addressed, such as: (a) how body dissatisfaction varies at different ages for different genders and across different ethnic and socioeconomic groups; (b) the need to develop more accurate measurements of body image, particularly in young children and adolescents from various ethnic and socioeconomic groups; and (c) developmental trends in body image development. In addition, more research needs to target body image importance (overestimated views about body shape and weight) and its role in the development of body dissatisfaction, weight loss strategies, and disordered eating [124, 125]. Likewise, there is a need to investigate whether childhood body dissatisfaction, high body mass index, and eating disturbances are risk factors for later development of eating disorders, obesity, or depression [100].

Enhancing health and quality of life for all children and adolescents is a desired outcome of health promotion and prevention programs. Health is not a static condition, but a dynamic interplay among the dimensions of physical, mental, emotional, social, and spiritual health. Consistent with this view of health, the promotion of healthy body image and the prevention of eating disturbances among children and adolescents should focus on the "whole child." For example, undernutrition, which often occurs with eating

disturbances, can have detrimental effects not only on children's physical health (e.g., growth retardation and delayed maturation) [126], but also on their cognitive development [127]. Undernourished students often experience irritability, decreased concentration, nausea, headache, lack of energy, and increased susceptibility to illness [127]. Therefore, it is very difficult for these youth to focus their energy on mastering important developmental tasks, such as succeeding in academics and developing social–emotional skills and a positive identity.

### 10.3.3 Promoting Healthy Model: Strategies and Programs

#### 10.3.3.1 Approaching a Healthy Model

Research about the determinant factors affecting healthy habits during childhood and adolescence focuses in the analysis of the individual, family, sociocultural groups, schools, and peers factors. From this research, the promotion of a healthy model can be approached from different points and by different people and organizations.

As the social learning theory postulates, children imitate adults (family), and it is during adolescence when they reinterpret those social behaviors by comparing them to other social patterns (friends or school). This process lasts throughout life (peers), in spite of its extreme importance during childhood [128]. Therefore, these influences are key to developing and establishing healthy habits during life [129, 130].

#### 10.3.3.2 Strategies and Programs

##### Primary Prevention

Primary prevention focuses on keeping body image and eating disturbances from developing among children and adolescents (i.e., stopping the problem before it starts). Due to the varied tasks that comprise healthy development across childhood and adolescence, prevention programs must not only be age and developmentally appropriate, but must also address the relevant skills and challenges for each stage of development [131]. Because body dissatisfaction and inappropriate weight management practices start early and can lead to more serious body image and eating problems later, prevention should begin at an early age (e.g., elementary school years) [80].

Health education promoting healthy body image along with healthy eating and physical activity is a principal tool for primary prevention. Health education can be implemented both formally and informally in a variety of settings, including homes, schools, health care facilities, and the wider community. Primary prevention is effective when it incorporates strategies that emphasize multifarious components, including, but not limited to:

(a) Development of positive self-esteem and healthy body image.
(b) Development of essential life skills, including social–emotional and effective coping skills.
(c) Provision of experiences that encourage the development of self-efficacy.
(d) Skills training for lifelong balanced nutrition and physical activity.
(e) Opportunities to develop media literacy skills, and learn how to challenge sociocultural myths and attitudes regarding body shape and size.
(f) Positive youth development.

##### Individual

A crucial aspect of prevention that targets the individual is the development of self-esteem, which has emerged as a significant predictor of eating problems in numerous research studies [43, 132, 133].

Branden [133], defines *self-esteem* as possessing two components—self-respect and self-efficacy. Among the hallmarks of self-respect are an individual's assurance of his/her value and basic right to experience a fulfilling life, plus comfort in appropriately asserting thoughts, wants, and needs [133]. A caring adult can foster a child's *self-respect* through increasing the individual's sense of positive uniqueness (i.e., characteristics that set her apart from others in a way that brings honor and a healthy sense of pride). Self-respect does not arise from physical characteristics, but rather from internal attributes. A child also needs to feel significant and a distinct sense

of belonging. This sense of significance and belonging should begin in the family, but also can be fostered through other settings, such as the school and other community-based groups. For example, a school-based prevention program called Everybody's Different [134] focuses on an individual's uniqueness. Initial findings revealed that boys and girls and those students who were overweight or at high risk of developing body image or eating problems experienced significantly improved body image compared to controls [132].

*Self-efficacy* is a person's belief or confidence that she/he can successfully accomplish a task [133]. A sense of competence (power) is foundational for sound mental and emotional health and preventative against the development of negative body image and eating problems. For example, Troop and Treasure [135], found that women who reported experiencing more feelings of helplessness and lower levels of mastery in childhood were more likely to develop EDs in adulthood.

One avenue for instilling a sense of competence in children and adolescents is to help them develop health literacy, which is comprised of these four competencies: effective communication, self-directed learning, critical thinking and problem solving, and responsible, productive citizenship [136]. Health literate individuals are more likely to be resilient and make healthy, life-affirming choices. In terms of preventing body dissatisfaction and eating problems, health literacy competencies are imperative. For example, critical thinking and problem solving are integral skills youth need to effectively counter the barrage of unrealistic, "thin" sociocultural messages they frequently encounter from a variety of sources.

Self-efficacy also can be promoted through goal setting, particularly short-term goals, so that children and youth can take "baby steps" of progress. In turn, they can experience multiple increments of success, thereby developing confidence in their abilities. In addition, repeated opportunities for rehearsal of essential life skills help youth increase a sense of mastery. This sense of competence empowers them to persist in spite of setbacks, and press forward to learn new skills.

Positive social support is another critical element for fostering self-efficacy that can be derived from the family, peers, and caring adults and mentors (e.g., school personnel, health care providers, and other members of the community). These significant relationships provide opportunities for children to cultivate resilience and emulate positive role models and mentors.

### Family

As primary caregivers in the early years of life, parents are considered the main teachers and socializing agents for a child's interaction with the larger environment [137]. Families are a major health and social influence in the lives of children and adolescents; therefore, strong family involvement is essential to health promotion and prevention.

There are numerous strategies that the family can employ to promote positive body image and healthy eating and physical activity habits in children and youth. First of all, positive, nurturing relationships are integral to family health. Parents/caregivers should make it an utmost priority to invest time in building strong, loving, and supportive relationships with their children. In addition, parents/caregivers should be intentional in building self-esteem and fostering resilience in children, starting at a very early age. Parents also should encourage and model essential life skills such as clear communication, problem solving, decision making, and stress management. Even preschool-aged children can be taught about the value of eating healthy foods and being physically active for health and wellness, the importance of respecting different body types, and how to effectively communicate feelings and needs. All children, regardless of their weight and size, should feel that they are unconditionally loved and accepted by the family.

Studies have shown that family involvement plays a key role in a variety of school-based health promotion efforts targeting children and youth, including cardiovascular health promotion [138–140], fruit and vegetable consumption [141], and alcohol prevention [142]. The family also can play a powerful role in preventing negative body image and eating problems in children

and adolescents [98, 100, 137, 143], yet few school-based prevention programs have included a family involvement component [137], such as home-based activities that the family completes together, family fun nights at the school, parent education workshops, and experiential "role play" activities for skill development.

If a family suspects that a child is engaging in restrictive eating and/or other maladaptive behaviors, they should seek help from a qualified professional [For a more comprehensive list of suggestions, see Levine] [144].

### Sociocultural Groups

Progress is lagging in terms of altering cultural norms concerning thinness, body image, and weight management practices. Experts contend that sociocultural changes must occur in order to experience a decline in the growing numbers of children and adolescents with body image problems and disordered eating [77, 88]. Sociocultural influences encompass schools, peers, health care providers, media, and the larger society, all of which interact dynamically in the prevention of body image and eating disturbances.

(a) *Schools*: Findings from the National Longitudinal Study on Adolescent Health revealed that of all the factors that influence adolescent health-risk behavior, the most critical are the family and schools [145]. Schools are logical venues for primary prevention programs due to the sheer number of children they serve, and upper elementary appears to a viable age with which to target interventions. Enhanced knowledge, critical-thinking and problem-solving skills, and realistic perceptions may offset unhealthy influences concerning body shape, weight, and eating practices that are likely to be well established by the middle-school years [146].

Of the 42 school-based prevention programs that Levine and Smolak [77], located and reviewed, 10 had been developed for elementary-aged children. Results of these programs varied, ranging from those revealing minimal change to those demonstrating increases in knowledge and body satisfaction. However, in some cases, at least for a significant minority of the girls, program content may have increased or created negative attitudes about their bodies. An upper-elementary prevention curriculum that appears to hold promise is *Healthy Body Image: Teaching Kids to Eat and Love Their Bodies Too!* [147]. An uncontrolled pilot study with 222 boys and girls in grades four through six revealed positive changes in knowledge of program content, acceptance of diverse body weights and shapes, positive body esteem, and rejection of the thin ideal [148]. Later, a controlled study with 415 boys and girls measured pre- and post-test changes related to: (a) body image; (b) knowledge about the biology of size, shape, and restricted hunger or dieting; (c) body size prejudice; (d) media awareness; (e) self-image; and (f) lifestyle behavior. Students who completed the curriculum showed significant or noteworthy improvements in most areas compared to a control group not exposed to the curriculum [118]. Another prevention program entitled *Eating Smart, Eating for Me* is designed for fourth and fifth grade students and centers around five goals encompassing healthy body image, healthy eating and physical activity, and media literacy. A controlled evaluation of this ten-lesson curriculum revealed an increase in student knowledge about nutrition and decreased negative attitudes toward overweight people. However, attitudes like body esteem and behaviors did not significantly change [149]. A 2-year follow-up study revealed that when, compared to new controls, adolescents who participated in the prevention program 2 years earlier were more knowledgeable, engaged in fewer unhealthy weight management behaviors, and had higher body esteem. The latter difference was particularly significant for girls [150].

In terms of evaluated school-based programs that have been developed for middle schools and high schools, most have had only a modest effect on knowledge, with little impact on attitudes, behavior, or weight change. This partly could be due to the

short-term nature of these interventions. Programs may be more effective if they: are designed for the stages of change for the participants; are based on the lived experiences of the participants; include relational components; coincide with developmental issues; and incorporate active, experiential, and peer-mediated formats [68, 77, 143, 151].

There seems to be some disagreement as to whether universal programs or programs targeting high-risk groups have the best likelihood for achieving successful outcomes. Research has demonstrated that it is feasible to deliver different interventions by providing a universal prevention program to all students, while targeting those at risk [152]. In either case, school-based prevention programs should be a part of an overall coordinated school health program (CSHP) that promotes the health and well-being of students through eight interrelated components—health education; healthy school environment; school health services; school food services; counseling, psychological, and social services; physical education; staff health promotion; and family and community involvement [20]. For example, a comprehensive program for preventing body image and eating disturbances can include: (a) evidence-based curricula for preventing body image and eating problems; (b) a school environment that cultivates caring, supportive, relationships; (c) school food services that reinforce healthy eating; (d) counseling services for all students, particularly those at high risk, that includes peer support groups and referral networks; (e) daily, quality physical education that emphasizes the importance of staying physically active for health and vitality; (f) ongoing prevention trainings for staff; and (g) outreach activities that form partnerships among school personnel, families, and the broader community. Furthermore, individuals who are responsible for implementing prevention programs should be stakeholders in the process. For example, engaging teachers is paramount to implementing a successful prevention curriculum [153]. This argument can be extended to include all of those involved in a CSHP—administrators, teachers, school nurses, school counselors/psychologists, school social workers, school food service workers, and other school staff, students, families, and the larger community. When each of these individuals embraces the importance of health promotion and takes ownership of prevention efforts that can change the school milieu, there is a strong possibility for a reduction in the incidence of body image and eating disturbances among youth.

(b) *Peers*: Peer groups often are perceived as risk factors for harmful health behaviors because of their role as strong sociocultural influences in the lives of children and youth. However, peer groups that model pro-social values actually can serve as crucial protective factors. A qualitative study of high school girls found that positive peer influences (e.g., being dissuaded from dieting or purging) protected girls from excessive weight concerns that can lead to eating disturbances [69]. Relationships with peers who value health and wellness and practice life-affirming behaviors are powerful and absolutely vital to the prevention of body image and eating disturbances in children and adolescents. Healthy peers do not place overemphasis on body weight and size, and value themselves and others for who they are on the inside (e.g., their spirit, character, talents, and gifts). They also can challenge each other's unrealistic thoughts and self-defeating actions concerning body weight and size. In addition, peer mentoring programs and peer support groups can make significant contributions to the prevention process. Peer inclusion that fosters a positive sense of belonging is a fundamental need of children and adolescents; and young people should have multiple opportunities to cultivate positive, healthy relationships with peers who model pro-social values and health-enhancing behaviors. These relationships can be formed with peers at school, during after-school programs and extracurricular activities, and

in faith-based youth groups and other community organizations.

(c) *Health Care Providers*: Due to the potentially irreversible effects of EDs on all aspects of child and adolescent health, the high mortality rate of EDs, and evidence that early treatment is vital for improved outcomes, the role of health care professionals in recognizing and preventing EDs is paramount [84, 88]. Many families look to health care providers as primary sources of credible health information, which places health care professionals in a powerful leadership position to make a significant difference in health promotion and prevention efforts. There are a number of ways that health care providers can proactively reduce the risk of body image and eating problems among children and adolescents. For example, office visits provide a ripe opportunity for communicating important information to children and their parents, including strategies for healthy eating and physical activity to enhance health and quality of life. It is important to note that many families are seeking the advice and care of physicians and/or dieticians regarding childhood obesity. Health professionals must be cognizant of the delicate balance when counseling children and their families about obesity and its risks. It is essential not to overemphasize dieting and to help children and adolescents build self-esteem, while addressing weight issues [88]. Health care professionals also can help both parents and children understand what to expect during various developmental stages, particularly puberty. Additionally, health care providers can contribute to primary prevention through school- and community-based interventions that deliver screenings and education, and through participating in advocacy efforts aimed at changing cultural norms that children and adolescents experience [88]. The *BodyWise* [154], information packet designed for health care providers offers useful tips and strategies to facilitate the prevention process.

(d) *Media*: Children and adolescents must be armed with knowledge and life skills to combat the extreme focus on appearance that pervades our society. A very influential medium for communicating society's thin ideal are the media, which have assumed a ubiquitous role in homes across America. According to a Kaiser Family Foundation study, "Young people today live "media-saturated" lives, spending an average of nearly 6 ½ (6:21) hours a day with media" [155]. Media literacy education is a valuable weapon against media messages that communicate unrealistic body images. Media literacy has been defined as "the ability to access, analyze, evaluate, and produce communication in a variety of forms" [156]. Media literacy education can help girls enhance their sense of self-acceptance and empowerment regarding media portrayal of female bodies, and teach them how to effectively counter messages that promote unrealistic body images and unhealthy eating [157, 158]. Prevention programs like GO GIRLS!™ [159], can build self-esteem, enhance body image, increase resilience, and empower participants to be strong media advocates. Most (if not all) media literacy education for the prevention of eating disorders has targeted high school and college-aged females, which leaves an alarming gap in terms of reaching prepubescent and younger girls. Even young children are highly impressionable to negative health messages, including ones about body weight and size. Therefore, media literacy education should target different age groups of children and adolescents, and hold relevance in terms of the types of media each group uses.

The proliferation of Internet Web sites that advocate or encourage youth to practice disordered eating warrants additional attention [84]. These pro-anorexia (pro-ana) and pro-bulimia (pro-mia) Web sites promote disordered eating as a lifestyle, and offer dangerous and negative reinforcement for youth who are practicing disordered eating or thinking about doing so. Disturbingly, these sites far outnumber that of professional or recovery sites [160], which calls for the

**Table 10.1** The model for healthy body image©

| Conceptual building blocks | Foundation | Desired outcome | Goal |
|---|---|---|---|
| Developmental change is inevitable<br>Normal changes of puberty include weight gain and temporary out-of-proportion growth; fat does not by itself define "overweight"<br>Genetics and other internal weight regulators strictly limit the degree to which shape, weight, and Body Mass Index can be manipulated through healthy means<br>Restricted or restrained hunger (dieting) results in predictable consequences that are *counterproductive* to weight loss and interfere with normal hunger regulation | Recognize and respect basic biology; understand what *cannot* be controlled about size, shape, and hunger | Accept the innate body: "This is the body I was born to have" | Healthy body image |
| Balance attention to *many* aspects of identity. Looks are only one part<br>Consistently satisfy hunger with *enough* varied, wholesome food in a stable, predictable manner<br>Limit sedentary choices to promote a physically active lifestyle at all ages<br>Choose role models that reflect a realistic standard | Emphasize what *can* be influenced or chosen | Enjoy eating for health, energy, and hunger satisfaction<br>Create a physically active lifestyle for fitness, endurance, fun, relaxation, and stress relief | Prevention of unhealthy and disordered eating |
| Promote historical perspective on today's cultural attitudes related to body image<br>Teach critical thinking about media messages that influence body image<br>Support others in resisting unhealthy norms about weight, dieting, low nutrient food choices, eating for entertainment, and sedentary entertainment | Develop social and cultural resiliency | Develop autonomy, self-esteem, confidence, and the ability for critical thinking | |

Developed by Kathy J. Kater, LICSW ©1998—www.BodyImageHealth.org

need to increase awareness among parents, teachers, health care providers, and other caring adults and mentors.

(e) *Society*: In order for prevention of body image and eating disturbances among children and adolescents to be successful, there must be inside-out changes at the grassroots level that lead to broader sociocultural changes. These changes should include how females are portrayed in the media; expectations regarding gender roles; acceptance of a wide range of body weights and shapes; and increased opportunities for healthy eating and physical activity, particularly in low-income neighborhoods [161]. In addition, Kater [162], addresses four "toxic" sociocultural myths that need to be overcome in order to help children and adolescents develop healthy body image: valuing image over substance (e.g., marketing extremely thin models as if they were normal); denial of biological diversity of body sizes and shapes; denial of the harmful effects of restrictive eating for weight loss purposes; and discounting the value of eating well and being physically fit. The Model for Healthy Body Image (MHBI) [163] was designed to combat these myths by helping children learn to value health and wellness, and resist sociocultural pressures that promote negative body image and unhealthy behaviors (Table 10.1). This model is addressed in an elementary prevention curriculum [147], so that children can develop a healthy body image before they hit the transitional period of the middle school years.

Other credible prevention strategies and programs that address various sociocultural influences can be located online. In addition to BodyWise [154], the National Eating Disorders Association (www.edap.org) provides an array of prevention information and tips.

#### 10.3.3.3 Positive Youth Development

Numerous researchers and practitioners have recognized the importance of positive youth development in promoting health-enhancing behaviors and preventing health-risk behaviors among youth. In a comprehensive study of positive youth development programs in the USA, Catalano and colleagues [164], found that various youth development approaches can result in positive youth behavior outcomes (e.g., enhanced relational skills and quality of peer and adult relationships, improved problem solving and self-efficacy) and prevention of youth risk behaviors (e.g., drug and alcohol use, smoking, school misbehavior, and risky sexual behavior). Data from longitudinal studies over the past 30 years have identified similar risk and protective factors in individuals, peer groups, families, schools, and neighborhoods that can accurately predict diverse youth problem behaviors [165–170]. However, exposure to ever-increasing numbers of protective factors has been found to prevent problem behaviors in spite of the presence of multiple risks [171, 172].

As a forerunner in the research and implementation of positive youth development, the Social Development Research Group emphasizes the importance of a comprehensive approach to preventing youth problem behaviors by addressing both risk and protective factors. The framework for guiding the positive youth development process is Social Development Strategy (SDS), which is part of a comprehensive model of behavioral development—The Social Development Model [167]. SDS organizes protective factors into a framework for promoting positive youth development, despite the presence of risk. Additionally, SDS focuses on the outcome of health-enhancing behaviors through exposing children and adolescents to two critical protective factors: (1) pro-social bonding to family, school, and peers and (2) healthy beliefs and clear standards for behavioral norms. The mechanisms that help create these protective factors are: opportunities for meaningful participation in productive pro-social roles, life skills to facilitate participation in these roles, consistent systems of recognition and positive reinforcement for pro-social involvement, and individual characteristics.

Positive youth development programs usually strive to achieve one or more of the following objectives: foster resilience; promote healthy bonding; promote social, emotional, cognitive, behavioral, and moral competencies; foster self-determination; foster self-efficacy; foster clear and positive identity; cultivate spirituality; foster belief in the future; provide recognition for positive behavior; provide opportunities for pro-social involvement; and foster pro-social norms [164]. Although positive youth development programs can target youth in one particular setting, the majority of successful programs capitalize on the resources of multiple settings (family, schools, community) [164].

Positive youth development that is guided by SDS can be considered a viable framework for promoting healthy body image and preventing eating disturbances among children and adolescents. In order to develop positive body image and healthy eating behaviors, all children and adolescents must be enveloped in environments that consistently provide the protective factors depicted in Fig. 10.1. Youth who live with multiple risk factors for developing body image and eating problems can benefit from all of these steps and need to be even more fully immersed in protective environments. For positive youth development to be successful, families and communities must identify healthy beliefs and clear standards for healthy body image and eating behaviors. Then, they must foster those healthy beliefs, and communicate the standards in multiple arenas of a young person's life—at home and school, in peer groups, within youth-serving and faith-based organizations, and in all segments of the larger community. Children and adolescents must have numerous opportunities to cultivate strong, relational bonds with those who embrace and model positive body image along with other health-promoting behaviors, such as healthy eating and PA. These protective bonds can be created through providing youth meaningful opportunities for participation/involvement in a variety of pro-social activities (e.g., advocacy efforts aimed at changing cultural norms promoting the thin ideal). In addition, children and adolescents need repetitive opportunities to develop crucial cognitive, social-emotional, and

behavioral skills to help them successfully accomplish these activities, as well as important developmental tasks. They also need to be consistently recognized and affirmed for their prosocial involvement at all levels. Finally, recognizing and nurturing individual strengths can enhance children's resilience and provide a foundation for external protective factors to optimally function.

## 10.4 Contemporary Understanding of the Issues

A number of factors, including peer groups, schools, family, media, and society, greatly affect children's and adolescents' body-image perceptions. In a time when the media have such a strong influence on how we feel we should look, it is essential that we treat these issues seriously, as the consequences could be deadly. The best defenses we have against eating disorders and body-image disturbances are early prevention, through family support and positive influences, and, for those already affected by these disorders, timely diagnosis and treatment. It is crucial, now more than ever, that a child develops a positive self-image and self-esteem at a young age and that his or her sense of self-worth is fostered into adolescence and young adulthood.

## 10.5 Future Directions

There are several prevention questions that deserve further study. For example: Which protective factors should be targeted and enhanced to reduce the incidence of body image and eating disturbances among children and adolescents from different age and ethnic groups? Which interventions are more effective—universal or targeted ones? How long should interventions last, and what are the most cost-effective approaches? [143] How can high-risk youth be reached, and what interventions are most effective with this group? [84, 143] Future studies also need to directly compare promising prevention programs in randomized trials, particularly in ecologically valid settings where large numbers of children and adolescents can be reached (e.g., schools) [173]. There is an additional need to further explore the pathogenesis of early-onset eating disorders, and enhance the current diagnostic system to address the "unique spectrum of early-onset eating disorders and the development of effective treatments for adolescent eating disorders" [84]. Finally, research is needed to determine whether positive youth development programs that employ SDS can produce positive outcomes in terms of decreasing body image and eating disturbances among children and youth.

## 10.6 Concluding Remarks

Body image and eating disturbances are highly complex and wield a harsh blow to our nation in terms of health care costs, diminished quality of life, and tragic loss of life. Youth, parents, school personnel, health care providers, and entire communities can be effective change agents for the prevention of body image and eating disturbances among children and adolescents. Health promotion and prevention efforts need to be multifaceted and encompass the whole child by addressing physical, mental, emotional, social, and spiritual health. Furthermore, these efforts should target interests, skills, and challenges that are distinctive for particular stages of development [137]. No individual should underestimate his or her ability to make a positive difference in the prevention of body image and eating disturbances in children and adolescents. Even small changes have the capacity to produce a powerful ripple effect that can transform society. When individuals, families, and communities unite and engage in proactive endeavors to promote health and quality of life for children and adolescents, they create a legacy of tremendous impact and enduring value [174].

## References

1. Jimenez Pavon D, Kelly J, Reilly J. Associations between objectively measured habitual physical activity and adiposity in children and adolescents: systematic review. Int J Pediatr Obes. 2010;5(1):3–18.
2. LaMonte MJ, Blair SN. Physical activity, cardiorespiratory fitness, and adiposity: contributions to

disease risk. Curr Opin Clin Nutr Metab Care. 2006;9(5):540–6.
3. Tremblay MS, Colley RC, Saunders TJ, Healy GN, Owen N. Physiological and health implications of a sedentary lifestyle. Appl Physiol Nutr Metab. 2010;35(6):725–40.
4. Biddle SJ, Pearson N, Ross GM, Braithwaite R. Tracking of sedentary behaviours of young people: a systematic review. Prev Med. 2010;51(5):345–51.
5. Janz KF, Dawson JD, Mahoney LT. Tracking physical fitness and physical activity from childhood to adolescence: the muscatine study. Med Sci Sports Exerc. 2000;32(7):1250–7.
6. Westenhoefer J. Establishing dietary habits during childhood for long-term weight control. Ann Nutr Metab. 2002;46(1):18–23.
7. Moreno L, González-Gross M, Kersting M, et al. Assessing, understanding and modifying nutritional status, eating habits and physical activity in European adolescents: the HELENA (Healthy Lifestyle in Europe by Nutrition in Adolescence) Study. Public Health Nutr. 2008;11(03):288–99.
8. Nicklas T, Johnson R. Position of the American Dietetic Association: dietary guidance for healthy children ages 2 to 11 years. J Am Diet Assoc. 2004;104(4):660.
9. Cooke L. The importance of exposure for healthy eating in childhood: a review. J Hum Nutr Diet. 2007;20(4):294–301.
10. Mota J, Queirós P. Children's behavior. Physical activity regarding parents' perception vs. children's activity. Int Rev Sociol Sport. 1996;31(2):173–83.
11. Fredricks JA, Eccles JS. Family Socialization, Gender, and Sport Motivation and Involvement. J Sport Exerc Psychol. 2005;27:3–31.
12. Fredricks JA, Eccles JS. Parental influences on youth involvement in sports. In: Weiss, Maureen R. Developmental sport and exercise psychology: a lifespan perspective. (pp. 145–164). Morgantown, WV, US: Fitness Information Technology; 2004. pp. xvii, 596.
13. Woolger C, Power TG. Parent and sport socialization: views from the achievement literature. J Sport Behav. 1993;16(3):171–85.
14. Smith RA, Biddle SJ. Psychological factors in the promotion of physical activity. In: Biddle SJH, editor. European perspectives on exercise and sport psychology. Champaign, IL: Human Kinetics; 1995. p. 85–108.
15. Aznar S. Environmental and significant others' influences on children's physical activity behaviour. Bristol: England, University of Bristol; 1998.
16. Sallis JF, Prochaska JJ, Taylor WC. A review of correlates of physical activity of children and adolescents. Med Sci Sports Exerc. 2000;32(5):963–75.
17. Crocker P, Bailey DA, Faulkner RA, Kowalski KC, McGrath R. Measuring general levels of physical activity: preliminary evidence for the Physical Activity Questionnaire for Older Children. Med Sci Sports Exerc. 1997;29(10):1344.
18. Waxman A. WHO global strategy on diet, physical activity and health. Food Nutr Bull. 2004;25(3):292–302.
19. Brodersen NH, Steptoe A, Boniface DR, Wardle J. Trends in physical activity and sedentary behaviour in adolescence: ethnic and socioeconomic differences. Br J Sports Med. 2007;41(3):140–4.
20. CDC. Centers for Disease Control and Prevention. Healthy youth! Coordinated school health program. 2013. http://www.cdc.gov/healthyyouth/yrbs/index.htm. Accessed 12 Apr 2013.
21. Engel GL. The need for a new medical model: a challenge for biomedicine. Psychodyn Psychiatry. 2012;40(3):377–96.
22. Santrock JW. Life-span development. Princeton, NJ: Recording for the Blind & Dyslexic; 2007.
23. WHO. Preamble to the Constitution of the World Health Organization as adopted by the International Health Conference, New York, 19–22 June, 1946; signed on 22 July 1946 by the representatives of 61 States (Official Records of the World Health Organization, no. 2, p. 100) and entered into force on 7 April 1948. Retrieved Mar 1948;16.
24. Becker MH, Maiman LA. Sociobehavioral determinants of compliance with health and medical care recommendations. Med Care. 1975;13:10–24.
25. Ajzen I, Madden TJ. Prediction of goal-directed behavior: attitudes, intentions, and perceived behavioral control. J Exp Soc Psychol. 1986;22(5):453–74.
26. Bandura A. Social foundations of thought and action. In: Marks DF, editor. The health psychology reader. London: Sage; 2002. p. 94–106.
27. Prochaska JO, DiClemente CC. Transtheoretical therapy: toward a more integrative model of change. Psychother Theory Res Pract. 1982;19(3):276.
28. Montil Jiménez M. Determinantes de la conducta de actividad física en población infantil, Tesis(Doctoral), Ciencias; 2004.
29. CDC. Centers for Disease Control and Prevention. Youth Risk Behavior Surveillance—United States, 2009. Morbidity and Mortality Weekly Report 2009; http://www.cdc.gov/mmwr/pdf/ss/ss5905.pdf. Accessed 12 Apr 2012.
30. Nolen-Hoeksema S. Abnormal psychology. 5th ed. New York, NY: McGraw-Hill; 2011.
31. Kaye W. Neurobiology of anorexia and bulimia nervosa. Physiol Behav. 2008;94(1):121–35.
32. Strober M. Family-genetic studies of eating disorders. J Clin Psychiat. 1991;52(Suppl):9–12.
33. Graber JA, Brooks-Gunn J, Paikoff RL, Warren MP. Prediction of eating problems: an 8-year study of adolescent girls. Dev Psychol. 1994;30(6):823.
34. Swarr AE, Richards MH. Longitudinal effects of adolescent girls' pubertal development, perceptions of pubertal timing, and parental relations on eating problems. Dev Psychol. 1996;32(4):636.
35. Williams JM, Currie C. Self-esteem and physical development in early adolescence: pubertal timing and body image. J Early Adolesc. 2000;20(2):129–49.

36. Attie I, Brooks-Gunn J. Development of eating problems in adolescent girls: a longitudinal study. Dev Psychol. 1989;25(1):70.
37. Cattarin JA, Thompson JK. A three-year longitudinal study of body image, eating disturbance, and general psychological functioning in adolescent females. Eat Disord. 1994;2(2):114–25.
38. Leon GR, Fulkerson JA, Perry CL, Keel PK, Klump KL. Three to four year prospective evaluation of personality and behavioral risk factors for later disordered eating in adolescent girls and boys. J Youth Adolesc. 1999;28(2):181–96.
39. Stice E, Killen JD, Hayward C, Taylor CB. Age of onset for binge eating and purging during late adolescence: a 4-year survival analysis. J Abnorm Psychol. 1998;107(4):671.
40. Tobin-Richards MH, Boxer A, Petersen AC. The psychological significance of pubertal change: sex differences in perceptions of self during early adolescence. In: Brooks-Gunn J, Petersen AC, editors. Girls at puberty: biological and psychosocial perspectives. New York, NY: Plenum; 1983. p. 127–54.
41. Thomas K, Ricciardelli LA, Williams RJ. Gender traits and self-concept as indicators of problem eating and body dissatisfaction among children. Sex Roles. 2000;43(7):441–58.
42. Tiggemann M. The impact of adolescent girls' life concerns and leisure activities on body dissatisfaction, disordered eating, and self-esteem. J Genet Psychol. 2001;162(2):133–42.
43. Shisslak C, Crago M. Risk and protective factors in the development of eating disorders. In: Kevin Thompson J, Smolak L, editors. Body image, eating disorders, and obesity in youth. Washington, DC: American Psychological Association; 2001. p. 103–25.
44. Steiner H, Kwan W, Shaffer TG, et al. Risk and protective factors for juvenile eating disorders. Eur Child Adolesc Psychiatry. 2003;12 Suppl 1:I38–6.
45. Coughlin JW, Heinberg LJ, Marinilli A, Guarda AS. Body image dissatisfaction in children: prevalence and parental influence. Healthy Weight J. 2003;17(4):56.
46. Keel PK, Fulkerson JA, Leon GR. Disordered eating precursors in pre-and early adolescent girls and boys. J Youth Adolesc. 1997;26(2):203–16.
47. Fairburn CG, Welch SL, Doll HA, Davies BA, O'Connor ME. Risk factors for bulimia nervosa: a community-based case-control study. Arch Gen Psychiatry. 1997;54(6):509.
48. APA. American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-IV-TR. Washington, DC: American Psychiatric Publishing, Inc; 2000.
49. Agras S, Hammer L, McNicholas F. A prospective study of the influence of eating-disordered mothers on their children. Int J Eat Disord. 1999;25(3): 253–62.
50. Park RJ, Senior R, Stein A. The offspring of mothers with eating disorders. Eur Child Adolesc Psychiatry. 2003;12 Suppl 1:I110–9.
51. Byely L, Archibald AB, Graber J, Brooks-Gunn J. A prospective study of familial and social influences on girls' body image and dieting. Int J Eat Disord. 2000;28(2):155–64.
52. Humphrey L. Family relationships. In: Halmi K, editor. Psychobiology and treatment of anorexia nervosa and bulimia nervosa. Washington, DC: American Psychiatric Press; 1994. p. 263–82.
53. Minuchin S, Rosman BL, Baker L. Psychosomatic families: anorexia nervosa in context. Cambridge: Harvard University Press; 1978.
54. Neumark-Sztainer D, Story M, Hannan P, Beuhring T, Resnick MD. Disordered eating among adolescents: association with sexual/physical abuse and other familial psychosocial factors. Int J Eat Disord. 2000;28:10.
55. Sands ER, Wardle J. Internalization of ideal body shapes in 9–12-year-old girls. Int J Eat Disord. 2003;33(2):193–204.
56. Connors M. Relationship of sexual abuse to body image and eating problems. In: Thompson JK, Smolak L, editors. Body image, eating disorders, and obesity in youth. Washington, DC: American Psychological Association; 2001. p. 149–70.
57. Wonderlich S, Crosby R, Mitchell J, et al. Pathways mediating sexual abuse and eating disturbance in children. Int J Eat Disord. 2001;29(3):270–9.
58. Steinberg A, Phares V. Family functioning, body image, and eating disturbances. In: Thompson JK, Smolak L, editors. Body image, eating disorders, and obesity in youth. Washington, DC: American Psychological Association; 2001. p. 127–47.
59. Wiseman CV, Gray JJ, Mosimann JE, Ahrens AH. Cultural expectations of thinness in women: an update. Int J Eat Disord. 2006;11(1):85–9.
60. Gowers SG, Shore A. Development of weight and shape concerns in the aetiology of eating disorders. Br J Psychiatry. 2001;179(3):236–42.
61. Groesz LM, Levine MP, Murnen SK. The effect of experimental presentation of thin media images on body satisfaction: a meta-analytic review. Int J Eat Disord. 2002;31(1):1–16.
62. Lieberman M, Gauvin L, Bukowski WM, White DR. Interpersonal influence and disordered eating behaviors in adolescent girls: the role of peer modeling, social reinforcement, and body-related teasing. Eat Behav. 2001;2(3):215–36.
63. Horowitz A. A report on the NIH consensus development conference on diagnosis and management of dental caries throughout life. J Dent Res. 2004;83 suppl 1:C15–7.
64. Rodin J, Streigel-Moore R, Silberstein L. Vulnerability and resilience in the age of eating disorders: risk and protective factors for bulimia nervosa. In: Rolf J, Masten A, Cicchetti D, Neuchterlein K, Weintraub S, editors. Risk and protective factors in the development of psychopathology. Cambridge: Cambridge University; 1990. p. 361–83.
65. Connors M. Developmental vulnerabilities for eating disorders. In: Smolak LLM, Striegel-Moore R, editors. The developmental psychopathology of

eating disorders. Mahwah, NJ: Erlbaum; 1996. p. 285–310.
66. Cole TJ, Flegal KM, Nicholls D, Jackson AA. Body mass index cut offs to define thinness in children and adolescents: international survey. BMJ. 2007; 335(7612):194.
67. Striegel-Moore R, Cachelin FM. Body image concerns and disordered eating in adolescent girls: risk and protective factors. In: Johnson N, Roberts M, editors. Beyond appearance: a new look at adolescent girls. Washington, DC: American Psychological Association; 1999. p. 85–108.
68. Littleton HL, Ollendick T. Negative body image and disordered eating behavior in children and adolescents: what places youth at risk and how can these problems be prevented? Clin Child Fam Psychol Rev. 2003;6(1):51–66.
69. Wertheim EH, Paxton SJ, Schutz HK, Muir SL. Why do adolescent girls watch their weight? An interview study examining sociocultural pressures to be thin. J Psychosom Res. 1997;42(4):345–55.
70. Telama R, Yang X, Viikari J, Välimäki I, Wanne O, Raitakari O. Physical activity from childhood to adulthood: a 21-year tracking study. Am J Prev Med. 2005;28(3):267–73.
71. Twisk J, Kemper H, Van Mechelen W. Prediction of cardiovascular disease risk factors later in life by physical activity and physical fitness in youth: general comments and conclusions. Int J Sports Med. 2002;23(S1):44–50.
72. Stathopoulou G, Powers MB, Berry AC, Smits JAJ, Otto MW. Exercise interventions for mental health: a quantitative and qualitative review. Clin Psychol (New York). 2006;13(2):179–93.
73. Chandy JM, Harris L, Blum RW, Resnick MD. Female adolescents of alcohol misusers: disordered eating features. Int J Eat Disord. 1995;17(3): 283–9.
74. Berndt T, Hestenes S. The developmental course of social support: family and peers. In: Smolak L, Levine MP, Striegel-Moore R, editors. The developmental psychopathology of eating disorders. Mahwah, NJ: Erlbaum; 1996.
75. Smolak L, Murnen SK, Ruble AE. Female athletes and eating problems: a meta-analysis. Int J Eat Disord. 2000;27(4):371–80.
76. Gerner B, Wilson PH. The relationship between friendship factors and adolescent girls' body image concern, body dissatisfaction, and restrained eating. Int J Eat Disord. 2005;37(4):313–20.
77. Thompson JKSL, Smolak JK. Introduction: body image, eating disorders, and obesity in youth—the future is now. In: Thompson J, Smolak L, editors. Body image, eating disorders, and obesity in youth. 1st ed. Washington, DC: American Psychological Association; 2001. p. 1–18.
78. Shisslak CM, Crago M, Estes LS. The spectrum of eating disturbances. Int J Eat Disord. 2006; 18(3):209–19.
79. Stice E, Killen JD, Hayward C, Taylor CB. Support for the continuity hypothesis of bulimic pathology. J Consult Clin Psychol. 1998;66(5):784.
80. Smolak L, Levine MP. Body Image in Children. In: Thompson JK, Smolak L, editors. Body image, eating disorders, and obesity in youth. 1st ed. Washington, DC: American Psychological Association; 2001. p. 41–66.
81. Croll J, Neumark-Sztainer D, Story M, Ireland M. Prevalence and risk and protective factors related to disordered eating behaviors among adolescents: relationship to gender and ethnicity. J Adolesc Health. 2002;31(2):166–75.
82. Fisher M, Golden NH, Katzman DK, Kreipe RE. Eating disorders in adolescents: a background paper. J Adolesc Health. 1995;16(6):420–37.
83. Jones JM, Rodin G. Disordered eating attitudes among Canadian teenagers. Can Med Assoc J. 2002; 166(6):707–8.
84. Golden NH, Katzman DK, Kreipe RE, et al. Eating disorders in adolescents: position paper of the Society for Adolescent Medicine. J Adolesc Health. 2003;33(6):496–503.
85. Smink FR, van Hoeken D, Hoek HW. Epidemiology of eating disorders: incidence, prevalence and mortality rates. Curr Psychiatry Rep. 2012;14(4):406–14.
86. Sullivan PF. Mortality in anorexia nervosa. Am J Psychiatry. 1995;152(7):1073–4.
87. Johnson JG, Cohen P, Kasen S, Brook JS. Childhood adversities associated with risk for eating disorders or weight problems during adolescence or early adulthood. Am J Psychiatry. 2002;159(3):394–400.
88. Rosen DS. Identification and management of eating disorders in children and adolescents. Pediatrics. 2010;126(6):1240–53.
89. Yager J, Devlin M, Halmi K, Herzog D, Mitchell J, Powers P. American Psychiatric Association practice guideline for the treatment of patients with eating disorders. Washington, DC: American Psychiatric Association; 2006.
90. Le Grange D, Swanson SA, Crow SJ, Merikangas KR. Eating disorder not otherwise specified presentation in the US population. Int J Eat Disord. 2012;45(5):711–8.
91. Cash T. The psychology of physical appearance: aesthetics, attributes, and images. In: Cash T, Pruzinsky T, editors. Body images: development, deviance, and change. New York: Guilford; 1990. p. 51–79.
92. Powers PS. Initial assessment and early treatment options for anorexia nervosa and bulimia nervosa. Psychiatr Clin North Am. 1996;19(4):639.
93. Jordan J, Joyce PR, Carter FA, et al. Anxiety and psychoactive substance use disorder comorbidity in anorexia nervosa or depression. Int J Eat Disord. 2003;34(2):211–9.
94. Fishy S. Associated mental health conditions and addictions. http://www.something-fishy.org/isf/mentalhealth.php. Accessed 19 Apr 2013.

95. Austin SB, Ziyadeh NJ, Forman S, Prokop LA, Keliher A, Jacobs D. Peer reviewed: screening high school students for eating disorders: results of a national initiative. Prev Chronic Dis. 2008;5(4):A114.
96. CDC. Centers for Disease Control and Prevention. Childhood obesity facts: obesity rates among all children in the United States. 2009. http://www.cdc.gov/obesity/data/childhood.html. Accessed 12 April 2012.
97. Davison K, Markey C, Birch L. Etiology of body dissatisfaction and weight concerns among 5-year-old girls. Appetite. 2000;35(2):143–51.
98. Smolak L, Levine MP. Toward an empirical basis for primary prevention of eating problems with elementary school children. Eat Disord. 1994;2(4):293–307.
99. Grabe S, Hyde JS. Ethnicity and body dissatisfaction among women in the United States: a meta-analysis. Psychol Bull. 2006;132(4):622.
100. Schur EA, Sanders M, Steiner H. Body dissatisfaction and dieting in young children. Int J Eat Disord. 1999;27(1):74–82.
101. Ferron C. Body image in adolescence: cross-cultural research—results of the preliminary phase of a quantitative survey. Adolescence. 1997;32(127):735–45.
102. Gustafson-Larson AM, Terry RD. Weight-related behaviors and concerns of fourth-grade children. J Am Diet Assoc. 1992;92(7):818.
103. Maloney MJ, McGuire J, Daniels SR, Specker B. Dieting behavior and eating attitudes in children. Pediatrics. 1989;84(3):482–9.
104. Pierce JW, Wardle J. Cause and effect beliefs and self-esteem of overweight children. J Child Psychol Psychiatry. 1997;38(6):645–50.
105. Neumark-Sztainer D. "I'm, Like, SO Fat!": helping your teen make healthy choices about eating and exercise in a weight-obsessed world. New York: Guilford Press; 2005.
106. Neumark-Sztainer D, Story M, Falkner NH, Beuhring T, Resnick MD. Sociodemographic and personal characteristics of adolescents engaged in weight loss and weight/muscle gain behaviors: who is doing what? Prev Med. 1999;28(1):40–50.
107. Joiner GW, Kashubeck S. Acculturation, body image, self-esteem, and eating-disorder symptomatology in adolescent Mexican American women. Psychol Women Quart. 1996;20(3):419–35.
108. Gardner RM, Sorter RG, Friedman BN. Developmental changes in children's body images. J Soc Behav Pers. 1997;12(4):1019–36.
109. Mellin LM, Irwin Jr CE, Scully S. Prevalence of disordered eating in girls: a survey of middle-class children. J Am Diet Assoc. 1992;92:851–3.
110. Killen JD, Taylor CB, Hayward C, et al. Pursuit of thinness and onset of eating disorder symptoms in a community sample of adolescent girls: a three-year prospective analysis. Int J Eat Disord. 1994;16(3):227–38.
111. Kaplan SL, Busner J, Pollack S. Perceived weight, actual weight, and depressive symptoms in a general adolescent sample. Int J Eat Disord. 1988;7(1):107–13.
112. McCabe M, Marwit SJ. Depressive symptomatology, perceptions of attractiveness, and body image in children. J Child Psychol Psychiatry. 1993;34(7):1117–24.
113. Gardner R. Body image, eating disorders, and obesity in youth: assessment, prevention and treatment. Assessment of body image disturbance in children and adolescents. In: Thompson JK, Smolak L, editors. Washington, DC: American Psychological Association; 2001. p. 193–213.
114. Garner DM, Garfinkel PE. Body image in anorexia nervosa: measurement, theory and clinical implications. Int J Psychiatry Med. 1981;11(3):263–84.
115. Keeton WP, Cash TF, Brown TA. Body image or body images?: comparative, multidimensional assessment among college students. J Pers Assess. 1990;54(1–2):213–30.
116. Banasiak SJ, Wertheim EH, Koerner J, Voudouris NJ. Test-retest reliability and internal consistency of a variety of measures of dietary restraint and body concerns in a sample of adolescent girls. Int J Eat Disord. 2001;29(1):85–9.
117. Veron-Guidry S, Williamson DA. Development of a body image assessment procedure for children and preadolescents. Int J Eat Disord. 1998;20(3):287–93.
118. Williamson DA, Davis C, Bennett SM, Goreczny AJ, Gleaves DH. Development of a simple procedure for assessing body image disturbances. Behav Assess. 1989;11(4):433–46.
119. Fisher S. The evolution of psychological concepts about the body. In: Press G, editor. Body images: development, deviance, and change. New York: Guildford Press; 1990. p. 3–20.
120. Thompson JK, Heinberg LJ, Altabe M, Tantleff-Dunn S. Exacting beauty: theory, assessment, and treatment of body image disturbance. Washington, DC: American Psychological Association; 1999.
121. Cohn LD, Adler NE, Irwin CE, Millstein SG, Kegeles SM, Stone G. Body-figure preferences in male and female adolescents. J Abnorm Psychol. 1987;96(3):276.
122. Gardner RM, Stark K, Natalie AJ, Brenda NF. Development and validation of two new scales for assessment of body-image. Percept Mot Skills. 1999;89(3):981–93.
123. Smolak L. Body image in children and adolescents: where do we go from here? Body Image. 2004;1(1):15–28.
124. Cooper PJ, Fairburn CG. Confusion over the core psychopathology of bulimia nervosa. Int J Eat Disord. 1993;13(4):385–9.
125. Ricciardelli LA, McCabe MP. Sociocultural and individual influences on muscle gain and weight loss strategies among adolescent boys and girls. Psychol Schools. 2003;40(2):209–24.

126. Netemeyer SB, Williamson DA. Assessment of eating disturbance in children and adolescents with eating disorders and obesity. In: Thompson JK, SL, editors. Body image, eating disorders, and obesity in youth. 1st ed. Washington, DC: American Psychological Association; 2001. p. 215–33.
127. Policy TUSoNSa. Statement on the link between nutrition and cognitive development in children. 1998; http://www.eecom.net/mfsp/projects_school_links.pdf. Accessed 19 Apr 2013.
128. Bandura A. Self-efficacy: the exercise of control. New York: Freeman; 1997.
129. Costa M, López E. Educación para la salud. Una estrategia para cambiar los estilos de vida. Madrid: Pirámide; 1996. p. 29–33.
130. Gustafson SL, Rhodes RE. Parental correlates of physical activity in children and early adolescents. Sports Med. 2006;36(1):79–97.
131. Compas BE. Promoting positive mental health during adolescence. In: Millstein SG, Petersen AC, Nightingale EO, editors. Promoting the health of adolescents. New York, NY: Oxford; 1993. p. 159–79.
132. O'Dea J. Evidence for a self-esteem approach in the prevention of body image and eating problems among children and adolescents. Eat Disord. 2004;12(3):225–39.
133. Branden N. Six pillars of self-esteem. New York: Bantam; 1995.
134. O'Dea J. Everybody's different: a self esteem program for young adolescents. Sydney: University of Sydney; 1995.
135. Troop NA, Treasure JL. Setting the scene for eating disorders, II. Childhood helplessness and mastery. Psychol Med. 1997;27(03):531–8.
136. Joint Committee on National Health Education Standards. Achieving health literacy: an investment in the future. Atlanta, GA: American Cancer Society; 1997.
137. Graber JA, Brooks-gunn J. Prevention of eating problems and disorders: including parents. Eat Disord. 1996;4(4):348–63.
138. Hopper CA, Munoz KD, Gruber MB, Nguyen KP. The effects of a family fitness program on the physical activity and nutrition behaviors of third-grade children. Res Q Exerc Sport. 2005;76(2):130–9.
139. Nader PR, Sellers DE, Johnson CC, et al. The effect of adult participation in a school-based family intervention to improve Children's diet and physical activity: the Child and Adolescent Trial for Cardiovascular Health. Prev Med. 1996;25(4):455.
140. Sallis JF, McKenzie TL. Physical education's role in public health. Res Q Exerc Sport. 1991;62(2):124.
141. Domel SB, Baranowski T, Davis H, et al. Development and evaluation of a school intervention to increase fruit and vegetable consumption among 4th and 5th grade students. J Nutr Educ. 1993;25(6):345–9.
142. Perry CL, Williams CL, Veblen-Mortenson S, et al. Project Northland: outcomes of a communitywide alcohol use prevention program during early adolescence. Am J Public Health. 1996;86(7):956–65.
143. Taylor CB, Altman T. Priorities in prevention research for eating disorders. Psychopharmacol Bull. 1997;33(3):413–7.
144. Levine P. Prevention guidelines & strategies for everyone: 50 says to lose the "3 Ds"—dieting, drive for thinness, and body dissatisfaction. 2002; http://www.nationaleatingdisorders.org/sites/default/files/ResourceHandouts/50WaystoLosethe3Ds.pdf. Accessed 15 Jan 2013.
145. Resnick MD, Bearman PS, Blum RW, et al. Protecting adolescents from harm. JAMA. 1997;278(10):823–32.
146. Thelen MH, Powell AL, Lawrence C, Kuhnert ME. Eating and body image concern among children. J Clin Child Adolesc Psychol. 1992;21(1):41–6.
147. Kater KJ. Healthy body image: teaching kids to eat and love their bodies too!: a comprehensive resource manual with introductory scripted-lessons for grades four, five or six. Seattle, WA: Eating Disorders Awareness and Prevention, Incorporated; 1998.
148. Kater KJ, Rohwer J, Levine MP. An elementary school project for developing healthy body image and reducing risk factors for unhealthy and disordered eating. Eat Disord. 2000;8(1):3–16.
149. Smolak L, Levine MP, Schermer F. A controlled evaluation of an elementary school primary prevention program for eating problems. J Psychosom Res. 1998;44(3):339–53.
150. Smolak L, Levine MP. A two-year follow-up of a primary prevention program for negative body image and unhealthy weight regulation. Eat Disord. 2001;9(4):313–25.
151. Piran N. Prevention: can early lessons lead to a delineation of an alternative model? A critical look at prevention with schoolchildren. Eat Disord. 1995;3(1):28–36.
152. Abascal L, Brown JB, Winzelberg AJ, Dev P, Taylor CB. Combining universal and targeted prevention for school-based eating disorder programs. Int J Eat Disord. 2003;35(1):1–9.
153. Smolak L, Harris B, Levine MP, Shisslak CM. Teachers: the forgotten influence on the success of prevention programs. Eat Disord. 2001;9(3):261–5.
154. Boes S, Ng V, Daviston T. Resource: bodywise handbook: eating disorders information for middle school personnel. Professional School Counsel. 2004;7:376–7.
155. Rideout V, Roberts D, Foehr U. Generation M: media in the lives of 8-18 year-olds. Executive summary. A Kaiser Family Foundation Study. Menlo Park, CA: The Henry J Kaiser Family Foundation; 2005.
156. Foundation KF. Key facts: media literacy; 13 April 2003. Available at http://www.kff.org/entmedia/Media-Literacy.cfm. Accessed 2013.
157. Irving LM, DuPen J, Berel S. A media literacy program for high school females. Eat Disord. 1998;6(2):119–31.
158. Piran N, Levine MP, Irving LM. GO GIRLS! Healthy Weight. 2000;14(6):89.

159. Trends C. Go Girls! 2007; http://www.childtrends.org/lifecourse/programs/GoGirls.htm. Accessed 14 Mar 2013.
160. Chesley EB, Alberts J, Klein J, Kreipe R. Pro or con? Anorexia nervosa and the Internet. J Adolesc Health. 2003;32(2):123–4.
161. Neumark-Sztainer D. School-based programs for preventing eating disturbances. J School Health. 1996;66(2):64–71.
162. Kater K. Promotion of healthy weight: helping without harming. Synergy Learn. 2006;19:22–3.
163. Kater KJ. The model for healthy body image and weight. North St. Paul, MN. www.bodyimagehealth.org; 2012.
164. Catalano RF, Berglund ML, Ryan JA, Lonczak HS, Hawkins JD. Positive youth development in the United States: research findings on evaluations of positive youth development programs. Prevntion Treat. 2002;5(1):15a.
165. Hawkins J, Arthur M, Catalano R. Preventing substance abuse. In: Farrington DP TM, editor. Building a safer society: strategic approaches to crime prevention, crime and justice: a review of research. Chicago: University of Chicago; 1995. p. 343–427.
166. Lonczak HS, Huang B, Catalano RF, et al. The social predictors of adolescent alcohol misuse: a test of the social development model. J Stud Alcohol. 2001; 62(2):179.
167. Catalano R, Hawkins J. The social development model: a theory of antisocial behavior. In: Hawkins J, editor. Delinquency and crime: current theories. New York, NY: Cambridge University; 1996. p. 149–97.
168. Lonczak HS, Abbott RD, Hawkins JD, Kosterman R, Catalano RF. Effects of the Seattle Social Development Project on sexual behavior, pregnancy, birth, and sexually transmitted disease outcomes by age 21 years. Arch Pediatr Adolesc Med. 2002; 156(5):438.
169. Huang B, Kosterman R, Catalano RF, Hawkins JD, Abbott RD. Modeling mediation in the etiology of violent behavior in adolescence: a test of the social development model. Criminology. 2001;39(1): 75–108.
170. Hawkins JD, Guo J, Hill KG, Battin-Pearson S, Abbott RD. Long-term effects of the Seattle Social Development Intervention on school bonding trajectories. Appl Dev Sci. 2001;5(4):225–36.
171. Hawkins JD, Catalano RF, Miller JY. Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: implications for substance abuse prevention. Psychol Bull. 1992;112(1):64.
172. Mrazek PB, Haggerty RJ. Reducing risks for mental disorders: frontiers for preventive intervention research. Washington, DC: National Academies Press; 1994.
173. Stice E, Shaw H. Eating disorder prevention programs: a meta-analytic review. Psychol Bull. 2004; 130(2):206.
174. Massey-Stokes M, Barton B, Golman M, Holland D. An ecological approach to the prevention of eating disorders in children and adolescents. In: Goodheart K, Clopton JR, Robert-Mccomb JJ, editors. Eating disorders in women & children: prevention, stress management & treatment. 2nd ed. Boca Raton, FL: CRC Press; 2012. p. 225–70.

# The Physiology of Anorexia Nervosa and Bulimia Nervosa

11

Jacalyn J. Robert-McComb, Kembra D. Albracht, and Annette Gary

**Abstract**

Eating disorders (ED) are psychological disorders that are characterized by abnormal eating, dysfunctional relationships with food, and a preoccupation with one's weight and shape. The incidence of EDs in women ranges from 0.5 to 3 % with the incidence increasing from 1963 to 2013. Currently, the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) recognizes two specific EDs: anorexia nervosa (AN) and bulimia nervosa (BN), although there are subtypes associated with each. The DSM-IV-TR and the International Classification of Diseases (ICD-10) have different criteria for diagnosing AN and BN. Early identification of an ED is associated with shorter duration and fewer medical complications. Yet, it is estimated that only about 33 % of AN patients and 6 % of BN are receiving proper treatment for their illnesses. Gastrointestinal upset, fluid and electrolyte imbalances are common in AN in the short term and can eventually lead to long-term complications such as, pernicious anemia, osteoporosis, and heart disease. On the other hand, BN can cause short-term adverse effects like erosion of the teeth, enlargement of the parotid salivary glands, and acidic stomachs leading to heartburn. Long-term adverse effects caused by BN are gynecological problems, hormonal disturbances, hypercholesterolemia, and hypertension. Successful treatment of EDs should be managed with a team-based approach including the physician, psychologist, and registered dietitian.

**Keywords**

Physiology of anorexia nervosa • Physiology of bulimia nervosa • Genetics

J.J. Robert-McComb, Ph.D., FACSM, ACSM P.D. (✉)
K.D. Albracht, B.S.
Department of Health, Exercise, and Sports Sciences, Texas Tech University, Lubbock, TX, USA
e-mail: jacalyn.mccomb@ttu.edu; kembra.albracht@ttu.edu

A. Gary, Ph.D., M.S.N., R.N.
Starcare Specialty Health System, 904 Ave O, Lubbock, TX 79401, USA
e-mail: agary@starcarelubbock.org

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_11, © Springer Science+Business Media New York 2014

## 11.1 Learning Objectives

After completing this chapter, you should have an understanding of:

- The significance of an eating disorder
- The varying types of eating disorders
- Differentiating and similar signs and symptoms of each disorder
- The physiological changes that occur in anorexia nervosa and bulimia nervosa, specifically
- The potential long-term physiological consequences of anorexia nervosa and bulimia nervosa
- The importance of genetics, neurotransmitters, and key hormones in eating disorders

## 11.2 Introduction

Eating disorders (EDs) are psychological disorders that are characterized by abnormal eating, dysfunctional relationships with food, and a preoccupation with one's weight and shape [1]. More and more, these disorders are being recognized by medical professionals as biologically based psychiatric disorders [2]. EDs affect daily functioning and often result in physical complications and psychological distress [1]. The current *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV-TR*) [1] recognizes two specific EDs: anorexia nervosa (AN) and bulimia nervosa (BN). There are two subtypes associated with each specific ED: anorexia nervosa, restricting type (AN-R); anorexia nervosa, binge/purge type (AN-BP); bulimia nervosa, purging type (BN-P); and bulimia nervosa, non-purging type (BN-N). A third category, eating disorder not otherwise specified (EDNOS), is included for EDs of clinical significance that do not meet criteria for AN or BN. Within this broad category, there are currently three subdivisions: (1) binge-eating disorder (BED), which is currently under review and has been recommended to be included in the *DSM-IV-TR* as a specific clinical ED [3]; (2) subthreshold AN or BN (i.e., disorders very similar in symptoms or presentation to either AN or BN but not fully meeting the criteria for a diagnosis of either disorder); and (3) disorders with symptoms of both AN and BN but again not meeting the criteria for either disorder [4]. Additionally, it has been recommended that purging disorder (PD) be included in the *DSM-IV-TR* under the EDNOS classification for further review so that the appropriateness of including PD as a full-threshold ED can be determined [5].

Several characteristics that affect health outcomes are represented across EDs. Some of these characteristics include: (1) demographic characteristics; (2) experiences in adolescence; (3) low self-esteem and negative ideations; (4) medical and psychological comorbidity; and (5) issues with weight, shape, and the stereotype of beauty [6]. The majority of people with EDs experience other psychological disorders as well. Factors such as swiftness of weight loss, current weight, and chronicity of the ED are related to the intensity of the comorbid illness [7]. One study found that 56.2 % of individuals with AN, 94.5 % with BN, 78.9 % with BED, and 63.3 % with subthreshold BED met the criteria for another mental disorder, most often mood disorders, anxiety disorders, impulse control disorders, or substance use disorders [8]. Additionally, some form of personality disorder affects between 27 and 93 % of ED patients [9, 10]. Eating disturbances that do not meet full criteria for a clinical eating disorder are associated with elevated risk of depression, anxiety disorders, substance abuse, or health complications [11].

It is difficult to estimate the true prevalence of EDs due to underreporting [7, 12]. Even with considerable underreporting, the incidence of EDs has increased over the past 50 years. Increase in the prevalence of EDs might be due to improved understanding of the symptomatology and risk factors, as well as changes in diagnostic criteria, referral practices, and accessibility to help [12].

Generally, the incidence of EDs decreases with age. Many young people suffer from some form of disordered eating, whereas the incidence of EDs in women ranges from 0.5 to 3 % [13, 14]. Even though these numbers might seem low, this incidence rate is problematic as EDs are commonly

listed in the top ten causes of disability and mental illness in young women [15, 16]. Frequently, the onset age for AN and BN is between 15 and 19 [13, 15, 17, 18]. Although EDs and body dissatisfaction are typical for young women, they can occur in older women. In a randomly selected nonclinical sample of 1,000 women aged 60–70 years, more than 80 % used strategies to control their weights and over 60 % reported body dissatisfaction. Eighteen women (3.8 %) met the criteria for an eating disorder [19]. Because EDs can develop when people experience life transitions, are independent, and have an ample amount of privacy [20], one possible reason that some older women have is because they have lost their spouses, are living alone, or are at a time of transition in their lives.

As one ages, there is also a slight change in the expression of EDs. Adolescents and young women are more likely to show signs and symptoms related to AN and BN, whereas older adults may exhibit signs and symptoms more closely aligned to BED [16, 18]. These differences may exist because young people are more likely than older people to internalize cognitive distortions about body image and pressure from society to be thin [12].

At any age, EDs are complex, serious, maladaptive, and result in adverse health consequences [1, 7, 21]. EDs typically last several years and tend to have high relapse rates [11, 15, 20]. Specifically, the average duration of BN is 8.3 years, and the average duration of BED and subthreshold levels of BED is 8.1 and 7.2 years, respectively [8]. By the fifth year of the ED, the symptoms, pathology, and the clinical track of the ED will likely stabilize [22]. Unfortunately, the chronicity of EDs can ultimately result in diminished health, decreased psychosocial functioning, and compromised interpersonal interactions [7, 11, 17, 23].

Early identification of an ED is associated with shorter duration and fewer medical complications [24]. Unfortunately, recent estimates show that only about 33 % of AN patients and 6 % of BN patients are receiving proper treatment for their illnesses [12]. Comorbidity plays an important role in the treatment of EDs, as people are more likely to seek treatment for their non-ED mental health problems than for the ED itself [8]. In clinical settings, women and girls are ten times more likely than men and boys to receive treatment [7, 15, 20], but this ratio might not accurately represent the number of men and boys compared to women and girls who actually have eating disorders [20]. Thus, increased understanding of EDs is imperative so that treatment for people with EDs is more accessible and more effective.

### 11.2.1 Diagnostic Criteria for Anorexia Nervosa

AN is a drastic reduction in eating resulting in very low body weight [25]. Although patients with AN consume food, they eat with extreme limitations. Strict calorie and food restriction, as well as obsessive exercise, can produce an unhealthy level of weight loss in AN patients [7, 25]. Weight concerns, fear of fatness, social influence, distorted body shape, eating concerns, yearning for thinness, and body dissatisfaction are consistently found to be predictors of EDs and lie at the core of AN [11–29]. Often, the AN patient is totally preoccupied by thoughts of shape and weight, including obsession about weight gain and the perceived largeness of her body, and she often sees herself as being overweight regardless of how much weight she loses [7, 17, 18, 29]. An excess of physical activity has been recognized as hyperactivity in AN patients and is present in roughly 80 % of those with AN [30].

*DSM-IV-TR* [1] includes specific diagnostic criteria for AN. Initially, the individual must be at a weight that is 85 % or less of her expected weight, based on her height and her age, which is equivalent to a body mass index (BMI) of less than 18 (A BMI that falls between 18.5 and 24.9 is considered to be in the healthy range). Additionally, individuals who have started menstruating will develop secondary or functional hypothalamic amenorrhea. Amenorrhea is the absence of menstrual bleeding. Secondary amenorrhea is defined as the cessation of menses for 6 or more months sometime after menarche has occurred.

Functional hypothalamic amenorrhea is a reversible form of gonadotropin-releasing hormone deficiency commonly triggered by stressors such as excessive exercise, nutritional deficits, or psychological distress. For those girls who have not reached menarche, they will typically fail to begin menstruating at the expected time this is known as primary amenorrhea. Primary amenorrhea is the failure of menses to occur by age 16 years, in the presence of normal growth and secondary sexual characteristics. Psychological criteria include extreme disturbance in self-perception of the body and an overwhelming fear of fatness. AN patients can be further subdivided into two subtypes: restricting subtype (AN-R) and binge/purge subtype (AN-BP). The AN-R subtype describes those individuals who severely restrict their food intake and do not use compensatory behaviors, like self-induced vomiting, to compensate for calories consumed. The AN-BP subtype is diagnosed when periods of restriction are accompanied by periods of overeating and extreme compensatory purging behaviors, like self-induced vomiting. AN, particularly the restricting type (AN-R), is the most rare form of eating disorder.

Some question the validity of the diagnostic criteria for AN that are included in the *DSM-IV-TR*. The strongest debate has risen regarding the inclusion of amenorrhea. Some suggest that the differences between AN patients who do and do not menstruate are very limited [31–33]. Additionally, a large number of females use birth control or other substances that affect hormones, so it can be difficult to determine if a woman would develop amenorrhea if she were not using these substances [7]. Although some argue that fear of weight is a classic feature of AN, this fear may not be present in all individuals with AN [34]. Furthermore, the subtypes associated with AN have been questioned. The AN-R and AN-BP subtypes were originally differentiated because women in these two subgroups were thought to be different in terms of comorbidity and recovery cycles, but recent evidence suggests that the current separation might not be needed [34]. Still, others have pushed to eliminate the AN-BP subtype all together and limit AN to only those individuals who severely restrict their food intake and to include AN-BP with BN and include a low-weight specifier [35].

Another commonly used classification system for EDs and other illnesses is the *tenth edition of the International Classification of Diseases* (ICD-10). The ICD-10 specifies that patients with AN have a BMI equal to or below 17.5, which is well below the healthy range (18.5–24.9) [36]. Other ICD-10 criteria for AN include amenorrhea, weight loss that is self-induced and purposeful, a fear of fatness, and a perception of being fat. Unlike the *DSM-IV-TR* classification, the ICD-10 specifies that binge eating is an exclusionary criteria for AN. The ICD-10 criteria are based on behavioral symptoms and methods of weight loss, in contrast to the *DSM-IV-TR* that emphasizes psychological distortions and disturbances [17, 36]. Table 11.1 presents a comparison of the *DSM-IV-TR* and ICD-10 diagnostic criteria for AN.

There are different theories that attempt to explain the onset and continuation of AN. However, it is thought that a combination of genetics, environmental factors, and specific personality traits likely contributes to the development and maintenance of AN [37].

Research suggests that personality disorders most commonly associated with AN-R include avoidant, dependent, obsessive-compulsive, and borderline personality disorders [9, 38]. Additionally, anxiety and mood disorders affect about 25 % of people with AN [39], and comorbid conditions like depression, anxiety, phobias, and personality disorders might contribute to worse outcomes for AN patients [39].

It has been suggested that AN patients have lower reward sensitivity than other individuals [25]. For example, they often deny food can be satisfying; therefore, it might be easier for them to skip food because the food is not as pleasing for them as it is for people without AN.

Some research suggests that AN patients have lower levels of novelty seeking and higher levels of harm avoidance, and both factors can prevent a person from developing binge eating and purging behaviors [9, 40]. However, AN-BP patients do develop binge eating and purging behaviors, and these behaviors are intermingled with periods of fasting, excessive exercise, and other compensatory behaviors [7].

**Table 11.1** Comparison of DSM-IV-TR and ICD-10 diagnostic criteria for anorexia nervosa

| | DSM-IV | ICD-10 |
|---|---|---|
| Code | 307.1 | F50.0 |
| Weight | Refusal to maintain body weight at or above minimal normal weight for age and height (e.g., weight loss leading to maintenance of body weight <85 % of expected weight) or<br>Failure to make expected weight gain during growth period, leading to weight <85 % of expected normal body weight | Body weight is maintained at least 15 % below that expected (either lost or never achieved)<br>Quetelets's body mass index is 17.5 kg/m$^2$ or less or<br>Prepubertal patients may show failure to make the expected weight gain during the period of growth |
| Phobia/associated behaviors | Intense fear of gaining weight or becoming fat, even though underweight<br>DSM-IV behaviorally differentiates between types:<br>Restricting = not engaging in binge-eating or purging behavior<br>Binge eating/purging = regularly engaging in binging or purging behavior | Weight loss self-induced by avoidance of "fattening foods" and<br>One or more of the following: self-induced vomiting, self-induced purging, excessive exercise, use of appetite suppressants and/or diuretics |
| Body perception | Disturbance in the way in which one's body weight and shape are experienced<br>Undue influence of body weight or shape on self-evaluation or<br>Denial of the seriousness of the current low body weight | Body-image distortion in the form of a specific psychopathology whereby a dread of fatness persists as an intrusive, overvalued idea and<br>Patient imposes a low weight threshold on himself or herself |
| Amenorrhea/hormonal fluctuations | In postmenarcheal females, amenorrhea, i.e., the absence of at least three consecutive menstrual cycles (amenorrhea exists if periods occur only via hormone induction) | In women, amenorrhea, and in men, loss of sexual interest and potency (an apparent exception is the persistence of vaginal bleeds in anorexic women who are receiving replacement hormonal therapy, most commonly taken as a contraceptive pill)<br>There may also be elevated levels of growth hormone, raised levels of cortisol, changes in the peripheral metabolism of the thyroid hormone, and abnormalities of insulin secretion |
| Pubertal development | Not specified | With prepubertal onset, the sequence of pubertal events is delayed or even arrested (growth ceases; in girls, the breasts do not develop and there is a primary amenorrhea; in boys, the genitals remain juvenile). With recovery, puberty is often completed normally, but the menarche is late |

*Source*: Bulik, C. M., L. Reba, A. M. Siega-Riz, and T. Reichborn-Kjennerud. 2005. Anorexia nervosa: Definition, epidemiology, and cycle of risk. *Int J Eat Disord* 37: s2–s9. Reprinted with permission of John Wiley & Sons, Inc.

### 11.2.2 Diagnostic Criteria for Bulimia Nervosa

Prevalence rates of BN range from 1 to 4 %, making BN approximately three times more common than AN [6, 7, 12, 18, 39]. BN is a complex disorder characterized by recurrent binge eating, compensatory behaviors to avoid weight gain, and related behavioral and physiological symptoms. The diagnostic criteria for BN include several criteria that reflect the physical manifestation of the disorder. Specifically, the current *DSM-IV-TR* criteria indicate that all women with BN have recurrent episodes of binge eating [1].

According to the *DSM-IV-TR*, BN is characterized by compulsive, extreme binge eating followed by a compensatory method like self-induced vomiting, misuse of diuretics or laxatives, or excessive exercise to make up for the excessive amount of calories consumed. Diagnostic criteria state that this type of cyclic behavior must occur for at least 3 months, and binge eating and purging episodes are to occur at least two times per week. The main psychological factor in BN is loss of control during episodes of binge eating. Additionally, the BN patient places high importance on body weight and physical appearance. There are two subtypes of BN: The purging subtype (BN-P) includes those people who make themselves vomit or use laxative or diuretics to compensate for a binge, and the non-purging subtype (BN-N) includes those people who use other forms of compensatory behavior, such as excessive exercise or fasting [1].

An episode of binge eating is characterized by both of the following: (1) eating within any 2-h period an amount of food that is definitely larger than most people would eat; and (2) a sense of lack of control overeating or a feeling that one cannot stop eating or control what or how much is being consumed. To compensate for the large amount of food that has been consumed during the eating binge, the individual with BN engages in behaviors to prevent weight gain, such as (1) self-induced vomiting; (2) misuse of laxatives, diuretics, enemas, or other medications; (3) fasting; or (4) excessive exercise. To be diagnosed with BN using the current *DSM-IV-TR* criteria, the binge eating and compensatory behaviors must occur, on average, at least twice a week for 3 months. Another diagnostic criterion for BN is that self-evaluation is unduly influenced by body shape and weight [41].

As with AN, there is also ICD-10 criteria for BN diagnosis. The ICD-10 criteria for BN are similar to the *DSM-IV-TR* criteria. The ICD-10 criteria for BN include having an adverse relationship or preoccupation with food, engaging in binge eating and purging behaviors, and attempting to keep body weight below a level that would optimize health [36]. Table 11.2 provides a comparison of the DSM-IV-TR and ICD-10 diagnostic criteria for BN.

**Table 11.2** Comparison of DSM-IV-TR and ICD-10 diagnostic criteria for bulimia nervosa

| | DSM-IV-TR | ICD-10 |
|---|---|---|
| Code | 307.51 | F50.2 |
| Relationship with food | Binge/purge cycle must occur for at least 3 months, at a rate of two times per week, on average | Continued obsession with food<br>Strong cravings<br>Fear of weight gain |
| Binge eating | Eating, in 2 h or less, a portion of food that is substantially larger than most others would eat<br>Loss of control when consuming this food | Period of overeating when are a great deal of food is eaten quickly |
| Purging tendencies | Recurrent methods of compensation (vomiting, diuretics, other medications, fasting or exercise) | Methods to compensate for the binge<br>Vomiting, restriction of food intake, drug use may include appetite suppressants, thyroid treatments or diuretics |
| Beauty ideal | Weight and shape are a major influence in defining the identity of a BN patient | Strives to achieve a weight that is well standard weight or weight expected for a particular person |

*Source*: Bulik, C. M., L. Reba, A. M. Siega-Riz, and T. Reichborn-Kjennerud. 2005. Anorexia nervosa: Definition, epidemiology, and cycle of risk. *Int J Eat Disord* 37: s2–s9. Adapted with permission of John Wiley & Sons, Inc.

Individuals who engage in binge eating also commonly engage in the excessive use of weight-loss supplements. According to Reba-Harrelson et al. [42] women with BN are more likely to use diet pills if they have a higher BMI, higher novelty seeking, anxiety disorders, alcohol abuse, or borderline personality disorder. Many of these characteristics are commonly found to co-occur with BN [42].

### 11.2.3 Eating Disorder Crossover and Identification

It is difficult for researchers to design well-controlled studies for EDs, particularly studies that will distinguish the causes and early signs of EDs. There are multiple reasons for this difficulty. For instance, it is often difficult to accumulate a large enough sample of people with EDs, and AN and BN often consist of similar or overlapping characteristics [13, 17, 37].

Although AN and BN are separate disorders, there are several traits commonly found in both AN and BN patients: Specifically, high neuroticism, perfectionism, obsessiveness, and low self-directedness are characteristic of both AN and BN. These shared traits may increase the chance of crossover from AN to BN [22]. Commonalities between AN and BN patients may be attributed to the symptom overlap between the two disorders or may help account for the high crossover rate from AN to BN [17, 22]. Other traits associated with the crossover from AN to BN include prior anxiety, childhood sexual abuse, negative affect, and improvement in the AN condition [22, 37].

The symptoms associated with AN-BP and both subtypes of BN overlap, making diagnosis difficult. A person's weight distinguishes those with BN from those with AN-BP. People with BN typically have a weight that is in the normal range, and some are overweight [43].

Similar to AN, some diagnostic criteria for BN are controversial. For example, individuals who binge eat and purge once each week rather than twice each week experience similar levels of eating-related pathology, and the definition of a binge (i.e., "an amount of food that is larger than most people would eat") is not objective or easily measurable [7].

Also, ED classification can be difficult as crossover from one form of ED to another is fairly common. It has been estimated that between 8 and 62 % of patients with AN eventually crossover to BN [17, 22]. The highest percentage of crossover occurs when restricting AN patients crossover to the binge/purge subtype of AN (AN-BP) or to BN [7, 22]. Additionally, BN can crossover to BED [16, 44]. Crossover from one ED to another typically occurs during the first 5 years of the illness; after 10 years, the rate of crossover is substantially reduced [7, 17]. Furthermore, certain personality traits in AN patients, such as novelty seeking and low self-directedness, have been associated with higher crossover probability compared to other AN patients without these traits [45]. There are theories attempting to explain why the crossover from AN to EDs with bingeing behavior occurs frequently. Several animal and historical studies have shown that restricted eating will result in binge eating later; therefore, many believe that the restricting behavior directly causes a person to begin binge eating [25].

## 11.3 Research Findings

### 11.3.1 Short-Term Adverse Effects of Anorexia Nervosa

Numerous physiological signs and symptoms are associated with AN, and many of these physical symptoms are life-threatening [39]. A summary of some of the physical consequences of AN is provided in Table 11.3.

Although numerous physical abnormalities may be found in people with AN, research findings indicate that laboratory results may be normal even in the presence of profound malnutrition [46].

The body weight of the individual with AN generally reflects the degree of calorie restriction, the severity of purging behaviors (if present), and the amount of exercise engaged in by the individual. Adults who are diagnosed with AN generally

**Table 11.3** Physical consequences of anorexia nervosa

| *Cardiovascular* | *Gastrointestinal* |
|---|---|
| Bradycardia | Abdominal discomfort |
| Tachycardia | Bloating/feeling of fullness |
| Arrhythmias | Constipation |
| Hypotension | Delayed gastric emptying |
| Fainting | Decreased gastric and intestinal motility |
| Dizziness | Pancreatitis |
| *Endocrine* | *Integumentary* |
| Amenorrhea | Dry, flaky/scaly, yellowish orange skin |
| Cold sensitivity | Decreased body fat |
| Oligomenorrhea | Lanugo (fine facial and body hair) |
| Anovulation | Thinning hair |
| | Brittle nails |
| *Hematologic* | *Central nervous system* |
| Anemia | Poor problem-solving skills and memory |
| Hypercortisolism | Decreased concentration and attention |
| Leukopenia | Depressed mood |
| Pancytopenia | Peripheral neuropathy |
| Thrombocytopenia | Seizures |
| *Skeletal* | *Fluids and electrolytes* |
| Osteopenia | Electrolyte imbalance |
| Osteoperosis | Dehydration |
| Bone fractures | Rebound peripheral edema |
| Stunted growth | Renal failure |
| | Metabolic acidosis |

show extreme weight loss and may be described as thin or emaciated. Children and adolescents may not lose an extreme amount of weight, because weight loss goes against the body's natural tendency to grow larger. Rather, children and adolescents might lose smaller amounts of weight (e.g., 5–10 lb) or might not grow to a weight that would be expected for them based on their height, age, and developmental level [47].

#### 11.3.1.1 Gastrointestinal Abnormalities

Individuals with AN frequently describe mealtime as an uncomfortable experience associated with symptoms of anxiety, such as sweating and increased pulse and respiratory rates [48]. When a person eats little food, a series of adverse consequences affect normal digestion. Food is held for longer periods in the stomach and intestines, which can produce bloating and a feeling of being full, stomachache, and constipation. Food normally passes through the stomach in about an hour, but when the consumption of food is restricted, food may stay in the stomach for 4 or 5 h [47]. So, it may be difficult for a young woman with anorexia to resume a normal pattern of eating. After eating a normal amount of food for lunch, she may still feel full when it is time for supper.

Due to malnutrition [49] and repeated episodes of binge eating [50], pancreatitis is a common occurrence in patients with eating disorders. Patients with pancreatitis may complain of steady and intense upper abdominal pain that may diffuse to the back, chest, or lower abdomen. Numerous mechanisms have the potential to cause pancreatitis, including a sudden increase in calorie intake after malnutrition or the ingestion of various medications including the laxatives and diuretics used in purging [51].

#### 11.3.1.2 Fluid and Electrolyte Abnormalities

Individuals with AN can develop imbalances in body fluid and electrolyte levels due to prolonged malnutrition and dehydration. These imbalances can reduce fluid and mineral levels and produce a condition known as *electrolyte imbalance.* Fluid and electrolyte imbalances can become more serious when an individual also engages in purging behaviors, such as vomiting and laxative abuse. Dehydration may result from inadequate fluid intake or excessive fluid loss during purging or exercise. Dehydration leads to increased blood levels of urea, urate, and creatinine and dehydration may result in decreased urine volume and renal failure. A *rebound peripheral edema* (i.e., swelling of body tissue due to excessive fluid retention) may also occur and can contribute to a dramatic increase in body weight (approximately 10–45 lb). *Metabolic acidosis*, a condition when the body produces too much acid or the kidneys do not remove enough acid, may result from vomiting and loss of stomach acid and sodium bicarbonate. If the individual also engages in laxative abuse, the loss of alkaline bowel fluids

may result in metabolic acidosis [52]. Individuals who abuse laxatives are four times more likely to suffer serious medical complications than non-laxative abusers [53]. Reduced blood flow, and lower blood pressure have all been linked to electrolyte imbalance. Electrolytes, such as calcium and potassium, are critical for maintaining the electric currents necessary for a normal heartbeat. These imbalances can be very serious and can even be life-threatening unless fluids and minerals are replaced.

#### 11.3.1.3 Integumentary Abnormalities

Malnutrition, loss of body fat, and dehydration can also cause changes in skin and tissues. Frequently, the skin is dry, scaly, and covered with *lanugo*, a fine, downy hair resembling that of newborn babies. Fingernails and hair are often brittle, and hair loss may occur in patches or uniformly over the scalp and other body areas [54]. A yellowish or orangish discoloration of the skin occurs in approximately 80 % of patients with AN [46]. This unusual skin color, which is "most noticeable on the palms of the hands, the soles of the feet, and the creases inside the elbows," is due to faulty metabolism of β-carotene in the liver leading an excessive level of β-carotene circulating in the blood, some of which is deposited under the skin [47].

Additionally, severe starvation has been linked to depressed mood, decreased concentration and attention, and poor problem-solving skills and memory. These problems may contribute to poor judgment about the severity of the illness and, therefore, hamper the individual's recognition of the need for treatment [48].

### 11.3.2 Long-Term Adverse Health Effects of Anorexia Nervosa

#### 11.3.2.1 Hematologic and Immunologic Abnormalities

Hematologic and immunologic abnormalities are often found in patients with AN. Poor nutrition with severe weight loss often results in dramatic decreases in red blood cells, white blood cells, and blood platelets. Anemia is a common result of anorexia and starvation. One particularly serious blood problem is pernicious anemia, which can be caused by severely low levels of vitamin $B_{12}$. In some severe cases of AN, the bone marrow dramatically reduces its production of blood cells, a life-threatening condition called *pancytopenia.* Impairment of the immune system is also common and is believed to be a consequence of *hypercortisolism* (i.e., excessive amounts of the hormone cortisol). These effects can be corrected with nutritional improvement and weight restoration.

#### 11.3.2.2 Skeletal Problems

Although long-term complications of AN can involve any of the body's systems, the bones are significantly affected. Since puberty is a critical time for skeletal development, developing AN during this period can interfere with the development of peak bone mass and, therefore, produce permanent long-term skeletal effects [48]. When a young child is severely underweight, there is a danger that the child's growth will be limited. However, if AN starts after puberty begins and ends before the growth plates in her bones have closed, then a young woman's growth in height will not be stunted [55].

Bone loss and decreased bone density are common problems for people with AN and is particularly prevalent in individuals with AN who have been severely emaciated for a prolonged period of time [56–58]. However, fractures of the long bones, vertebrae, and sternum have been reported in individuals with AN who have had amenorrhea for as short a period as 1 year [59]. Approximately 90 % of women with anorexia experience osteopenia (decrease in bone mass) and some have osteoporosis (brittle and fragile bones) [60]. In a study assessing decreased bone density and bone loss in women with AN, nearly half of the women had osteopenia at the hip, and 16 % had osteoporosis at the hip. More than half had osteopenia at the spine, and almost 25 % had osteoporosis at the spine. Over 90 % of women had abnormally low bone density at one or more sites in the skeleton. Additionally, weight was the factor most related to bone loss. The less a woman weighed, the more likely it was that she would have substantial bone loss [60].

In a long-term study of 103 patients with AN, osteoporosis with multiple fractures and terminal renal deficiency accounted for the most severe disabilities experienced by the patients [61]. Bone loss, osteopenia, and associated stress fractures have been linked to endocrine disturbances that alter normal hormonal mechanisms and lead to oligomenorrhea and amenorrhea [62]. Oligomenorrhea is infrequent (or, in occasional usage, very light) menstruation. More strictly, it is menstrual periods occurring at intervals of greater than 35 days, with only four to nine periods in a year. Amenorrhea is the absence of a menstrual period in a woman of reproductive age.

Dietary deficiency, low circulating estrogen levels, hypercortisolism, laxative misuse, and disturbed acid–base balance also contribute to adverse physical consequences [63]. Other factors contributing to bone loss include high levels of stress hormones (which impair bone growth) and low levels of calcium, certain growth factors, and dehydroepiandrosterone (DHEA). Skeletal problems can be minimized or prevented by early recognition and intervention, but long-term complications can be expected to occur and progress as long as an individual continues to exercise without proper nutritional intake [64]. Unfortunately, weight gain does not completely restore bone loss, but achieving regular menstruation as soon as possible can protect against permanent bone loss. The longer the eating disorder persists the more likely the bone loss will be permanent.

Over 60 % of patients with AN have leukopenia (a reduction in the number of leukocytes in the blood), and this abnormality may be related to bone marrow hypoplasia and decreased neutrophil (a granular leukocyte having a nucleus of three to five lobes) lifespan [46]. Leukopenia accompanied by a relative lymphocytosis has also been reported [59]. Normochromic anemia, normocytic anemia, and thrombocytopenia have been found in approximately one-third of patients with AN [65].

#### 11.3.2.3 Central Nervous System Abnormalities

Disruptions in neuroendocrine and neurotransmitter systems are prevalent in people with AN and affect the brain and other parts of the body. Seizures, disordered thinking, and *peripheral neuropathy* (i.e., numbness or odd nerve sensations in the hands or feet) have all been reported. Structural changes in the brain include: widening of the sulcal spaces and cerebroventricular enlargement [66] and reductions in the size of the pituitary [67]. People with AN also demonstrate increased metabolism in the cortex and caudate nucleus [68].

#### 11.3.2.4 Effects on Pregnancy

Research suggests that most pregnant women with a history of EDs have healthy pregnancies [69]. However, some research suggests that women who have had EDs may face higher risks for a number of complications, including cesarean sections, postpartum depression, miscarriages, complicated deliveries, and premature birth [47, 70]. In one of the few studies that investigated pregnancy outcomes for women who had a previous diagnosis of AN, a large sample of women who were discharged from hospital with a diagnosis of AN during 1973 to 1996 and who gave birth during 1983 to 2002 were compared with a large sample of healthy women who gave birth during the same years. The researchers collected information about preeclampsia (i.e., pregnancy-induced hypertension), instrumental delivery, prematurity, small for gestational age, birth weight, Apgar score, and perinatal mortality. Results showed that the main birth outcome measures in women with a history of AN were very similar to those without a history of AN. The only observed difference was a slightly lower mean birth weight for babies whose mothers had a history of AN [69]. This research suggests that women who have a history of AN and who have been treated for the disorder are often able to become pregnant and have healthy pregnancies.

### 11.3.3 Comorbidities and Mortality Rates for Anorexia Nervosa

#### 11.3.3.1 Cardiovascular Abnormalities

Some of the most serious and life-threatening complications of AN result from impairment of the cardiovascular system. For example, people with AN might complain of heart palpitations,

dizziness, fainting, shortness of breath, and chest discomfort. If these cardiovascular abnormalities are not recognized and treated, they could result in death.

Heart disease is the most common medical cause of death in people with AN, and a primary danger to the heart is from abnormalities in the balance of minerals, such as potassium, calcium, magnesium, and phosphate. Prolonged starvation leads to decreased sympathetic tone in the heart and blood vessels. The heart's ability to pump and the vessels' ability to transport blood may be altered, which could result in *bradycardia* (i.e., heart beats too slowly), *tachycardia* (i.e., heart beats too quickly), or extremely low blood pressure [65]. Bradycardia may occur due to a starvation-induced metabolic decrease controlled by circulating *catecholamines* (i.e., "fight or flight" hormones released in response to stress) and a change in thyroid hormone levels. Tachycardia can occur when the circulating fluid volume decreases as a result of dehydration, and the heart is forced to pump faster to compensate for the decrease [71]. The reduction in blood pressure may lead to light-headedness or dizziness, and the individual with anorexia may experience orthostatic hypotension (lightheadedness when standing up or getting out of bed) [47]. Episodes of fainting may occur because of abnormally low blood pressure. Studies have shown that 91 % of individuals with AN have pulse rates less than 60 beats per minute [72], and up to 85 % of patients with AN also have hypotension, with blood pressures below 90/60 [73]. Individuals with AN have been found to have higher incidences of mitral valve abnormalities and left ventricular dysfunction than individuals who do not have eating disorders [74]. All of these factors contribute to a significant risk of sudden death due to cardiovascular problems in this population [75, 76].

Cardiovascular abnormalities also contribute to the coldness that people with AN experience. Because the blood circulates more slowly, a person's hands and feet turn cold and also appear blue because red blood cells have been depleted of oxygen. However, another reason that individuals with anorexia feel cold is the loss of the insulation normally provided by a thin layer fat all over the body [47].

#### 11.3.3.2 Endocrine Abnormalities

AN affects the endocrine system by producing numerous alterations in neuroendocrine mechanisms. Changes in the hypothalamic-pituitary-adrenal axis (HPA axis) result in hypercortisolemia and increased cerebrospinal fluid (CSF) levels of corticotropin-releasing hormones. Since the hypothalamus controls the pituitary gland, pituitary function is also inhibited, resulting in alterations in the normal circulating levels of gonadotropins, cortisol, growth hormone, and thyroid hormones. As a result, prepubertal patients may have altered sexual maturation and arrested physical development and growth patterns [77].

Hormonal problems are one of the most serious effects of AN. People with AN have decreased levels of reproductive hormones, including estrogen and DHEA. Estrogen is important for heart health and bone health. DHEA, a weak male hormone, is also important for bone health. For women, these hormonal abnormalities may result in menstrual cycle disruptions, including anovulation (lack of regular ovulation), oligomenorrhea, and amenorrhea [78, 79], and these abnormalities can occur even *before* a person has lost a significant amount of weight. Estrogen levels are usually restored and menses usually resume after a person has been treated and her weight has increased. However, in some cases, menstruation may never return, resulting in infertility.

Starvation and weight loss are known to create hypothalamic abnormalities that profoundly affect other organs within the endocrine system. A chain of interrelated events begins when the hypothalamus fails to signal the release of gonadotropin-releasing hormones from the pituitary. The absence of this signal causes a decrease in luteinizing hormone (LH) and follicular-stimulating hormone (FSH) levels and inhibits the positive feedback mechanism to the ovaries. Consequently, the ovaries do not release estrogen or progesterone in normal amounts, which further inhibits the pituitary gland. Ovarian volume

and uterine volume are decreased, and the vaginal mucosa becomes atrophic [65].

Normal functioning of the thyroid gland is also disrupted in individuals who have eating disorders. Individuals with AN frequently demonstrate thyroid abnormalities as a result of decreased calorie intake and starvation. Free thyroxine (free T4) decreases to low normal levels, whereas triiodothyronine (T3) levels decrease to abnormally low levels in proportion to the degree of weight loss [65], but thyroid-stimulating hormone (TSH) levels are usually within normal range [80]. Thyroid function tests reveal low T3 levels in proportion to weight loss, low normal T4 levels, and decreased metabolic rates [65].

#### 11.3.3.3 Mortality Rates

AN is recognized as having the highest mortality rate of any psychiatric condition in young females [15, 16, 18, 26, 81, 82]. Estimates of premature deaths in AN patients range from 5 to 6 % [7, 39]. Causes of death in women with EDs include starvation, suicide, and electrolyte imbalance [6, 12, 39, 83]. Several factors may be predictors of mortality, including having a body weight that is less than 77 lb, repeated inpatient admissions, and severe alcohol and substance use disorders [6, 84]. Adults with AN also have a high mortality rate [85, 86]. AN is also associated with elevated levels of suicide ideation and high rates of suicide [18, 83, 87].

### 11.3.4 Short-Term Adverse Effects of Bulimia Nervosa

Behaviors associated with BN may have few adverse consequences for individuals who briefly engage in self-induced vomiting, purging, or fasting [88]. However, when those behaviors are recurrent and persistent enough to lead to a diagnosis for BN, individuals are likely to have these physiological consequences: (1) erosion of the teeth, (2) enlargement of the parotid salivary glands, and (3) acidic stomachs leading to regurgitation of acidic stomach and heartburn [88, 89]. Signs and symptoms related to BN are listed in Table 11.4. This table is not all inclusive but lists some of the more common pathologies in BN.

**Table 11.4** Signs and symptoms of bulimia nervosa

| | |
|---|---|
| Anovulation | Hypotension |
| Calluses on back of hand and fingers | Integumentary |
| Cardiomyopathy | System |
| Cheilosis | Disorders |
| Constipation | Metabolic |
| Dental abscesses | Acidosis |
| Dental caries | Alkalosis |
| Diarrhea | Mitral valve prolapse |
| Dry, flaky skin | Muscle cramps |
| Dyspepsia | Musculoskeletal weakness |
| Endocrine disorders | Palpitations |
| Esophagitis | Pancreatitis |
| Heart failure | Pruitis |
| Hematemesis | Sore throat |
| | TetanY |

Excessive vomiting causes erosion on the enamel and dentin on teeth, increasing the susceptibility to cavities and gum disease [88, 89]. It can also cause acid from the stomach to rise up to the esophagus, which leads to infections, gastro-esophageal reflux disease, and may eventually cause a ruptured esophagus [89]. Other short-term complications resulting from BN include impaired satiety, decreased resting metabolic rate, and abnormal neuroendocrine responses. These symptoms increase in severity with continued disordered eating [90]. These complications should be thought of as occurring on a continuum, and in some cases, the symptoms are reversible.

### 11.3.5 Long-Term Adverse Health Effects of Bulimia Nervosa

#### 11.3.5.1 Gynecological Problems and Hormones

Gynecological problems are one of the most frequent long-term complications of EDs [91, 92]. The unsatisfactory nutrition in BN results in hormonal dysfunction, menstrual disturbances, and infertility [93]. These symptoms may be reversible with early treatment of the ED [91, 92]. Menstrual irregularities as a result of BN may be caused by weight fluctuations, nutritional deficiency, and

prolonged stress [94]. This same menstrual irregularity or oligomenorrhea can lead to polycystic ovary syndrome in individuals who are bulimic, especially if they are also obese [94, 95].

Gonadal steroids are among the many factors that influence food intake and body weight in mammals [97]. A key role of estradiol is related to food intake and energy balance. The actions of estradiol may have a gender-specific effect on the regulations of eating, which could explain why BN is more common in women than men [96]. During the estrogen-releasing cycle, the amount of food being consumed fluctuates in response to ovarian rhythms in bulimic women [97, 98].

Disturbances in hormonal regulation in BN can also lead to severe mood changes and aggressive behavior patterns. Researchers have found that individuals with BN have a decrease in plasma levels of prolactin and estradiol, and an increase in cortisol and testosterone [99, 100]. There is a positive correlation between testosterone plasma levels [101] and aggressiveness in individuals with BN that is not seen in other individuals [99, 100]. Individuals with BN tend to have a higher score when rating depressive symptoms and aggressiveness on eating-related psychopathology assessments, which suggests that BN plays a role in the modulation of aggressiveness [99].

#### 11.3.5.2 Cardiovascular Abnormalities

Hypercholesteremia (the presence of high levels of cholesterol in the blood) is a cardiovascular risk factor associated with BN. Hypercholesterolemia is not a disease but an abnormal metabolic state that can be secondary to many diseases and can also contribute to many forms of disease, most notably cardiovascular disease [102]. Pauporte and Walsh [103] found that the mean serum cholesterol levels of patients with BN were significantly higher than the cholesterol levels of individuals in a comparison group (patients: 194 ± 36 mg/dl; comparison group: 176 ± 34 mg/dl; $t = 2.77$; df = 159; $p = 0.006$). Additionally, individuals who binge or overeat, or who are obese, are also at high risk for developing hypertension, which is another pathway to long-term cardiovascular disease [103].

Mira et al. [104] found that individuals with BN and other EDs not only had higher levels of cholesterol, but they also had lower levels of electrolytes, such as, potassium, chloride, and phosphate in the plasma. The misuse of laxatives and weight-loss supplements over time can cause these electrolyte imbalances and gastrointestinal abnormalities [104].

Cardiac autonomic regulation and stress reactivity may also be altered in BN patients due to energy restriction. Altered eating patterns in BN can result in metabolic and cardiovascular abnormalities [105]. Messerli-Bürgy et al. [106] found that heart rate stress reactivity was highest in BN patients when looking at biological stress responses. During the stress recovery stage of the laboratory stressor, heart rate variability (HRV) decreased in the participants with BN compared to a group of other women [106]. A decrease in HRV is associated with coronary artery disease and congestive heart failure [107–111]. A similar study investigated cardiac autonomic regulation and stress reactivity in relation to biochemical markers of dietary restriction in women diagnosed with BN. These investigators found that women with BN who were fasting (compared to women who had BN but were not fasting or women who did not have BN) showed increased vagal dominance and decreased sympathetic modulation during both resting and recovery periods. These results support the notion of cardiac sympathetic inhibition and vagal dominance during dietary restriction, and suggest the specificity of starvation related to biochemical changes for cardiac autonomic control [169]. Vögele et al. [105] also found that individuals with BN have higher resting cardiac vagal tone than controls [105]. Based on the findings from their studies, Murialdo et al. [112]. hypothesized that BN patients have sympathetic failure, prevalent vagal activity, and impaired sympathetic activation. These findings indicate a relationship between energy restriction and vagal dominance [112].

Elevated homocysteine levels (an amino acid in the blood) are associated with cognitive decline in dementia and healthy elderly people and are also associated with a high risk of cardiovascular diseases, stroke, and peripheral vascular disease

[113, 114]. While elevated homocysteine levels are more common in AN than BN patients, BN patients also exhibit signs of elevated homocysteine levels [114, 115]. This condition can be caused by several conditions, such as malnutrition, starvation, alcohol abuse, or genetic predisposition [116]. Deficiencies of three vitamins—folic acid ($B_9$), pyridoxine ($B_6$), or cyanocobalamin ($B_{12}$)—can also lead to high homocysteine levels. Wilhem et al. [117] found a small decrease in levels of homocysteine following a 12-week treatment period for individuals with ED; however, the change was small and statistically nonsignificant. Nonetheless, their conclusion was that during effective treatment that concomitantly increased BMI, hyperhomocysteinemia was partially reversible. In light of the findings from Frieling et al. [114], decreasing homocysteine levels may not improve memory in an ED population. Interestingly, in a mixed group of patients (14 with AN and 12 with BN), elevated homocysteine levels were associated with normal short- and long-term verbal memory, and normal plasma homocysteine levels were associated with poorer memory performance. These results indicate that, under the special circumstances of ED, elevated homocysteine levels improve memory signaling, possibly by facilitating long-term potentiation.

#### 11.3.5.3 Immunologic Abnormalities

Individuals with BN may also have a comprised immune system. Several studies have reported changes in immune cells and natural killer cells important for immunity in patients with AN and BN [118]. With a decrease in lymphocyte number, individuals with BN are more vulnerable to disease.

### 11.3.6 Comorbidities and Mortality Rates for Bulimia Nervosa

BN is a long-term disorder with a waxing and waning course. Comorbid medical and psychiatric conditions associated with BN include: (1) irritable bowel syndrome; (2) fibromyalgia; (3) mood disorders, such as major depression; (4) anxiety disorders, such as generalized anxiety disorder, panic disorder, and phobias; (5) alcoholism and substance abuse, (6) personality disorders, and (7) aggressive behavior and poor impulse control [119]. These comorbid conditions are similar for BN and AN [119].

Recent data suggest that mortality rates for BN are around 3.9 % [120]. Mortality rates are slightly higher (5.2 %) for Eating Disorder Not Otherwise Specified (EDNOS), a disorder in which an individual's behavior may meet some but not all of the diagnostic criteria for BN [120].

## 11.4 Contemporary Understanding of the Issues

### 11.4.1 Genetic Variables and Eating Disorders

An area of interest in research is the role that genetics plays in EDs. Genetic research is attempting to explicate the behavioral, neurobiological, and temperamental variables that represent the core features of both the anorexic and bulimic phenotype. However, there is shared variance between genetic variables and other risk factors, such as an individual's environment or her attempts at dieting and losing weight.

The role of genetics in the etiology of EDs has long been postulated to be a risk factor based on information about the relatives and siblings of individuals with EDs. For example, individuals are more susceptible to developing an ED if a close relative also has an ED [121]. First-degree relatives of AN persons are six times more likely to develop AN [122]. Twins have a tendency to share specific patterns of ED symptoms, such as obesity, AN, or BN [123]. The compilation of several twin studies has indicated a 48–76 % heritability in AN and 50–83 % heritability for BN [16]. However, there is little statistically relevant evidence linking AN and heritability in twin studies [122]. The importance of genetic predisposition in BN is shown by the difference in concordance rates for monozygotic and dizygotic twins. The concordance rate for BN is 23 % for monozygotic twins and 9 % for dizygotic twins

[119, 123]. In other words, when one member of a twin pair has BN, the other twin is more likely to also have BN if the twins are identical genetically than if their genetic similarity is that of any other pair of siblings. The fact that the concordance rates for BN found in twin studies are nowhere near 100 % demonstrates clearly that many factors other than genetic predisposition contribute to BN.

The role of shared environmental influences must be considered in studies of twins and other siblings, given our knowledge of the importance of environment in the development of an ED. Evidence supports a strong association between genetically determined factors, such as serotonin (5-Hydroxytryptamine [5-HT]) and dopamine (DOP) levels, and environmental risk factors; suggesting that environmental risk factors play a large role in the expression of behaviors that are also genetically determined [124]. Epigenetics, changes in gene expression, is one factor of many that bridges the gap between genetics and environmental factors. Epigenetic modifications, including the methylation of deoxyribonucleic acid (DNA), can be influenced by various environmental factors, including stress and eating behavior. Methylation of the dopaminergic genes dopamine receptor D2 (DRD2) and dopamine active transporter 1 gene (DAT1) have been exhibited in patients with AN [122]. However, others investigators have noted only a small overlap between genetically determined and environmental risk factors. Thus, there is considerable independence between these two types of risk factors in the development of an eating disorder [26, 125–129].

The behavior components of BN, such as, self-induced vomiting, have also been found to be inheritable [130]. Some have suggested that overeating or behaviors consistent with BN are related to genetically determined, dysfunctional neurotransmitter systems [131].

It is clearly understood that genetics significantly contribute to the etiology and development of BN, however, details of genetic contributions to AN remain uncertain [132]. Recent studies have suggested that focusing on the level of severity of AN, instead of simply the presence of AN, when selecting subjects is beneficial when examining genetic contributions [128]. It is a challenge to understand the link between heritability and EDs because no certain gene(s) have been connected with eating disorder phenotypes [2]. It is also estimated that it is the small contributions of many genes instead of large contributions from a few genes that aid in the development of an ED [128]. An inherent limitation in the research methodology is the difficulty in linking the symptoms of both AN and BN to one single variable, such as genetics [132].

## 11.4.2 Neurotransmitters and Neuropeptides

### 11.4.2.1 Serotonin and Tryptophan

The ingestion of food produces chemical changes in the brain that cause a variety of neurochemical responses throughout the body. Specific hormones are released to create instructional pathways for neural communication. Tryptophan is an essential amino acid that is found in many common foods, such as nuts, meats, and dairy products, and is a precursor for serotonin (5-HT). Therefore, dietary deficiency of tryptophan may lead to low levels of 5-HT. Low levels of tryptophan and 5-HT are commonly seen in individuals with psychological disorders, such as depression, AN and BN [132, 133]. A decrease in 5-HT can contribute to the abnormal eating patterns seen in individuals with BN by interfering with the homeostatic regulation of eating by the hypothalamus [134].

Serotonin is a monoamine neurotransmitter that helps the body regulate appetite, sleep patterns, and mood. As stated previously, 5-HT is biochemically derived from tryptophan and is primarily found in the gastrointestinal tract, platelets, and central nervous system of humans and animals. Regulation of serotonin is important in the pathophysiology of an ED [135, 136]. Serotonin is responsible for regulation or involvement in some of the main functions of the central nervous system, such as: control of mood, appetite, sleep, muscle contraction, pain sensitivity, blood pressure, and some cognitive functions including memory and learning [137, 138].

Serotonin transports, especially the serotonin transport protein 5-HTT (or SERT), are considered good markers for gene studies focused on eating disorders [139]. Serotonin is involved in the etiology of AN by altering physiological and behavioral functions that affect mood, impulse regulation and appetite [139]. Serotonin also influences the hyperactivity associated with AN [132]. Serotonin is likely involved in the etiology of BN by modulating physiological and behavioral functions including anxiety, perception, and appetite [140].

#### 11.4.2.2 Neural Signaling Response to Food Consumption

The neural signaling that occurs in response to food consumption is a link in the feedback mechanisms that normally keep carbohydrate and protein intake more or less constant [141]. Carbohydrate consumption causes insulin secretion which also increases 5-HT release, whereas the consumption of protein lacks this effect on insulin. The consumption of carbohydrates causes the secretion of insulin from the pancreas into the blood, reducing plasma levels of glucose and allowing the uptake of tryptophan in the brain. Tryptophan enhances 5-HT release and also increases the saturation of tryptophan hydroxylase [136, 141]. Hydroxylase is the enzyme responsible for 5-HT synthesis. When BN patients are given a pharmacological stimulus for the production of 5-HT (serotonin-stimulated prolactin secretion), the number of their eating binges decreases [134].

Other investigators have suggested that protein should be added to the diet of BN patients in order to reduce binge eating [142]. Wurtman and Wurtman [136] reported that individuals whose eating binges consist of primarily protein have fewer eating binges than those whose eating binges consist primarily of carbohydrates [136]. In that study, individuals with BN reported less hunger and greater fullness, and consumed less food at test meals, after protein intake than after carbohydrate intake (673 kcal vs. 856 kcal). This discrepancy between protein and carbohydrate consumption during eating binges deserves attention in future research.

#### 11.4.2.3 Receptor Subtypes for Serotinin

The pharmacology of 5-HT is extremely complex, with its actions being mediated by a large and diverse range of 5-HT receptors. At least seven different receptor subtypes (5-HT1–7) are known to exist, each located in different parts of the body and triggering different responses. Serotonin receptors include: 5HT1Dβ, 5HT2A, 5HT2C, and 5-HT7 tryptophan hydroxylase 1. Associations between a functional variant in the 5-HT transporter gene have been found with other psychiatric symptoms such as depression, alcoholism, and suicidal behavior [143]. Alleles are different forms of the DNA sequence of a particular gene. By conferring the allele-specific transcriptional activity on the 5-HT transporter gene promoter in humans, it has been found that the 5-HT transporter gene-linked polymorphic region (5-HTTLPR-a serotonin-transporter-linked promoter region) influences a constellation of personality traits related to anxiety and increases the risk for neurodevelopmental, neurodegenerative, and psychiatric disorders [144]. The S, G, and A alleles have been implicated in the transmission of an ED from mother to child [145]. It has been hypothesized that alterations in the S-allele contributes to the pathophysiology of AN and binge eating [139, 146]. Simply carrying the S-allele increases risk of AN and binge eating [139, 147].

Particularly the 5-HTTLPR (serotonin-transporter-linked promoter region) S-allele has been linked to AN, high anxiety and low levels of impulsiveness in some studies [139]. A study by Akkermann et al. [147], investigated the association between the 5-HTTLPR and binge eating to determine if the 5-HTTLPR genotype influenced the severity of binge eating. Women prone to binge eating and carrying the S-allele showed significantly higher levels of BN scores. Among these women, those with s/s genotype also had higher levels of state anxiety and a tendency for higher impulsivity [147].

Not all researchers are in agreement about the relationship of the S-allele and the pathophysiology of BN. Lee [148] found that overall EDs were significantly associated with the S-allele

and genotype, but a meta-analysis led to the conclusion that while AN was associated with the S-allele and the S carrier genotype, BN was not associated with this allele [148]. Racine et al. [149] found that the T-allele and the S-allele gene were associated with higher levels of impulsivity, but there were no main effects for the 5-HT genotypes on any binge eating measure, and interaction between genotypes, impulsivity, and dietary restraint were nonsignificant [149].

#### 11.4.2.4 Dopamine

Another important neurotransmitter (neural messenger) that merits discussion in the pathophysiology of both AN and BN is dopamine (DOP). Dopamine is classified as a catecholamine (a class of molecules that serve as neurotransmitters and hormones). Dopamine is a precursor (forerunner) of adrenaline and another closely related molecule, noradrenaline. Central DOP mechanisms are involved in the reward and motivational aspects of eating and food choices, and they play a role in the compulsive feeding patterns observed in BN and purging disorders [150]. Foods high in fats and sugars are likely to promote DOP stimulation [151]. It has been hypothesized that deficiencies in DOP may promote reward-seeking behaviors that result in instant gratification such as carbohydrate eating binges [152–154]. The role of DOP in the pathophysiology of AN remains unclear. Some studies have shown reduced DOP levels in certain regions of the brain (the hippocampus, dorsal striatum, and hypothalamus) to be associated with starvation/food restriction in AN [155]. Also, DOP can contribute to the hyperactive characteristic in AN patients via increased concentrations of DOP in the hypothalamus [155]. However, the complexity of the body's systems and number of different pathways and receptors make targeting the role/concentration of DOP in the pathophysiology of both AN and BN difficult.

#### 11.4.2.5 Catechol-*O*-methyltransferase

Although not classified as a neurotransmitter, catechol-*O*-methyltransferase (COMT) is an important protein in the degradation of DOP and other catecholamines in the brain, so it deserves attention in the discussion of eating disorders. It is one of several enzymes that degrade catecholamines such as DOP, epinephrine, and norepinephrine. Dysregulation of DOP has been implicated in many genetic studies related to BN [150, 151].

The COMT gene lies in a chromosomal region that is of interest in investigations of psychosis and mood disorders [145]. In particular, regions on chromosome 10 have been linked to BN and obesity [156]. However, despite a considerable research effort, a clear relationship between the genetic variation in specific chromosomes and the psychiatric phenotype has not been substantiated [157].

### 11.4.3 Peptides and Proteins

Individuals with an ED are less sensitive to the satiating effects of food [158, 159]. For example, after eating BN patients report lower subjective ratings of fullness than other individuals [159]. Ample evidence supports the notion that individuals with BN have a disturbance in satiation, which helps to explain the consumption of very large amounts of food that is recorded during binge meals in laboratory settings [159–163].

There are several specific physiological mechanisms that help to explain the deficit in the normal development of satiation when individuals with an ED consume food. Peptide signals from surrounding tissues communicate with the hypothalamus to control hunger and eating behavior [122]. Abnormal levels of leptin, ghrelin (the satiety peptide), cholecystokinin (CCK), and androgens have all been implicated as playing a role in food intake, satiety signaling and binge-eating behavior [164].

#### 11.4.3.1 Leptin

Leptin is a protein hormone that plays a key role in regulating energy intake and energy expenditure, including appetite and metabolism. It is one of the most important adipose-derived hormones [165]. Certain levels of leptin must exist in order to support menstruation; consequently low leptin levels have been linked with amenorrhea.

Low leptin levels have also been associated with AN [166, 167]. Some studies have found that individuals with BN have low levels of serum leptin [112, 168–170]. However, one study found that leptin concentrations were significantly higher in patients with BN than they were for individuals in a comparison group [171]. There is also no consensus among researchers examining ED patients about whether plasma levels of leptin are significantly related with patients' body weight or BMI [172–177].

Ghrelin is a hormone that stimulates hunger that is produced mainly by P/D1 cells lining the fundus of the human stomach and by the epsilon cells of the pancreas. Ghrelin levels increase before meals and decrease after meals. It is considered the counterpart to the hormone leptin, produced by adipose tissue, which induces satiation when present at higher levels. Both acute and chronic fasting increase ghrelin levels [122, 178]. Weight loss brought about by dieting causes ghrelin levels to rise as body weight and body fat decline. Ghrelin may blunt the appetite-reducing effect of leptin [179].

#### 11.4.3.2 Ghrelin

Because ghrelin levels increase before meals, it would be expected that ghrelin levels are higher in individuals in the acute stage of AN. Research has found this to be true. The effects of ghrelin are unclear in those who chronically suffer from AN. However, research has shown that ghrelin levels drop in individuals with AN who are receiving treatment. This information could explain why AN patients are even more resistant to food during treatment, making the recovery process very difficult [30].

It has been suggested that individuals with BN have high ghrelin levels [180]. Supporting the hypothesis that individuals with BN have high ghrelin levels, Kojima et al. [180]. found that patients with BN exhibit elevated ghrelin levels before meals and reduced ghrelin suppression after eating. They found that postprandial ghrelin suppression was significantly attenuated in patients with BN compared to individuals who did not have BN [180]. Monteleone et al. [179] also found that the ghrelin levels of individuals with BN did not decrease as much as would be expected after a meal. In healthy women, circulating ghrelin showed a drastic decrease after food intake, whereas this response was significantly blunted for individuals with BN. The blunted ghrelin response to food ingestion for individuals with BN may explain the impaired suppression of the drive to eat following a meal, which can lead to binge eating [179]. Elevated ghrelin levels have all been found to decrease significantly after treatment, despite similar BMI, percent body fat, and leptin levels [169]. When ghrelin levels return to normal for an individual with an ED, abnormal eating behavior and depressive symptoms both improve [169].

#### 11.4.3.3 Cholecystokinin

Cholecystokinin (CCK) is a peptide hormone of the gastrointestinal system responsible for stimulating the digestion of fat and protein. It also acts as a hunger suppressant and contributes to the feeling of satiation [181]. Individuals with BN have a reduced level of postprandial CCK compared to individuals who do not have EDs [160, 182–185].

The development of CCK and satiety has been greatly explored in BN, including gastric capacity, gastric emptying, gastric relaxation reflex and the postprandial release of CCK [41]. A significant enlarged gastric capacity has been found in women with BN compared to non-BN women [186]. This suggests that a larger amount of food must be consumed before the development of gastric signals. Along with this gastrointestinal abnormality, gastric emptying has found to be delayed in women with BN [182, 187–189]. As a result of this irregularity, there may be a delay in the development of satiety cues that result from the presence of food in the intestine. Finally, another gastrointestinal problem that arises with BN is that there is a reduced gastric relaxation occurring following food ingestion [190].

#### 11.4.3.4 Brain-Derived Neurotrophic Factor

Lastly, certain proteins, such as brain-derived neurotrophic factor (BDNF), have been implicated in the etiology of an ED. This protein may

influence an individual's vulnerability to AN and BN [156] via regulation of apatite control [122]. Specifically, the genetic contribution of the BDNF-specific receptor neurotrophic tyrosine kinase receptor type 2 (NTRK2), is implicated in the susceptibility of developing an ED [191]. In most candidate gene association studies (CGASs), decreased levels of BDNF are typically present in those with AN [122].

It is important to note the difficulty in linking protein levels to genetic traits associated with an eating disorder. If protein levels return to normal with restored weight and proper nutrition, the disturbed protein levels may be related to improper nutrition rather than the traits associated with the eating disorder [122].

## 11.5 Future Directions

### 11.5.1 Anorexia Nervosa

> The treatment of patients with anorexia nervosa is both a science and an art. The science deals with the physical aspects that resulted from undernutrition, and the art deals with the person in whom the disorder exists [47].

Individuals with AN frequently lack insight into their problems and often deny the existence of problems related to eating. They are often reluctant to seek help from friends, family members, or health professionals, because the eating disorder becomes a lifestyle and they fear changing their habits and gaining weight. When they do seek help on their own, it may be due to severe distress over physical or psychological problems that occur as a result of the eating disorder or in conjunction with the eating disorder. In an attempt to conceal their disorder from health professionals, individuals with AN may try to hide signs of this disorder or might provide inaccurate information to the clinician [47]. For example, an individual with AN might drink a lot of water prior to being weighed by a professional or might hide weights in her clothing to increase the number on the scale.

Treatment must be specific to each individual because of the different levels of severity and because of the unique characteristics each individual with AN develops [47]. Effective treatment of individuals with AN should include weight restoration and restoring healthy eating habits. However, successful treatment of AN requires more than a focus on eating and weight gain. Focus on emotional issues that are related to the disorder and family conflicts that contribute to the disorder are also needed [47].

Successful treatment depends on the individual with AN gaining weight and maintaining a normal weight and adequate nutrition. Initially in treatment, the focus is on supporting the individual with AN and building a cooperative relationship with her while she gains weight. Because of the cognitive impairments resulting from semi-starvation, it will be difficult to deal with emotional and interpersonal problems until the individual's weight returns to the normal range [47]. Sometimes, however, the focus on weight gain may be too narrow, so that the person gains weight during treatment, but has not accepted that weight gain or changed her attitudes and perceptions related to weight and eating. Many patients with anorexia gain weight in treatment but then lose it soon after leaving treatment. Also, some treatment programs focus on rapid weight gain, which will be difficult for the individual with anorexia both psychologically and physically, and may expose the individual to some serious health risks, such as heart failure [47].

Weight restoration must be done gradually and patiently. Additionally, returning the individual with AN to a normal pattern of eating can be either easily accomplished or extremely difficult, depending on how long the disorder has persisted. She may rebel against a 2,000–3,000-cal diet because she will feel as though she is being overfed and may therefore also stop cooperating with other aspects of treatment. Therefore, enlisting the help of a dietitian can assist in educating the person about her nutritional needs. However, including too many professionals into the treatment might pose problems for the person being treated for AN, so an ideal approach might be for the therapist to work closely with the nutritionist [47]. There are also physical reasons that she will be unable to resume a normal diet immediately.

Attempting rapid weight gain in a person who has been starving may lead to excessive fluid retention with a risk of heart failure. In addition, any nourishment may be difficult for her because of her empty and shrunken stomach. So, eating may trigger nausea and vomiting, and these physical responses must be carefully distinguished from common psychological variables, such as revulsion at food and self-induced vomiting [47]. Without early, aggressive intervention, AN will most likely last for several years, and it may persist or reoccur throughout the individual's life [47]. Long-term follow-up studies reveal a mortality rate as high as 18 %, with the majority of deaths related to medical complications of the disorder [39].

Current research is focusing on the use of ghrelin agonists/antagonists and the treatment of eating disorders; however, most studies are animal based studies and only few studies have used human subjects in regards to AN. For example, ghrelin antagonists have been found to reduce the hyperactivity associated with AN but may cause appetite depression. Therefore, treatment with ghrelin antagonists is controversial. Ghrelin agonists are a possible treatment option for AN-R due to the resultant increase in food intake seen in human subjects. Yet, larger studies using human subjects are needed to clearly understand the relationship between ghrelin and AN-R and more studies need to focus on ghrelin and the AN-BP subtype [30]. Serotonin is also being considered for the use of AN treatment [132].

### 11.5.2 Bulimia Nervosa

There are many effective treatment options for individuals with BN, such as pharmacology (most commonly anti-depressant medications) [192], psychological treatment, therapeutic exercise such as yoga, and behavioral modification. Cognitive behavior therapy is effective in reducing the symptoms of BN and BED [193]. However, even though improvement over a short-term period is commonly found in the research literature, treatment may have a more limited effect over the longer term [194]. Vigilance is needed in helping girls and women to have healthy eating patterns and to avoid BN and other EDs in a culture that places so much emphasis on physical appearance and has such unrealistic ideals regarding the weight and shape of the human body.

It would be great if EDs could be prevented from occurring (primary prevention), but that goal seems unattainable based on the research on past prevention programs. Efforts to prevent EDs shave produced temporary results, a change in knowledge but no change in attitudes or behavior, or an increase in symptoms of eating disorders [195–197]. Unfortunately, prevention efforts can lead girls and young women to focus even more than they had before on their bodies and on dieting, and may promote unhealthy behaviors among especially vulnerable girls [47]. Another reason that primary prevention is so difficult is that any prevention programs are unlikely to have as much of an effect on girls and young women as the influence of their peers and of media messages. Therefore, the best type of prevention for an ED may be secondary prevention—identifying the early signs of trouble and starting treatment as soon as possible.

### 11.5.3 Secondary Prevention and Education

Health professionals must be educated about the dangers and warning signs of EDs to promote early recognition, evaluation, and treatment. Parents, teachers, and coaches who recognize common signs of an eating disorder in girls or young women should express their concerns to these individuals and their parents and should also encourage them to seek further evaluation. Because individuals often develop an eating disorder in the aftermath of a diet, overweight individuals should be encouraged to lose weight through nutritionally balanced meals and exercise rather than by strict dieting that can trigger binge eating and purging cycles [49]. Health professionals must develop realistic attitudes about body weight and shape in order to communicate information effectively and to promote appropriate preventive efforts.

Early diagnosis of an ED is related to a better prognosis because the patient is more receptive to treatment. Earlier diagnosis is an important first step for many patients and allows for intervention before the adverse eating patterns are ingrained due to repetition [85]. Vigilant friends and family can notice signs and symptoms of the ED and attempt to seek proper help. If an ED is suspected, one of the most practical screening tools to use in the primary care setting is the SCOFF questionnaire [198]. Because of its 12.5 % false-positive rate, this test is not sufficiently accurate for diagnosing eating disorders, but it is an appropriate screening tool that physicians can use as a first step in identifying and treating the ED. Although a substantial amount of progress has been made in the field of eating disorder research and treatment, there are still many questions without answers. By reading this book, you will see where we are on the journey towards better understanding and treating eating disorders.

## 11.6 Concluding Remarks

The key feature of AN is the refusal of the girl or woman to eat an adequate amount of food [47]. BN is an eating disorder that involves binge eating and the use of inappropriate methods to avoid weight gain [199]. There are also shared qualities, as more than half of patients diagnosed with AN-R crossover to AN-BP and approximately one-third crossover to BN during the first 5 years of being diagnosed with an eating disorder [200].

All of the physiological changes that occur in AN are caused by malnutrition or "semi-starvation" [47]. Those changes are the adaptive responses of the body to survive despite inadequate intake of food: conservation of energy, shifts in electrolyte balances, attempts to use fat and spare the body's glucose and protein, and changes in the functioning of the hypothalamus and the pituitary gland.

With fluctuating eating patterns, individuals with BN are at risk for developing cardiovascular health problems [105] such as coronary artery disease, hypertension, and congestive heart failure [107–111]. Many other adverse health conditions are also associated with the disorder such as alcoholism, panic disorder, generalized anxiety disorder, phobia, and major depression [119]. Two of the most prevalent co-occurring conditions for individuals with BN are anxiety and depression [201].

Both genetics and environmental factors (culture and family) play large roles in the behavioral, neurobiological, and temperamental variables that represent the core features of ED development. The family environment is especially important in the development of an ED, since adolescence is a particularly vulnerable age for females [199].

The psychological and physiological aspects of BN are often tightly linked [202]. Biomarkers associated with BN include, but are not limited to the dysregulation of hormones that contribute to irregular dieting behaviors, possibly through serotonergic mechanisms [41]. Alterations in 5-HT and DOP can result in the dysregulation of mood, satiety, appetite, sleep, muscle contraction, and some cognitive functions including memory and learning [137, 138]. Research does not indicate a direct relationship between pathophysiological markers and the diagnosis of AN [128].

The initial evaluation of an individual with an eating disorder must include a comprehensive physical exam and health history to rule out existing physiological pathology. Several lab tests, including a complete blood count (i.e., full blood chemistry, electrolyte profile, liver and function tests, and urinalysis) should also be performed. An EKG is essential to evaluate the cardiovascular system and to rule out potentially life-threatening arrhythmias, and a chest X-ray may be performed to evaluate heart size and placement [63].

A number of long-term complications may result from the prolonged and severe malnutrition that often accompanies eating disorders. Medical complications can be expected to progress as long as the individual continues to exercise without proper nutritional intake [64]. Inadequate nutritional intake and poor absorption of nutrients result in physical consequences, including extreme weight loss, electrolyte imbalances,

cardiac abnormalities, hormonal changes, central nervous system abnormalities, bone loss, and muscle wasting. Unfortunately, these physical consequences can result in death; therefore, adequate nutritional intake and weight restoration are vital in the treatment of an ED [39].

## References


1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed. Washington, DC: American Psychiatric Association; 2000. Text Revision.
2. Easter M. "Not all my fault": genetics, stigma, and personal responsibility for women with eating disorders. Soc Sci Med. 2012;75:1408–16.
3. Wilfley DE, Bishop ME, Wilson GT, Agras WS. Classification of eating disorders: toward DSM-V. Int J Eat Disord. 2007;40:S123–9.
4. Fairburn CG, Cooper Z, Bohn K. The severity and status of eating disorder NOS: implications for DSM-V. Behav Res Ther. 2007;45:1705–15.
5. Keel PK. Purging disorder: subthreshold variant or full-threshold eating disorder? Int J Eat Disord. 2007;40:S89–94.
6. Berkman ND, Lohr KN, Bulik CM. Outcomes of eating disorders: a systematic review of the literature. Int J Eat Disord. 2007;40:293–309.
7. Herpertz-Dahlmann B. Adolescent eating disorders: definitions, symptomatology, epidemiology and comorbidity. Child Adolesc Psychiatr Clin N Am. 2008;18:31–47.
8. Hudson JI, Hiripi E, Pope HG, Kessler RC. The prevalence and correlates of eating disorders in the national comorbidity survey replication. Biol Psychiatry. 2007;61:348–58.
9. Cassin SE, von Ranson KM. Personality and eating disorders: a decade in review. Clin Psychol Rev. 2005;25:895–916.
10. Vitousek K, Manke F. Personality variables and disorders in anorexia nervosa and bulimia nervosa. J Abnorm Psychol. 1994;103:137–47.
11. Shaw H, Stice E, Becker CB. Preventing eating disorders. Child Adolesc Psychiatr Clin N Am. 2008;18:199–207.
12. Hoek HW. Incidence, prevalence and mortality of anorexia nervosa and other eating disorders. Curr Opin Psychiatry. 2006;19:389–94.
13. Pearson J, Goldklang D, Striegel-Moore RH. Prevention of eating disorders: challenges and opportunities. Int J Eat Disord. 2002;31:233–9.
14. Van Hoeken D, Seidell J, Hoek H. Epidemiology. In: Treasure J, Schmidt U, van Furth E, editors. Handbook of eating disorders. 2nd ed. Chichester, UK: Wiley; 2003. p. 11–34.
15. Stice E, Shaw H, Marti CN. A meta-analytic review of eating disorder prevention programs: encouraging findings. Annu Rev Clin Psychol. 2007;3:207–31.
16. Striegel-Moore RH, Bulik CM. Risk factors for eating disorders. Am Psychol. 2007;62:181–98.
17. Bulik CM, Reba L, Siega-Riz AM, Reichborn-Kjennerud T. Anorexia nervosa: definition, epidemiology, and cycle of risk. Int J Eat Disord. 2005; 37:s2–9.
18. Stice E, Wonderlich S, Wade E. Eating disorders, Chapter 20. In: Hersen M, Thomas JC, Ammerman RT, editors. Comprehensive handbook of personality and psychopathology. Hoboken, NJ: Wiley; 2006. p. 330–47.
19. Mangweth-Matzek B, Rupp CI, Hausmann A, et al. Never too old for eating disorders or body dissatisfaction: a community study of elderly women. Int J Eat Disord. 2006;39:583–7.
20. Treasure J. Eating disorders. Medicine. 2008;36: 430–5.
21. O'Brien KM, Vincent NK. Psychiatric comorbidity in anorexia and bulimia nervosa: nature, prevalence and causal relationships. Clin Psychol Rev. 2003;23:57–74.
22. Tozzi F, Thornton LM, Klump KL, et al. Symptom fluctuation in eating disorders: correlates of diagnostic crossover. Am J Psychiatry. 2005;162:732–40.
23. Striegel-Moore RH, Dohm FA, Kraemer HC, et al. Eating disorders in white and black women. Am J Psychiatry. 2003;160:1326–31.
24. Uyeda L, Tyler I, Pinzon J, Birmingham CL. Identification of patients with eating disorders. The signs and symptoms of anorexia nervosa and bulimia nervosa. Eat Weight Disord. 2002;7:116–23.
25. Treasure JL. Getting beneath the phenotype of anorexia nervosa: the search for viable endophenotypes and genotypes. Can J Psychiatry. 2007;52: 212–9.
26. Bulik CM, Slof-Op't Landt MC, van Furth EF, Sullivan PF. The genetics of anorexia nervosa. Annu Rev Nutr. 2007;27:263–75.
27. Jansen A, Smeets T, Martijn C, Nederkoorn CT. I see what you see: the lack of a self-serving body-image bias in eating disorders. Br J Clin Psychol. 2006;45: 123–35.
28. Lilenfeld LRR, Wonderlich S, Lawrence RP, Crosby R, Mitchell J. Eating disorders and personality: a methodological and empirical review. Clin Psychol Rev. 2006;26:299–320.
29. McKnight Investigators. Risk factors for the onset of eating disorders in adolescent girls: results of the McKnight longitudinal risk factor study. Am J Psychiatry. 2003;160:248–54.
30. Cardona Cano S, Merkestein M, Skibicka KP, Dickson SL, Adan RAH. Role of ghrelin in the pathophysiology of eating disorders. CNS Drugs. 2012;26(4):281–96.
31. Garfinkel P, Lin E, Goering P, Spegg C. Should amenorrhea be necessary for the diagnosis of anorexia nervosa? Evidence from a Canadian community sample. Br J Psychiatry. 1996;168:500–6.
32. Cachelin F, Maher B. Is amenorrhea a critical criterion for anorexia nervosa? J Psychosom Res. 1998;44:435–40.

33. Striegel-Moore RH, Marcus MD. Eating disorders in women: current issues and debates. In: Stanton AL, Gallant SJ, editors. The psychology of women's health: progress and challenges in research and application. Washington, DC: American Psychological Association; 2005. p. 445–87.
34. Wonderlich SA, Joiner Jr TE, Keel PK, Williamson DA, Crosby RD. Eating disorder diagnoses. Am Psychol. 2007;62:167–80.
35. Gordon K, Holm-Denoma J, Smith A, Fink E, Joiner T. Taxometric analysis: introduction and overview. Int J Eat Disord. 2007;40:S35–9.
36. World Health Organization. International statistical classification of diseases and related health problems. 10th ed. Geneva, Switzerland: World Health Organization; 1992.
37. Karwautz A, Rabe-Hesketh S, Hu X, et al. Individual-specific risk factors for anorexia-nervosa: a pilot study using a discordant sister-pair design. Psychol Med. 2001;31:317–29.
38. Bornstein RF. A meta-analysis of the dependency-eating-disorders relationship: strength, specificity, and temporal stability. J Psychopathol Behav Assess. 2001;23:151–62.
39. Steinhausen HC. The outcome of anorexia nervosa in the 20th century. Am J Psychiatry. 2002;159:1284–93.
40. Fassino S, Amianto F, Gramaglia C, Facchini F, Abbate Daga G. Temperament and character in eating disorders: ten years of studies. Eat Weight Disord. 2004;9:81–90.
41. Klein DA, Walsh TB. Eating disorders: clinical features and pathophysiology. Physiol Behav. 2004;81:359–74.
42. Reba-Harrelson LA, Von Holle LM, Thronton KL, et al. Features associated with diet pill used in individuals with eating disorders. Eat Behav. 2008;9:73–81.
43. Polivy J, Herman CP. Causes of eating disorders. Annu Rev Psychol. 2002;53:187–213.
44. Fichter MM, Quadflieg N, Hedlund S. Twelve-year course and outcome predictors of anorexia nervosa. Int J Eat Disord. 2005;39:310–22.
45. Wonderlich SA, Lilenfeld LA, Riso LP, Engel S, Mitchell JE. Personality and anorexia nervosa. Int J Eat Disord. 2005;37:s68–71.
46. Sharp CW, Freeman CPL. Medical complications of anorexia nervosa. Br J Psychiatry. 1993;162: 452–62.
47. Lucas AR. Demystifying anorexia nervosa: an optimistic guide to understanding and healing. New York: Oxford University Press; 2004.
48. Treasure J, Szmukler G. Medical complications of chronic anorexia nervosa. In: Szmukler G, Dare C, Treasure J, editors. Handbook of eating disorders: theory, treatment and research. New York: Wiley; 1995. p. 197–220.
49. Rock CL, Zerbe KJ. Keeping eating disorders at bay. Patient Care. 1995;11:78–86.
50. Gavish D, Eisenerg S, Berry EM, et al. Bulimia: an underlying behavioral disorder in hyperlipidemic pancreatitis: a prospective multidisciplinary approach. Arch Intern Med. 1987;147:705–8.
51. McClain CJ, Humphries LL, Hill KK, Nicki NJ. Gastrointestinal and nutritional aspects of eating disorders. J Am Coll Nutr. 1993;12:466–74.
52. Mitchell JE, Boutacoff LI, Hatsukami D, Pyle RL, Eckert ED. Laxative abuse as a variant of bulimia. J Nerv Ment Dis. 1986;174:174–6.
53. Mitchell JE, Hatsukami D, Pyle RL, Ekert ED, Boutacoff LI. Metabolic acidosis as a marker for laxative abuse in patients with bulimia. Int J Eat Disord. 1987;6:557–60.
54. Schwartz DM, Thompson MG. Do anorexics get well? Current research and future needs. Am J Psychiatry. 1981;138:319–23.
55. Pfeiffer RJ, Lucas AR, Ilstrup DM. Effect of anorexia nervosa on linear growth. Clin Pediatr. 1986;25:7–12.
56. Biller BM, Saxe KV, Herzog DB, Rosenthal DI, Holzman S, Klibanski A. Mechanisms of osteoporosis in adult and adolescent women with anorexia nervosa. J Clin Endocrinol Metab. 1989;68:548–54.
57. Hay PJ, Delahunt JW, Hall A, Mitchell AW, Harper G, Salmond C. Predictors of osteopenia in premenopausal women with anorexia nervosa. Calcif Tissue Int. 1992;50:498–501.
58. Kiriike N, Iketani T, Nakanishi S, et al. Reduced bone density and major hormones regulating calcium metabolism in anorexia nervosa. Acta Psychiatr Scand. 1992;86:358–63.
59. Mitchell JE. Medical complications of anorexia nervosa and bulimia. Psychiatr Med. 1983;1: 229–55.
60. Grinspoon S, Thomas E, Pitts S, et al. Prevalence and predictive factors for regional osteopenia in women with anorexia nervosa. Ann Intern Med. 2000;133:828–30.
61. Herzog W, Deter HC, Schellberg D, et al. Somatic findings at 12-year follow-up of 103 anorexia nervosa patients: results of the Heidelberg-Mannheim follow-up. In: Herzog W, Deter HC, Vandereycken W, editors. The comprehensive medical text. Berlin: Springer; 1992. p. 85–107.
62. Rigotti NA, Neer RM, Skates SJ, Herzog DB, Nussbaum SM. The clinical course of osteoporosis in anorexia nervosa: a longitudinal study of cortical bone mass. JAMA. 1991;265:1133–8.
63. Garner DM. Pathogenesis of anorexia nervosa. Lancet. 1993;341:1631–5.
64. Katz JL. Eating disorders. In: Shangold M, Mirkin G, editors. Women and exercise: physiology and sports medicine. 2nd ed. Philadelphia, PA: F.A. Davis; 1995. p. 292–312.
65. Carne CP, Andersen AE. Eating disorders: guide to medical evaluation and complications. Psychiatr Clin N Am. 1996;19:657–79.
66. Palazidou E, Robinson P, Lishman WA. Neuroradiological and neuropsychological assessment in anorexia nervosa. Psychol Med. 1990;20:521–7.
67. Husain MM, Black KJ, Doraiswamy PM, et al. Subcortical brain anatomy in anorexia and bulimia. Biol Psychiatry. 1992;31:735–8.

68. Kreig JC, Holthoff V, Schreiber W, Pirke KM, Herholz K. Glucose metabolism in the caudate nuclei of patients with eating disorders, measured by PET. Euro Arch Psychiatry Clin Neurosci. 1991;240:331–3.
69. Ekeus C, Lindberg L, Lindblad F, Hjern A. Birth outcomes and pregnancy complications in women with a history of anorexia nervosa. BJOG. 2006;113:925–9.
70. Russell GF, Treasure J, Eisler I. Mothers with anorexia nervosa who underfeed their children: their recognition and management. Psychol Med. 1998;28:93–108.
71. Hall RC, Hoffman RS, Beresford TP, et al. Physical illness encountered in patients with eating disorders. Psychosomatics. 1989;30:174–91.
72. Fohlin L. Body composition, cardiovascular and renal function in adolescent patients with anorexia nervosa. Acta Paediatr Scand Suppl. 1977;268:1–20.
73. Warren MP, van de Wiele RL. Clinical and metabolic features of anorexia nervosa. Am J Obstet Gynecol. 1973;117:435–49.
74. De Simone G, Scalfi L, Galderisi M, et al. Cardiac abnormalities in young women with anorexia nervosa. Br Heart J. 1994;71:278–92.
75. Hall RCW, Beresford TP. Medical complications of anorexia and bulimia. Psychiatr Med. 1989;7: 165–92.
76. Schocken DD, Holloway JD, Powers PS. Weight loss and the heart: effects of anorexia nervosa and starvation. Arch Intern Med. 1989;149:877–81.
77. American Psychiatric Association. Practice guidelines for eating disorders. Am J Psychiatry. 1993; 150:212–8.
78. Fairburn CG. Physiology of anorexia nervosa, Chapter 44. In: Brownell KD, Fairburn CG, editors. Eating disorders and obesity: a comprehensive handbook. New York: Guilford; 1995. p. 251–4.
79. Stewart DE, Robinson E, Goldbloom DS, Wright C. Infertility and eating disorders. Am J Obstet Gynecol. 1990;163:1196–9.
80. Schwabe AD, Lippe BM, Chang RJ, Pops MA, Yager J. Anorexia nervosa. Ann Intern Med. 1981;94:371–81.
81. Kaye WH, Bulik CM, Plotnicov K, et al. The genetics of anorexia nervosa collaborative study: methods and sample description. Int J Eat Disord. 2008; 41:289–300.
82. Klump KL, Bulik CM, Kaye WH, Treasure J, Tyson E. Academy for eating disorders position paper: eating disorders are serious mental illnesses. Int J Eat Disord. 2009;42:97–103.
83. Birmingham LC, Su J, Hlynsky JA, Goldner EM, Gao M. Mortality rates from anorexia nervosa. Int J Eat Disord. 2005;38:143–6.
84. Patton GC. Mortality in eating disorders. Psychol Med. 1988;18:947–51.
85. Gowers S, Bryant-Waugh R. Management of child and adolescent eating disorders: the current evidence base and future directions. J Child Psychol Psychiatry. 2004;45:63–83.
86. Nielsen S, Moller-Madsen S, Isager T, et al. Standardized mortality in eating disorders: a quantitative summary of previously published and new evidence. J Psychosom Res. 1998;44:412–34.
87. Herzog DB, Greenwood DN, Dorer DJ, Flores AT, Richards ER. Mortality in eating disorders: a descriptive study. Int J Eat Disord. 2000;28:20–6.
88. Roberts MW, Tylenda CA. Dental aspects of anorexia and bulimia nervosa. Pediatrician. 1989;16:178–84.
89. Bartlett DW, Evans DF, Smith BG. The relationship between gastro-oesophageal reflux disease and dental erosion. J Oral Rehabil. 1996;23:287–97.
90. Birketvedt GS, Drivenes E, Agledahl I, Sundsfjord J, Olstad R, Florholmen JR. Bulimia nervosa—a primary defect in the hypothalamic-pituitary-adrenal axis? Appetite. 2006;46:164–7.
91. Crow SJ, Thuras P, Keel PK, Mitchell JE. Long-term menstrual and reproductive function in patients with bulimia nervosa. Am J Psychiatry. 2002;159:1048–50.
92. Resch M, Szendei G, Haasz P. Bulimia from a gynecological view: hormonal changes. J Obstet Gynaecol. 2004;24:907–10.
93. Freizinger M, Franko DL, Dacey M, Okun B, Domar AD. The prevalence of eating disorders in infertile women. Fertil Steril. 2010;93:72–8.
94. Yager J, Landsverk J, Edelstein CK, Jarvik M. A 20-month follow-up study of 628 women with eating disorders: II course of associated symptoms and related clinical features. Int J Eat Disord. 1988;7:503–13.
95. Seidenfeld MEK, Rickert VI. Impact of anorexia, bulimia and obesity on the gynecologic health of adolescents. Am Fam Physician. 2001;64:445–50.
96. Sodersten P, Bergh C. Anorexia nervosa: towards a neurobiologically based therapy. J Eur Pharm. 2003;480:67–74.
97. Butera PC. Estradiol and the control of food intake. Physiol Behav. 2009;99:175–80.
98. Dalvit-McPhillips S. The effect of the human menstrual cycle on nutrient intake. Physiol Behav. 1983;31:209–12.
99. Contrufo P, Monteleone P, D'lstria M, Fuschino A, Serino I, Maj M. Aggressive behavioral characteristics and endogenous hormones in women with bulimia nervosa. Neuropsychobiology. 2000;42: 58–61.
100. Naessén S, Carlström K, Glant R, Jacobsson H, Hirschberg AL. Bone mineral density in bulimic women—influence of endocrine factors and previous anorexia. Eur J Endocrinol. 2006;155:245–51.
101. Monteleone P, Luisi M, De Filippis G, et al. Circulating levels of neuroactive steroids in patients with binge eating disorder: a comparison with non-obese health controls and non-binge eating obese subjects. Int J Eat Disord. 2003;34:432–40.
102. Matzkin VB, Geissler C, Coniglio R, Selles J, Bello M. Cholesterol concentrations in patients with anorexia nervosa and in healthy controls. Int J Psychiatr Nurs Res. 2006;11:1283–93.

103. Pauporte J, Walsh BT. Serum cholesterol in bulimia nervosa. Int J Eat Disord. 2000;30:294–8.
104. Mira M, Stewart PM, Abraham S. Hormonal and biochemical abnormalities in women suffering from eating disorders. Pediatrician. 1985;12: 148–56.
105. Vögele C, Hilbert A, Tushchen-Caffier B. Dietary restriction, cardiac autonomic regulation and stress reactivity in bulimic women. Physiol Behav. 2009;98:229–34.
106. Messerli-Bürgy N, Engesser C, Lemmenmeier E, Steptoe A, Laederach-Hofmann K. Cardiovascular stress reactivity and recovery in bulimia nervosa and binge eating disorder. Int J Psychophysiol. 2010;98:229–34.
107. Casolo G, Balli E, Taddei T, Amuhasi J, Gori C. Decreased spontaneous heart rate variability on congestive heart failure. Am J Cardiol. 1989;64: 1162–7.
108. Gordon D, Herrera VL, McAlpine L, et al. Heart rate spectral analysis: a noninvasive probe of cardiovascular regulation in critically ill children with heart disease. Pediatr Cardiol. 1988;9:69–77.
109. Kienzle MG, Ferguson DW, Birkett CL, Myers GA, Berg WJ, Mariano DJ. Clinical hemodynamic and sympathetic neural correlates of heart rate variability in congestive heart failure. Am J Cardiol. 1992; 69:482–5.
110. Mortara A, La Rovere MT, Signorini MG, et al. Can power spectral analysis of heart rate variability identify a high risk subgroup of congestive heart failure patients with excessive sympathetic activation? A pilot study before and after heart transplantation. Br Heart J. 1994;71:422–30.
111. Nolan J, Flapan AD, Capewell S, MacDonald TM, Neilson JMM, Ewing DJ. Decreased cardiac parasympathetic activity in chronic heart failure and its relation to left ventricular function. Br Heart J. 1992;69:761–7.
112. Murialdo G, Casu M, Falchero M, et al. Alterations in the autonomic control of heart rate variability in patients with anorexia or bulimia nervosa: correlations between sympathovagal activity, clinical features, and leptin levels. J Endocrinol Invest. 2007;30:356–62.
113. Bostom AG, Shemin D, Verhoef P, et al. Elevated fasting total plasma homocysteine levels and cardiovascular disease outcomes in maintenance dialysis patients: a prospective study. Arterioscler Thromb Vasc Biol. 1997;17:2554–8.
114. Frieling H, Röschke B, Kornhuber J, et al. Cognitive impairment and its association with homocysteine plasma levels in females with eating disorders—findings from the HEaD-study. J Neural Transm. 2005;112:1591–8.
115. Wilhelm J, Römer KD, Gruss B, et al. Cognitive impairment and its association with homocysteine plasma levels in females with eating disorders—findings from the HEaD-study. J Neural Transm. 2005;112:1591–8.
116. Geisel J, Hubner U, Bodis M, et al. The role of genetic factors in the development of hyperhomocysteinemia. Clin Chem Lab Med. 2003;41: 1427–34.
117. Wilhelm J, Müller E, de Zwaan M, et al. Elevation of homocysteine levels is only partially reversed after therapy in females with eating disorders. J Neural Transm. 2010;117:521–7.
118. Vaz-Leal FJ, Rodriquez-Santos L, Melero-Ruiz JM, Ramos-Fuentes IM, Garcia-Herraiz AM. Psychopathology and lymphocyte subsets in patients with bulimia nervosa. Nutr Neurosci. 2010;13: 109–15.
119. Kendler KS, MacLean C, Neale M, Kessler R, Heath A, Eaves L. The genetic epidemiology of bulimia nervosa. Am J Psychiatry. 1991;148:1627–37.
120. Crow SJ, Peterson CB, Swanson SA, et al. Increased mortality in bulimia nervosa and other eating disorders. Am J Psychiatry. 2009;166:1342–6.
121. Klump KL, Kaye WH, Strober M. The evolving genetic foundations of eating disorders. Psychiatr Clin North Am. 2001;24:215–25.
122. Clarke TK, Weiss ARD, Berrettini WH. The genetics of anorexia nervosa. Clin Pharmacol Ther. 2011;91:181–8.
123. Bulik CM, Thorton LM, Root TL, Pisetsky EM, Lichtenstein P, Pedersen NL. Understanding the relation between anorexia nervosa and bulimia nervosa in a Swedish national twin sample. Biol Psychiatry. 2010;67:71–7.
124. Hollander E. Treatment of obsessive-compulsive spectrum disorders with SSRIs. Br J Psychiatry. 1998;35:7–12.
125. Bulik CM, Sullivan PF, Wade TD, Kendler KS. Twin studies of eating disorders: a review. Int J Eat Disord. 2000;27:1–20.
126. Fairburn CG, Harrison PJ. Eating disorders. Eat Disord. 2003;361:407–16.
127. Gorwood P, Kipman A, Foulon C. The human genetics of anorexia nervosa. J Eur Pharmacol. 2003;480: 163–70.
128. Root TL, Szatkieqicz JP, Jonassaint CR, et al. Association of candidate genes with phenotypic traits relevant to anorexia nervosa. Eur Eat Disord Rev. 2011;19:487–93.
129. Tracey D, Wade TD, Treloar SA, Heath AC, Martin NG. An examination of the overlap between genetic and environmental risk factors for intentional weight loss and overeating. Int J Eat Disord. 2009;42: 492–7.
130. Bulik CM, Devlin B, Bacanu SA. Significant linkage on chromosome 10p families with bulimia nervosa. Am J Hum Genet. 2003;72:200–7.
131. Kaye WH, Klump KL, Frank GK, Strober M. Anorexia and bulimia nervosa. Annu Rev Med. 2000;51:299–313.
132. Haleem DJ. Serotonin neurotransmission in anorexia nervosa. Behav Pharmacol. 2012;23:478–95.
133. Bruce KRH, Steiger SN, Young N, Kin MKNY, Israël M, Lévesque M. Impact of acute tryptophan

depletion on mood and eating-related urges in bulimia and nonbulimic women. J Psychiatry Neurosci. 2009;34:376–82.
134. Jimerson DC, Wolfe BE, Metzger ED, Finkelstein DM, Cooper TB, Levine JM. Decreased serotonin function in bulimia nervosa. Arch Gen Psychiatry. 1997;54:529–34.
135. Bailer UF, Bloss CS, Frank GK, et al. 5-HT (1A) receptor binding is increased after recovery from bulimia nervosa compared to control women and is associated with behavioral inhibition in both groups. Int J Eat Disord. 2010;44(6):477–87.
136. Wurtman RJ, Wurtman JJ. Brain serotonin, carbohydrate-craving, obesity and depression. Obes Res. 1995;3:477S–80.
137. Berger M, Gray JA, Roth BL. The expanded biology of serotonin. Annu Rev Med. 2009;60:355–66.
138. Hildebrandt T, Alfano L, Tricamo M, Pfaff DW. Conceptualizing the role of estrogens and serotonin in the development and maintenance of bulimia nervosa. Clin Psychol Rev. 2010;30(6):655–68.
139. Calati R, De Ronchi D, Bellini M, Serretti A. The 5-HTTLPR polymorphism and eating disorders: a meta-analysis. Int J Eat Disord. 2011;44:191–9.
140. Hammer C. Functional variants of the serotonin receptor type 3A and B gene are associated with eating disorders. Pharmacogenet Geonomics. 2009;19:790–9.
141. Wurtman JJ. Carbohydrate craving, mood changes, and obesity. J Clin Psychiatry. 1988;49:37–9.
142. Latner JD, Wilson GT. Binge eating and satiety in bulimia nervosa and binge eating disorder: effects of macronutrients intake. Int J Eat Disord. 2004;36:402–15.
143. Gorwood P, Batel P, Adèsb J, Hamon M, Bonid C. Serotonin transporter gene polymorphisms, alcoholism, and suicidal behavior. Biol Psychiatry. 2000; 48:259–64.
144. Lesch KP, Meyer J, Glatz K, et al. The 5-HT transporter gene-linked polymorphic region (5-HTTLER) in evolutionary perspective: alternative biallelic variation in rhesus monkeys. J Neural Transm. 1997; 104:1259–66.
145. Mikolajczyk E, Grzywacz A, Samochowiec J. The association of the catechol-O-methyltransferase genotype with the phenotype of women with eating disorders. Brain Res. 2009;1307:142–8.
146. Steiger H, Joober R, Israel M, et al. The 5HTTLPR polymorphism, psychopathologic symptoms, and platelet [3H-] paroxetine binding in bulimic syndromes. Int J Eat Disord. 2005;37:57–60.
147. Akkermann K, Nordquist N, Oreland L, Harro J. Serotonin transporter gene promoter polymorphism affects the severity of binge eating in general population. Prog Neuropsychopharmacol Biol Psychiatry. 2010;34:111–4.
148. Lee Y. Association between serotonin transporter gene polymorphism and eating disorders: a meta analytic study. Int J Eat Disord. 2009;43:498–504.
149. Racine SE, Culbert KM, Larson CL, Klump KL. The possible influence of impulsivity and dietary restraint on associations between serotonin genes and binge eating. J Psychiatr Res. 2009;43:1278–86.
150. Bello N, Hajnal A. Dopamine and binge eating behaviors. Pharmacol Biochem Behav. 2010;97:25–33.
151. Avena NM, Rada P, Moise N, Hoebel BG. Sucrose sham feeding on a binge schedule accumbens dopamine repeatedly and eliminates the acetylcholine satiety response. Neuroscience. 2006;139:813–20.
152. Baptista T, Reyes D, Hernandez L. Antipsychotic drugs and reproductive hormones: relationship to body weight regulation. Pharmacol Biochem Behav. 1999;62:409–17.
153. Erlanson-Albertsson C. How palatable food disrupts appetite regulation. Basic Clin Pharmacol Toxicol. 2005;97:61–73.
154. Noble EP. D2 dopamine receptor gene in psychiatric and neurologic disorders and its phenotypes. Am J Med Genet B Neuropsychiatr Genet. 2003;116: 103–25.
155. Kontis D, Theochari E. Dopamine in anorexia nervosa: a systemic review. Behav Pharmacol. 2012;23: 496–516.
156. Scherag S, Heberbrand J, Hinney A. Eating disorders: the current status of molecular genetic research. Eur Child Adolesc Psychiatry. 2010;19:211–26.
157. Craddock N, Owen MJ, O'Donovan MC. The catechol-O-methyl transferase (*COMT*) gene as a candidate for psychiatric phenotypes: evidence and lessons. Mol Psychiatry. 2006;11:446–58.
158. Zimmerli EJ, Devlin MJ, Kissileff HR, Walsh TB. The development of satiation in bulimia nervosa. Physiol Behav. 2010;100:346–9.
159. Guss JL, Kissilef HR, Walsh BT, Devlin MJ. Binge eating behavior in patients with eating disorders. Obes Res. 1994;2:355–63.
160. Keel PK, Wolfe BE, Liddle RA, De Young KP, Jimerson DC. Clinical features and physiology response to a test meal in purging disorder and bulimia nervosa. Arch Gen Psychiatry. 2007;64:1058–66.
161. Kissileff HR, Walsh BT, Kral JG, Cassidy SM. Laboratory studies of eating behavior in women with bulimia. Physiol Behav. 1986;38:563–70.
162. LaChaussee JL, Kissileff HR, Walsh BT, Hadigan CM. The single-item meal as a measure of binge-eating behavior in patients with bulimia nervosa. Physiol Behav. 1992;51:593–600.
163. Walsh BT, Kissileff HR, Cassidy SM, Dantzic S. Eating behavior of women with bulimia. Arch Gen Psychiatry. 1989;46:54–8.
164. Bailer UF, Kaye WH. A review of neuropeptide and neuroendocrine dysregulation in anorexia and bulimia nervosa. Curr Drug Targets CNS Neurol Disord. 2003;2:53–9.
165. Brennan AM, Mantzoros CS. Drug insight: the role of leptin in human physiology and pathophysiology—emerging clinical applications. Nat Clin Pract Endocrinol Metab. 2006;2:318–27.
166. Herpertz S, Wagner R, Nobert A, et al. Circadian plasma leptin levels in patients with anorexia

nervosa: relation to insulin and cortisol. Horm Res. 1998;50:197–204.
167. Köpp W, Blum WF, von Prittwitz S, et al. Low leptin levels predict amenorrhea in underweight and eating disordered females. Mol Psychiatry. 1997;2:335–40.
168. Jimerson DC, Wolfe BE, Carroll DP, Keel PK. Psychobiology of purging disorder: reduction in circulating leptin levels in purging disorder in comparison with controls. Int J Eat Disord. 2009;43:584–8.
169. Tanaka M, Nakahara T, Muranaga T, et al. Ghrelin concentrations and cardiac vagal tone are decreased after pharmacologic and cognitive-behavioral treatment in patients with bulimia nervosa. Horm Behav. 2006;50:261–5.
170. Wolfe BE, Jimerson DC, Orlova C, Mantzoros CS. Effect of dieting on plasma leptin, soluble leptin receptor, adiponectin and resistin levels in healthy volunteers. Clin Endocrinol. 2004;61:332–8.
171. Gáti A, Pászthy B, Wittman I, Abrahám I, Jeges S, Túry F. Leptin and glucose metabolism in eating disorders. Psychiatr Hung. 2007;22:163–9.
172. Ferron F, Considine RV, Peino R, Lado IG, Dieguez C, Casanueva FF. Serum leptin concentrations in patients with anorexia nervosa, bulimia nervosa and non-specific eating disorders correlate with the body mass index but are independent of the respective disease. Clin Endocrinol. 1997;46:289–93.
173. Hebebrand J, Blum WF, Barth N, et al. Leptin levels in patients with anorexia nervosa are reduced in the acute stage and elevated upon short-term weight restoration. Mol Psychiatry. 1997;2:330–4.
174. Mantzoros C, Flier JS, Lesem MD, Brewerton TD, Jimerson DC. Cerebrospinal fluid leptin in anorexia nervosa: correlation with nutritional status and potential role in resistance to weight gain. J Clin Endocrinol Metab. 1997;82:1845–51.
175. Mantzoros CS, Moschos SJ. Leptin: in search of roles in human physiology and pathophysiology. Clin Endocrinol. 1998;49:551–67.
176. Mathiak K, Gowin W, Hebebrand J, et al. Serum leptin levels, body fat deposition, and weight in females with anorexia or bulimia nervosa. Horm Metab Res. 1999;31:274–7.
177. Monteleone P, Lieto AD, Tortorella A, Longobardi N, Maj M. Circulating leptin in patients with anorexia nervosa, bulimia nervosa or binge-eating disorder: relationship to body weight, eating patterns, psychopathology and endocrine changes. Psychiatry Res. 2000;94:121–9.
178. Xi Q, Reed JT, Wang G, Han S, Englander EW, Greeley GH. Gherlin secretion is not reduced by increase fat mass during diet-induced obesity. Am J Physiol Regul Integr Comp Physiol. 2008;295:R429–35.
179. Monteleone P, Martiadis V, Fabrazzo M, Serritella C, Maj M. Ghrelin and leptin responses to food ingestion in bulimia nervosa: implications for binge-eating and compensatory behaviors. Psychol Med. 2003;33:1387–94.
180. Kojima S, Nakahara T, Nagai N, et al. Altered ghrelin and peptide YY responses to meals in bulimia nervosa. Clin Endocrinol. 2005;62:74–8.
181. Helm KA, Rada P, Hoebel BG. Cholecystokinin combined with serotonin in the hypothalamus limits accumbens dopamine release while increasing acetylcholine: a possible satiation mechanism. Brain Res. 2003;963:290–7.
182. Devlin MJ, Walsh BT, Guss JL, Kissileff HR, Liddle RA, Petkova E. Postprandial cholecystokinin release and gastric emptying in patients with bulimia nervosa. Am J Clin Nutr. 1997;65:114–20.
183. Geliebter A, Melton PM, McCray RS, Gallagher DR, Gage D, Hashim SA. Gastric capacity, gastric emptying, and test-meal intake in normal and bulimic women. Am J Clin Nutr. 1992;56:656–61.
184. Geracioti Jr TD, Liddle RA. Impaired cholecystokinin secretion in bulimia nervosa. N Engl J Med. 1988;319:683–8.
185. Pirke KM, Kellner MB, Friess E, Krieg JC, Fichter MM. Satiety and cholecystokinin. Int J Eat Disord. 1994;15:63–9.
186. Geliebter A, Hashim SA. Gastric capacity in normal, obese, and bulimic women. Physiol Behav. 2000;74:743–6.
187. Cuellar RE, Kaye WH, Hsu LK, VanThiel DH. Upper gastrointestinal tract dysfunction in bulimia. Dig Dis Sci. 1988;33:1549–53.
188. Kiss A, Bergmann H, Abatzi TA, et al. Oesophageal and gastric motor activity in patients with bulimia nervosa. Gut. 1990;31:259–65.
189. Shih WJ, Humphries L, Digenis GA, Castellanos FX, Domstad PA, DeLand FH. Tc-99m labeled triethelene tetraamine polysterene gastric emptying studies in bulimia patients. Eur J Nucl Med. 1987;13:192–6.
190. Walsh BT, Zimmerli E, Devlin MJ, Guss J, Kissileff HR. A disturbance of gastric function in bulimia nervosa. Biol Psychiatry. 2003;54:929–33.
191. Ribases M, Gratacos M, Badia A, et al. Contribution of NTRK2 to the genetic susceptibility to anorexia nervosa, harm avoidance and minimum body mass index. Mol Psychiatry. 2005;10:851–60.
192. Zhu AJ, Walsh BT. Pharmacologic treatment of eating disorders. Can J Psychiatry. 2002;47:227–34.
193. Ricca V, Mannucci E, Zucchi T, Rotella CM, Faravelli C. Cognitive-behavioural therapy for bulimia nervosa and binge eating disorder. A review. Psychother Psychosom. 2000;69(6):287–95.
194. Keel PK, Mitchell JE. Outcome in bulimia nervosa. Am J Psychiatry. 1997;154:313–21.
195. Carter JC, Stewart DA, Dunn VJ, Fairburn CG. Primary prevention of eating disorders: might it do more harm than good? Int J Eat Disord. 1997;22:167–72.
196. Killen JD, Taylor CB, Hammer LD, et al. An attempt to modify unhealthful eating attitudes and weight regulation practices of young adolescent girls. Int J Eat Disord. 1993;13:369–84.

197. Mann T, Nolen-Hoeksema S, Huang K, Burgard D, Wright A, Hanson K. Are two interventions worse than none? Health Psychol. 1997;16:215–25.
198. Morgan JF, Reid F, Lacey JH. The SCOFF questionnaire: assessment of a new screening tool for eating disorders. BMJ. 1999;319:1467–8.
199. Ackard DM, Fulkerson JA, Neumark-Sztainer D. Prevalence and utility of DSM IV eating disorder diagnostic criteria among youth. Int J Eat Disord. 2007;40:409–17.
200. Ando T, Komaki G, Nishimura H, et al. A ghrelin gene variant may depict crossover rate from restricting-type anorexia nervosa to other phenotypes of eating disorders: a retrospective survival analysis. Psychiatr Genet. 2010;20:153–9.
201. Kaye WH, Cynthia BM, Thornton L, Barbarich N, Masters K, Price Foundation Collaborative Group. Comorbidity of anxiety disorders with anorexia and bulimia nervosa. Am J Psychiatry. 2004;161: 2215–21.
202. Sim L, Zeman J. Emotion awareness and identification skills in adolescent girls with bulimia nervosa. J Clin Child Adolesc Psychol. 2004;33:760–71.

# The Female Athletic Triad: Disordered Eating, Amenorrhea, and Osteoporosis

12

Jacalyn J. Robert-McComb and Andrew Cisneros

**Abstract**

The female athlete triad is defined as the interrelationships among energy availability, menstrual function, and bone mineral density. These dynamic components may transcend towards various clinical manifestations including eating disorders, functional hypothalamic amenorrhea, and osteoporosis. The occurrence of low energy availability, amenorrhea, and osteoporosis, alone or in combination, poses deleterious health risks to physically active girls and women. Deficits in energy intake may be acquired through excessive energy expenditure; however, disordered eating habits have been a tremendous concern and a risk factor for the female athlete triad. Therefore, clinicians and health care professionals must be highly aware of its prevalence for prevention. Low energy availability, with or without disordered eating, disrupts physiological function by suppressing the hypothalamic–pituitary–gonadal axis leading to functional amenorrhea. Additionally, recent literature has shown disturbances in endothelial function and may compromise the cardiovascular system. The prevalence of stress fractures has been linked to poor bone health and a severe risk factor for osteoporosis. The appropriate diagnosis and management is crucial to ameliorate health and quality of life. Recommendations have been made by various leading organizations, such as the American College of Sports Medicine, to successfully manage this syndrome. However, specific evidence-based guidelines are still being conducted. Nevertheless, solid background knowledge of the interrelationships of the various components of the triad is necessary for the allied health professional.

**Keywords**

Energy availability • Functional amenorrhea • Bone mineral density • Disordered eating • Osteoporosis

J.J. Robert-McComb, Ph.D., FACSM, ACSM P.D. (✉)
Health, Exercise and Sports Sciences, Texas Tech University, 3204 Main Street, Lubbock, TX 79409, USA
e-mail: jacalyn.mccomb@ttu.edu

A. Cisneros, M.S., NSCA, CSCS
School of Allied Health Sciences: Physical Therapy, Texas Tech University Health Sciences Center, 3601 4th Street MS 6294, Lubbock, TX 79430, USA
e-mail: Andrew.cisneros@ttuhsc.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_12, 

## 12.1 Learning Objectives

After completing this chapter, you should have an understanding of the following:

- Updated American College of Sports Medicine Position Stand on the female athlete triad
- Consequences of low energy availability with or without an eating disorder
- Progressive nature of functional menstrual disturbances in athletes
- Deleterious effects on bone metabolism leading to osteoporosis
- Interrelatedness of energy availability, amenorrhea, and bone mineral density
- Athletes at greatest risk for developing signs and symptoms associated with this syndrome

## 12.2 Introduction

In 1992, the term female athlete triad was introduced to describe the interrelationships among disordered eating, amenorrhea, and osteoporosis observed in adolescent and young adult female athletes [1]. The American College of Sports Medicine (ACSM), according to their 2007 Position Stand, updated its definition of the triad as a spectrum of interrelationships among energy availability, menstrual function, and bone mineral density that may transcend towards the following clinical manifestations; eating disorders, functional hypothalamic amenorrhea, and osteoporosis [2]. The occurrence of low energy availability (with or without eating disorders), amenorrhea, and osteoporosis, alone or in combination, poses deleterious health risks to physically active girls and women. Therefore, clinicians and health care professionals must be highly aware of its prevalence along with the interrelatedness of these components.

Energy availability (EA) is defined as dietary energy intake (DEI) minus exercise energy expenditure (EEE). In healthy individuals, energy balance occurs at approximately 45 kcal/kgFFM/day of EA. This state of balance in healthy adult females provides adequate energy for other normal physiological processes [3]. When energy availability is severely reduced, the body restores energy balance by suppressing energy-consuming physiological processes, including reproductive function. The medical consequences of this pathological form of energy balance are the price paid for preserving life. It should be noted that energy availability (DEI–EEE) is not the same as energy balance. Energy balance is defined as DEI minus total energy expenditure (heat from all cellular functions), not just EEE. A whole body calorimeter or chamber is used to directly measure total energy expenditure as the body's rate of heat production. Energy availability is much simpler and less costly to measure as it only requires diet analysis software, an ergo meter (such as an accelerometer or heart rate monitor), and an electrical impedance body composition scale.

Low EA may occur with or without eating disorders and/or excessive energy expenditure during exercise without compensation through dietary means. The consequences of low EA may distort physiological mechanisms for cellular maintenance, thermoregulation, growth, and reproduction [4]. A wide spectrum of abnormal eating behaviors such as excessive caloric restriction, binge eating, and purging or the use of diet pills, laxatives, diuretics, and enemas has been documented to reduce EA [2, 5, 6].

The prevalence of eating disorders among female athletes is of great concern since these behavioral syndromes are associated with considerable morbidity leading to one of the highest mortality rates among mental illness [7]. Unfortunately, sport participation for female athletes has become a possible risk factor for the potential of an eating disorder [8, 9]. Various forms of eating disorders, such as anorexia nervosa (AN) and bulimia nervosa (BN), have subtle signs initially. Anorexia nervosa is characterized as the following: restrictive eating by the self-conscious individual that views herself as overweight and is afraid of gaining weight despite a weight 15 % below expected weight for age and height [10]. Individuals with a normal weight range that continuously cycle with binge eating followed by purging or other compensatory behaviors such as fasting or excessive exercise are described as BN [10]. The spectrum of menstrual

function ranges from eumenorrhea (regular menstrual cycles) to amenorrhea, with the latter having negative physiological consequences. Primary amenorrhea is defined as the absence of menarche by the age of 15 after secondary sexual characteristics [2, 11]. The absence of menstrual cycles lasting more than 3 months after menarche cycles have been previously established is called secondary amenorrhea [2, 11]. In contrast, oligomenorrhea is defined as menstrual cycles occurring at intervals longer than 35 days, but anovulation and luteal deficiency have no perceptible symptoms [2, 3].

In the female athlete triad, low EA may cause functional hypothalamic amenorrhea. It is called functional because it is a functional problem, not an anatomical problem, and the pathology is reversible. During this occurrence, ovarian function is suppressed by an abnormally slow frequency of hormone pulsatility due to inhibition of the hypothalamic–pituitary–ovarian axis (HPO), also called the hypothalamic–pituitary–gonadal (HPG) axis [3]. Chronic energy deficiency directly affects the HPO axis by disrupting the pulsatile release of gonadotropin-releasing hormone (GnRH) by the hypothalamus. The disrupted pulsatility of GnRH disrupts the pulsatile release of luteinizing hormone (LH) and follicle stimulating hormone (FSH) by the pituitary. Without normal LH and FSH pulsatility, the follicles do not develop in the ovary, estrogen and progesterone production is decreased due to lack of ovarian stimulation, and menses either occurs irregularly or not at all (Figs. 12.1 and 12.2).

Loucks found that LH pulsatility is disrupted when EA is reduced below approximately 30 kilocalories (kcal) per kilogram (kg) of fat-free mass (FFM) per day (kcal/kgFFM/day) [2, 3]. Furthermore, endothelial dysfunction may be associated with the disruption of the menstrual cycle [12, 13].

Osteoporosis is a disease characterized by compromised bone strength leading to an increase risk of bone fracture. Low EA and menstrual dysfunction may predispose premenopausal osteoporosis in active young women due to decreases in ovarian hormone production and hypoestrogenemia [14]. The remodeling of bone is also

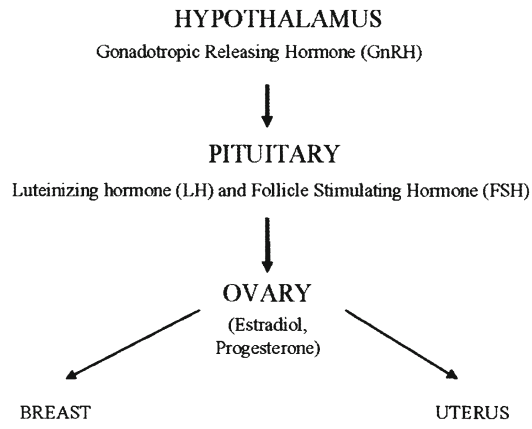



**Fig. 12.1** This figure shows the primary components of the female reproductive system and the hormones that communicate between the various organs. The hormones produced by each gland are shown in parentheses

dependent on EA. Bone resorption increases when exercising women reduce EA enough to suppress estradiol. In addition, bone formation decreases with the concentrations of anabolic hormones as EA declines from 30 to 20 kcal/kgFFM/day. This places female athletes at greater risk for sustaining stress fractures and osteoporotic fractures later in life [15].

Controversy over the diagnostic criteria for low bone mass in premenopausal women has led to the following criteria set by the International Society for Clinical Densitometry (ISCD) [16]. The ISCD has recommended bone mineral density (BMD) be expressed as *Z*-scores to compare individuals to age- and sex-matched controls with the following classifications: *Z*-scores below 2.0 be termed "low bone density below the expected range for age" in premenopausal women and as "low bone density for chronological age" in children. Furthermore, osteoporosis is to be diagnosed only when low BMD is accompanied by secondary risk factors such as chronic malnutrition, eating disorders, hypogonadism, glucocorticoid exposure, and previous fractures.

In 2007, the ACSM published its updated Position Stand on the female athlete triad to present clinical recommendations for guiding primary care [2]. These recommendations were evaluated in categories based off strength of scientific evidence: (A) consistent and good-quality

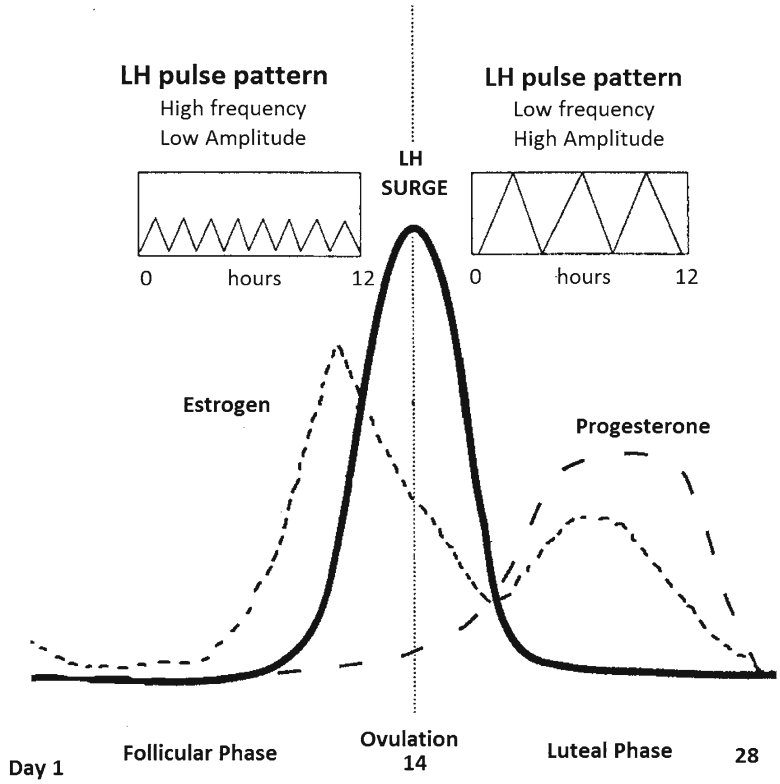



**Fig. 12.2** An LH surge occurs at the time of ovulation and marks the division between the follicular phase (days 1–14) and the luteal phase (days 15–28). LH pulse pattern also changes across the menstrual cycle; pulse frequency decreases from the follicular phase (−65- to 80-min intervals) to the luteal phase (−185- to 200-min intervals), whereas pulse amplitude increases from the follicular phase (−5 miU/mL) to the luteal phase (−12 miU/mL)

evidence for clinical outcomes on mortality, morbidity, symptoms, cost, and quality of life; (B) inconsistent or limited quality of evidence for these same clinical outcomes; (C-1) evidence based on biochemical, histological, physiological, and pathophysiological outcomes; and (C-2) evidence based on case studies, consensus, usual practice, and opinion. The ACSM recommendations are as follows:

1. Severe undernutrition impairs reproductive and skeletal health. Evidence category A.
2. Menstrual irregularities and low BMD increase stress fracture risk. Evidence category A.
3. Disordered eating, eating disorders, and amenorrhea occur more frequently in sports that emphasize leanness. Evidence category A.
4. To diagnose functional hypothalamic amenorrhea, other causes of amenorrhea must be excluded. Evidence category B.
5. Treatment for disordered eating and eating disorders included nutritional counseling and individual psychotherapy. Cognitive behavioral, group therapy, and/or family therapy may also be used. Evidence category B.
6. The first aim of treatment is to increase EA by increasing energy intake and/or reducing energy expenditure. Athletes without disordered eating or eating disorders should be referred for nutritional counseling. Evidence category C-1.
7. Athletes practicing restrictive eating behaviors should be counseled that increases in body weight are necessary to increase BMD. Evidence category C-1.
8. In functional hypothalamic amenorrhea, increases in BMD are more closely associated with increases in weight than with oral contraceptive pill (OCP) or hormone

replacement therapy (HRT) administration. Evidence category C-1.

9. BMD should be assessed after a stress or low-impact fracture and after a total of 6 months of amenorrhea, oligomenorrhea, disordered eating, or an eating disorder. Evidence category C-2.
10. Multidisciplinary treatment for the triad disorders should include a physician (or other health-care professional), a registered dietitian, and, for athletes with disordered eating or an eating disorder, a mental health practitioner. Evidence category C-2.
11. Screening for the triad should occur at the preparticipation exam or annual health-screening exam. Evidence category C-2.
12. Athletes with one component of the triad should be assessed for the others. Evidence category C-2.
13. Athletes with disordered eating should be referred to a mental health practitioner for evaluation, diagnosis, and recommendations for treatment. Evidence category C-2.
14. Athletes with disordered eating and eating disorders who do not comply with treatment may need to be restricted from training and competition. Evidence category C-2.
15. OCP should be considered in an athlete with functional hypothalamic amenorrhea over age 16, if BMD is decreasing with nonpharmacological management, despite adequate nutrition and body weight. Evidence category C-2.

Although any athlete may suffer from the disorders associated with the female athlete triad, girls and women who participate in sports that place a premium on appearance and thinness are especially susceptible [17]. According to the International Olympic Committee's Position Stand on the female athlete triad, high-risk sports include not only ones that emphasize a thin body size or shape (distance running, cycling, cross-country skiing) but also in sports that categorize weight classes (rowing, martial arts, wrestling, weightlifting), use revealing attire (swimming, volleyball, diving, cross-country skiing, track and field, cheerleading), are judged (diving, figure skating, gymnastics), or have an appearance aspect (rhythmic gymnastics) [18].

Additional stressors that contribute to disorders of the triad in young athletes are the natural biological changes that occur in puberty or the increase in sex-specific fat during puberty. These young athletes must not only cope with these biological changes but must also conform to the pressures to stay thin for increased sport performance. Disordered eating is often an unhealthy attempt to stay thin for increased sport performance.

Early recognition and awareness of athletes most at risk and the early signs of an eating disorder is essential when developing medical protocols for the triad of disorders. It is important for allied health professionals to be able to recognize disordered eating patterns before these subclinical disorders process to a clinical diagnosis.

## 12.3 Research Findings

### 12.3.1 Impact of Low Energy Availability Through Disordered Eating

The early stages of the female athlete triad are induced by disordered eating, intentionally or unintentionally, and low EA. Much emphasis has been placed on the impact of disordered eating in regard to diminishing levels of energy supply [5, 19]. This behavior has been shown to be a risk factor for more serious eating disorders such as AN and BN [20]. Although, low EA in the female athlete does not automatically imply that she has disordered eating or an eating disorder. Exercise training is also known to suppress appetite.

The diagnostic criteria for Eating Disorders in the Diagnostic and Statistical Manual of Mental Disorders-DSM-IV-TR have been continuously used to diagnose eating disorders [2, 21]. However, the ICD-10 criteria (International Classification of Diseases) may be effective in diagnosing AN and BN [22]. A gold standard screening criteria for disordered eating still remains controversial due the wavering of opinions between the behaviors associated with AN and BN. Nevertheless, the ACSM has continuously endorsed the utilization of the DMS-IV-TR [1, 2].

Individuals with AN may move back and forth between the two types of subgroups in AN since similar characteristics between these two subgroups exist. The two subtypes associated with AN are (1) AN restricting type (AN-R) and (2) AN binge/purge type (AN-BP). The restricting subtype accomplishes weight loss through dieting, fasting, and excessive exercise. The bulimic subtype purges after binge eating or even after the consumption of a small amount of food through self-induced vomiting or the misuse of laxatives, diuretics, or enemas. Although BN and AN share similar views of distorted body image and a drive for thinness, the individual with BN will typically have a normal body weight [23]. There are also two subgroups of BN: (1) BN purging type (BN-P) and (2) BN non-purging type (BN-N). BN poses a risk for the development of a range of secondary cognitive, behavioral, and physical impairments and disorders that may progress towards more serious psychological disorders [24].

Eating disorder not otherwise specified (EDNOS) is a diagnosis for eating disorders that meets some but not all of the specific criteria for AN or BN in reference to the DMS-IV-TR [25]. For example, all of the criteria for AN are met except that the individual has regular menses, or all of the criteria for BN are met except that the binge eating and inappropriate compensatory mechanisms occur at a frequency of less than twice a week or for a duration of less than 3 months.

Another diagnostic category included in the DMS-IV-TR is binge eating disorder (BED) and falls under the eating disorder not otherwise specified category. Interesting research has supported the view that BED may derive from neuropsychogenic (enhanced dopamine neurotransmission) origins leading towards rewards-based overeating [26]. Therefore the recent study of neuropsychopharmacology has become of great importance to understand the physiological mechanisms that occur with various eating disorders.

Disordered eating is a classification (within the Diagnostic and Statistical Manual of Mental Disorders [DSM-IV-TR]) used in the health care field to describe a wide range of irregular eating behaviors that do not warrant a diagnosis of a specific eating disorder. However, researchers have found evidence that disordered eating patterns, such as excessive dieting, fasting, and binging, can lead to more serious eating disorders. When individuals do not meet the criteria for EDNOS but still manifest partial symptoms, *disordered eating behavior* is the appropriate category.

### 12.3.2 Progressive Nature of Menstrual Disturbances in Athletes

Menstrual disorders occur due to abnormal pituitary gland function and secreting pulses of luteinizing hormone (LH) at the correct frequency. LH pulsatility reflects gonadotropin-releasing hormone (GnRH) secretion via the hypothalamus [27]; thereby, abnormality to this mechanism is known as functional hypothalamic amenorrhea. Low EA has also shown to attenuate levels of metabolic hormones such as insulin, cortisol, growth hormone, insulin-like growth factor-I (IGF-I), triiodothyronine (T3), and leptin [2, 28]. Studies have shown that low EA due to disordered eating and caloric restriction negatively impacts the menstrual cycle compared to excessive energy expenditure alone since restoring normal caloric intake ameliorates menstrual function [29, 30]. It is still recommended that female athletes increase caloric intake and decrease physical activity to promote the return of normal menses [31].

Amenorrhea in women can lead to infertility due to the absence of ovarian follicular development, ovulation, and luteal function. Moreover, luteal deficiency may be at risk for infertility due to poor follicular development. The progression of menstrual dysfunction has implications for increased risk of endometrial cancer because the follicle starts to develop, but the process ceases before ovulation, in which an environment of unopposed estrogen is created [32]. Recent research has also shown hypoestrogenism in amenorrheic athletes can induce impaired endothelium dysfunction in the arterial system [33]. Therefore, cardiovascular health may be compromised.

The prevalence of secondary amenorrhea, defined as the absence of menstrual cycles lasting

more than 3 months after menarche cycles, has been previously established and varies widely due to sport, age, training volume, and body weight. Previous reports in small studies have noted menstrual dysfunction in 69 % in dancers [34] and 65 % in long-distance runners [35] compared to a significantly smaller percentage in the normal population. Torstveit and Borgen have noted that a significant amount of female athletes suffer from the triad, especially in leanness sports; however, the presentation of this syndrome should not be ignored in the general population [9]. The progressive nature of menstrual disturbances in athletic women resembles the pattern depicted below. Stages 1–3 are usually asymptomatic but may present as infertility [36]:

1. Regular cycles with a shortened luteal phase—progesterone production stops early.
2. Regular cycles with inadequate progesterone production.
3. Regular cycles with failure to develop and release an egg (ovulation).
4. Irregular cycles but still ovulating.
5. Irregular cycles and anovulation.
6. Absence of menses and anovulation.

### 12.3.3 Low Bone Mineral Density and Osteoporosis

Current literature has shown a two- to fourfold greater incidence of stress fractures in athletes with irregular menses [37]. However, epidemiological data relating to BMD to fractures in premenopausal women are lacking along with the wide variety risk factors that contribute to bone health. These variables include bone mineral density for bone size, pubertal stage, skeletal maturity, or body composition in growing adolescents [2].

The International Society for Clinical Densitometry (ISCD) has recommended that BMD be objectively quantified in children and premenopausal women in terms of *Z*-scores compared to age, race, and sex characteristics [16]. A *Z*-score is the number of standard deviations above or below what is normally expected for someone of the same age, sex, weight, and ethnic or racial origin. Their recommendation was that *Z*-scores below 2.0 be termed "low bone density below the expected range for age" in premenopausal women and as "low bone density for chronological age" in children. Furthermore, secondary risk factors, such as undernutrition, hypogonadism, and a history of fractures, are combined to further diagnose osteoporosis if the *Z*-score lies below 2.0. With menstrual irregularities increasing the risk of stress fractures, clinicians should be aware of other factors such as age, ethnicity, prior exercise training, smoking, and alcohol consumption [38, 39]. Nonetheless, if an athlete has a *Z*-score that is −1, further investigation is justified and recommended [22]. The rational for this recommendation is that athletes in weight-bearing sports usually have 5–15 % higher BMD than nonathletes [2]. Therefore, low BMD is defined as a *Z*-score between −1.0 and −2.0 for physically active and athletic premenopausal woman and children.

Poor nutritional input along with excessive energy expenditure has shown to significantly impact bone health in the female athlete. Although the primary cause of osteoporosis in postmenopausal women is due estrogen deficiency, nutritional deficits more so lead to abnormal bone remodeling in athletes with functional hypothalamic amenorrhea in younger female athletes [28]. Miller et al. found that a 38 % increase in body weight over 3 months was associated with significant increases in BMD in anorexic women although amenorrhea persisted [40]. The utilization of pharmacotherapy (hormone replacement therapy and oral contraceptives) has not shown to fully restore bone mineral density in women with functional hypothalamic amenorrhea, thereby providing reasonable evidence that under nutrition severely impacts bone health [2, 41, 42].

### 12.3.4 Interrelatedness of Low Energy Availability, Amenorrhea, and Osteoporosis

A consistency of low EA, with or without disordered eating, leads to menstrual dysfunction with

concomitant effects on bone health. Disordered eating is a key risk factor that may progress towards eating disorders due to psychological implications from low self-esteem, depression, and anxiety disorders [8]. Management of disordered eating has become a tremendous concern in the female athlete triad because of its prevalence more so in leanness sports. Nevertheless, chronic energy deprivation directly affects the HPG axis by decreasing the amplitude and frequency of pulsatile release of gonadotropin-releasing hormone (GnRH) produced by the arcuate nucleus of the hypothalamus. This decreased release of GnRH causes decreased release of luteinizing hormone (LH) and follicle stimulating hormone (FSH) by the pituitary [2]. Ovulation does not occur without the LH surge imperative during the mid-cycle, thereby decreasing production of estrogen and progesterone. Decreases due to lack of ovarian stimulation and menses either occur irregularly or not at all [43]. Consequences of hypoestrogenism cause impaired endothelium-dependent arterial vasodilation, thereby attenuating the perfusion of working muscle, impaired skeletal muscle oxidative metabolism, and elevated low-density lipoprotein cholesterol levels [2, 33].

Increasing dietary energy intake to combat low EA has shown to normalize metabolic and reproductive function. De Souza et al. demonstrated an association between metabolic status and reproductive function supporting the existence of dose–response relationship between energy status (REE and metabolic hormones) and clinical categories of menstrual dysfunction [44]. Therefore, subtle changes in EA impact the reproductive axis associated with delays in follicular maturation and compromised luteal function. It appears that exercise training does not disrupt LH pulsatility or menstrual cycles beyond the impact of its energy cost on EA [30]. Low EA may occur due to disordered eating leading towards more serious eating disorders such as AN and BN. However, amenorrhea or luteal suppression may occur without restricting caloric intake or by failing to increase dietary energy intake in sufficient compensation for exercise energy expenditure [18].

A restricted EA with suppression of bone formation plus the attenuation in endogenous estrogen associated with amenorrhea can progressively decrease bone mass. This occurs when bone resorption exceeds bone formation during bone remodeling. During childhood (11–14 years old), bone formation is dominant over bone resorption in females. Estrogen enhances growth and modeling that occurs during puberty. However, a deficit in estrogen during adolescents compromises peak bone formation in the final stages of pubertal progression [45].

In women, peak bone mass is ultimately reached between 25 and 30 years of age determined by estrogen status, diet, exercise, body weight, gender, and genetic influences [46]. In hypothalamic amenorrhea in the female athlete, hypoestrogenic states are not the predominant variable affecting bone health as is during puberty. A recent systemic review by Vescovi et al. found that therapies containing an estrogen given for 8–24 months result in variable improvements (1.0–19.0 %) in BMD, yet failed to restore bone mass in comparison to age-matched controls [47]. Furthermore, nine studies included in this systemic review reported that an increase in caloric intake increases weight gain, resumption of menses with a 1.1–16.9 % increase in BMD in conjunction with an improvement in bone formation, and reduction in bone resorption markers [47]. A recent publication from the American Journal of Clinical Nutrition found that female adolescent runners ($n = 13$) with an elevated bone turnover had a lower body mass, fewer menstrual cycles in the past year, lower estradiol and 25-hydroxycholecalciferol concentrations, vitamin D insufficiency, amenorrhea, and low bone mass. Furthermore, the runners with an elevated bone turnover had a profile assessment consistent with energy deficiency implying the significance of EA [48].

Current literature has included endothelial dysfunction as a possible forth component to the female athlete triad. The clinical implications pertain to cardiovascular disease, which is known to be the number one cause of death in women [8]. Hoch et al. found a stronger relationship between athletic amenorrhea and brachial artery

endothelial dysfunction compared to oligomenorrheic athletes and a control group. Results implied that brachial artery flow-mediated dilation (FMD) was significantly decreased in amenorrheic athletes (1.08 ± 0.90 %) compared to oligomenorrheic athletes (6.44 ± 1.28 %) and a control group (6.38 ± 1.38 %) [49]. A recent prospective cross-sectional study in 2011 found that 64 % of ballet dancers in a group of 32 had abnormal brachial artery FMD defined as less than 5 % (2.9 ± 1.5 %). Furthermore, 4 weeks of folic acid supplementation (10 mg/day) significantly increased FMD (7.1 ± 2.3 %, $p < 0.001$) [33]. Therefore, it seems that adequate nutritional intake ameliorates FMD to prevent the potential for cardiovascular disease, along with normalizing the function of menstrual cycles and improving bone health.

### 12.3.5 Athletes at Greatest Risk for Developing Signs and Symptoms Associated with the Triad

Female athletes in sports where thinness confers a competitive advantage are at greatest risk for low EA. Low EA may be related to restricting caloric intake, excessive energy expenditure, vegetarian diet, and purposely limiting certain foods. Disordered eating is the prime risk factor to develop much more serious eating disorders such as AN and BN [8]. The diagnosis of certain disordered eating behaviors has been of much concern with many clinicians because it is attributed to psychological stressors including environmental and social factors, psychological predisposition, family dysfunction, physical and mental abuse, low self-esteem, and genetics [2]. Additionally, various reports have demonstrated negative attitude scores in female athletes related to leanness sports [6, 50].

A study by Torstveit, Rosenvinge, and Sundgot-Borgen investigated the percentage of female elite athletes ($n = 186$) and controls ($n = 145$) with disordered eating behavior and clinical EDs between the ages of 13 and 39 [51]. Results showed that more athletes in leanness sports (46.7 %) had clinical EDs compared to non-leanness sport athletes (19.8 %) and controls (21.4 %) ($p > 0.001$). Furthermore, the authors found menstrual dysfunction in leanness athletes, self-reported EDs in non-leanness athletes, and self-reported use of abnormal weight control methods in controls as valid screening procedures. Thereby, specific risk factors appear to not be universal pertaining to athletes and non-athletes.

Screening for menstrual dysfunction has also become vital because of its progressive nature towards interrupting the reproductive system. Various studies have reported a higher percentage of leanness athletes compared to controls with menstrual dysfunction occurrence. A recent publication in the Medicine and Science in Sports Exercise found that athletes competing in high-risk sports (endurance, weigh classes, leanness sports) produced significantly more stress fractures compared to other female athletes competing in low-risk sports [52]. Additionally, there is no association between the female athlete triad and body mass index.

## 12.4 Contemporary Understanding of the Issues

A general consensus exists among researchers and leading sport organizations that disordered eating and menstrual dysfunction is a health issue for many female athletes competing in sports focusing on leanness or low body weight. The existence of the triad components has been well documented in the collegiate ranks, yet limited information exists about this syndrome in high school female athletes. However, a recent cross-sectional design study examined 249 female athletes competing in sport teams, dance teams, or cheerleading in high school. The results showed the prevalence of menstrual irregularity and musculoskeletal injury in 19.7 % and 63.1 %, respectively. Furthermore, it was reported that menstrual dysfunction sustained a higher percentage of severe injuries (missing more than 22 days of practice or competition) compared to athletes with regular menses [53]. Therefore, the female athlete triad is affecting many athletes of all ages.

The appropriate screening and management of the female athlete triad has become an enormous issue within all clinicians practicing. Treatment of the triad must involve a multidisciplinary team approach that includes a physician, registered sports dietician, certified sports psychologist, athletic trainers, coaches, and along with friends with family members [2]. Many colleges and high schools do not use a medical history form that particularly asks questions that may determine if various components of the triad exist, such as disordered eating, amenorrhea, and low bone mineral density. Therefore, the allied health professionals such as athletic trainers, school nurses, team physicians, physical therapists, nutritionists, and exercise physiologists must implement effective screening protocols for such.

Unfortunately, allied health professionals may be inadequate in recognizing various risk factors and components of the female athlete triad. A recent publication in the Physical Therapy in Sport found that only 54 out of 205 physical therapists used specific treatment methods such as education for the female athlete triad. Moreover, only 13 out of 54 physical therapists assisted in the athletic screening for the triad disorders [54]. Clinicians are responsible for recognizing, evaluating, and preventing this syndrome, in which a greater awareness and knowledge of the triad is vital.

Preparticipation exams and annual health-screening exams are recommended for all athletes to screen for the triad. Screening for the triad requires a solid understanding of the interrelationships of the components along with the various spectrums of health for eating behaviors, menstrual function, and bone health. Screening tools are available to diagnose various disordered eating behaviors that may potentially lead to eating disorders. The key is to prevent reduced EA due to abnormal eating behaviors or uncompensated energy expenditure through intense exercise training. If low EA is suspected, it is recommended that increased caloric intake be applied through appropriate eating behaviors [2, 31]. Athletes must also be assessed for the other components of the triad if one component exists with a referral to their physician [2, 8].

## 12.5 Future Directions

The battle continues for allied health professionals to assess and intervene with athletes suffering from the female athlete triad. The physiological relationships between low EA, functional amenorrhea, and low bone mineral density have been established through many publications along with the heightened prevalence among female athletes of all ages. However, specific guidelines for screening and managing the triad are lacking in the literature. The ACSM has set out recommendations on screening and diagnosing primarily through evidence based off case studies, consensus, usual practice, and opinion. Therefore, consistent and high-quality designed studies are needed to validate evidence-based practice for the allied health professional.

Nevertheless, recent literature has provided well-established research to recommend that undernutrition through low EA impairs reproductive and skeletal health. Thereby, increasing caloric intake should be the first intervention utilized to combat the progressive nature of the female athlete triad [31]. A recent study by Becker et al. [55] showed promising results in reducing risk factors for eating disorders and the female athlete triad with two evidence-based programs; athlete-modified dissonance prevention and healthy weight intervention. Both interventions were able to reduce thin-ideal internalization, dietary restraint, bulimic pathology, shape and weight concern, negative affect at 6 weeks, bulimic pathology, shape concern, and negative affect at 1 year.

The challenges of recognizing the female athlete triad along with the appropriate management for the various components have become the focus in the scientific literature recently. Additionally, endothelial dysfunction may become a forth component of the triad since it appears to accompany amenorrhea and low BMD [2, 8, 12]. The relationship between brachial artery endothelial dysfunction and coronary artery dysfunction is vital due to coronary artery endothelial dysfunction positively correlating with an increased number of cardiovascular events [13]. Therefore, it is

postulated that the association of endothelial dysfunction and the female athlete triad may compromise the cardiovascular system.

## 12.6 Concluding Remarks

Over the last 30 years, participation by girls and women in organized athletics has increased dramatically [8]. President Nixon signed Title IX into law in 1972, which required that all school districts receiving federal funding provide equal opportunities for men and women. Although female sports participation has dramatically flourished in recent years, it has brought about new health concerns for active females, especially where leanness is paramount.

The triad disorders seen in women athletes are interrelated via low EA, amenorrhea, and osteoporosis. Low EA, with or without disordered eating, disrupts physiological function of the female body and notably leads to functional amenorrhea [2, 3, 8, 21]. The energy-deprived athlete triggers suppression of the hypothalamic–pituitary–ovarian (HPO) axis as also referred to as hypothalamic–pituitary–gonadal (HPG) axis in literature. This compromises the menstrual and reproductive system along with endothelial dysfunction [12, 13]. Bone health may be also compromised by the prevalence of stress fractures and is a severe risk factor for osteoporosis [2, 28].

The appropriate diagnosis and management is crucial to ameliorate the health of the female athlete during the triad. Recommendations have been made by various leading organizations to successfully treat this syndrome; however, specific guidelines are lacking. Nevertheless, solid background knowledge of the interrelationships of the various components of the triad is crucial for the allied health professional. The disorders associated with the triad can be prevented and are not a result of exercise or sports performance alone. There are many positive benefits to participation in sports and exercise, and most would argue that the benefits far outweigh the risks. Nevertheless, a systemic overview of the female athlete must be assessed periodically for the prevention of the female athlete triad.

**Table 12.1** Subtle signs of eating disorders

| |
|---|
| • Poor body image |
| • Excessive exercise |
| • Fear of eating in public |
| • Fine body hair known as lanugo (symptom of starvation) |
| • Cooking elaborate meals for others |
| • Dry and blotchy skin |
| • Feeling cold |
| • Swollen cheeks |
| • Fixating on “safe” foods |
| • Strange food combinations |

Adapted from Gardner, A. Subtle signs of an eating disorder. Health. (Accessed February 13, 2013 at http://www.health.com/health/gallery/0,,20665980,00.html)

In the sports arena and educational school system, it is important for coaches, athletic trainers, and educators to be aware of the subtle signs of an eating disorder. Table 12.1 lists subtle signs that can be used to alert professionals to the possibility of disordered eating. Normally, the qualifications of the supporting educational team (coaches, teachers, etc.) for female athletes would limit their ability to counsel an at-risk female athlete, but they could provide guidance and direction. It is hoped that school systems and sport clubs have a referral system in place for these at-risk females. It is also important to educate parents about the subtle sign of an eating disorder. Together, we can make a difference in keeping girls and women to participate in sport and exercise healthily.

## References

1. Otis C, Drinkwater B, Johnson M, et al. ACSM position stand on the female athlete triad. Med Sci Sports Exerc. 1997;29(5):i–ix.
2. Nattiv A, Loucks AB, Manore MM, et al. ACSM position stand on the female athlete triad. Med Sci Sports Exerc. 2007;39(10):1867–82.
3. Manore MM, Kam LC, Loucks AB. The female athlete triad: components, nutrition issues, and health consequences. J Sports Sci. 2007;25(S1):S61–71.
4. Wade GN, Schneider JE, Li HY. Control of fertility by metabolic cues. Am J Physiol. 1996;270:E1–19.
5. Coelho GM, Soares EA, Ribeiro BG. Are female athletes at increased risk for disordered eating and its complications? Appetite. 2010;55:379–87.

6. Beals KA, Hill AK. The prevalence of disordered eating, menstrual dysfunction, and low bone mineral density among US collegiate athletes. Int J Sport Nutr Exerc Metab. 2006;16:1–23.
7. Marquez S. Eating disorders in sports. Risk factors, health consequences, treatment and prevention. Nutricion Hospitalaria. 2008;23(3):183–90.
8. Zach KN, Machin AL, Hoch AZ. Advances in management of the female triad and eating disorders. Clin Sports Med. 2011;30:551–73.
9. Sundgot-Borgen J, Torstveit MK. Prevalence of eating disorders in elite athletes is higher than in the general population. Clin J Sports Med. 2004;14:25–32.
10. American Psychiatric Association Work Group On Eating Disorders. Practice guideline for the treatment of patients with eating disorders. Am J Psychiatry. 2000;157:1–39.
11. American Society of Reproductive Medicine Practice Committee. Current evaluation of amenorrhea. Fertil Steril. 2006;86:S148–55.
12. Lanser EM, Zach KN, Hoch AZ. The female athlete triad and endothelial dysfunction. PM R. 2011;3(5): 458–65.
13. Hock AZ, Papenek P, Szabo A, et al. Folic acid supplementation improves vascular function in professional dancers with endothelial dysfunction. PM R. 2011;3(11):1005–12.
14. Drinkwater BL, Bruemner B, Chesnut CH. Menstrual history as a determinant of current bone density in young athletes. JAMA. 1990;263(4):545–8.
15. Ducher G, Turner AI, Kukuljan S, et al. Obstacles in the optimization of bone health outcomes in the female athlete triad. Sports Med. 2011;41(7):587–607.
16. International Society For Clinical Densitometry Writing Group For The ISCD Position Development Conference. Diagnosis of osteoporosis in men, women, and children. J Clin Densitom. 2004;7: 17–26.
17. Otis CL. Too slim, amenorrheic, fracture-prone: the female athlete triad. ACSM Health Fitness J. 1998; 2(1):20–5.
18. International Olympic Committee Medical Commission Working Group Women in Sport. IOC medical commission position stand on the female athlete triad. http://www.olympic.org/Documents/Reports/EN/en_report_917.pdf Accessed 23 May 2012.
19. Hincapie CA, Cassidy JD. Disordered eating, menstrual disturbances, and low bone mineral density in dancers: a systemic review. Arch Phys Med Rehabil. 2010;91(11):1777–89.
20. Quatromon PA. Clinical observations from nutrition services in college athletics. J Am Diet Assoc. 2008;108(4):689–94.
21. Witkop CT, Warren MP. Understanding the spectrum of the female athlete triad. Obstet Gynecol. 2010; 116(6):1444–8.
22. Herpertz S, Hagenah U, Vocks S, et al. Clinical practice guideline: the diagnosis and treatment of eating disorders. Dtsch Arztebl Int. 2011;108(4):678–85.
23. Putukian M. The female athlete triad. Clin Sports Med. 1998;17:675–96.
24. Williams SE, Watts TK, Wade TD. A review of the definitions of outcome used in the treatment of bulimia nervosa. Clin Psychol Rev. 2012;32(4):292–300.
25. American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-IV-TR (text revision). 4th ed. Washington, DC: American Psychiatric Association; 2000. p. 583–95. 785–788.
26. Davis C, Levitan RD, Yilmaz Z, et al. Binge eating disorders and the dopamine D2 receptor: genotypes and sub-phenotypes. Prog Neuropsychopharmacol Biol Psychiatry. 2012;1:1–8.
27. Filicori M, Tabarelli C, Casadio P, et al. Interaction between menstrual cyclicity and gonadotropin pulsatility. Horm Res. 1998;49:169–72.
28. Ihle R, Loucks AB. Dose–response relationships between energy availability and bone turnover in young exercising women. J Bone Miner Res. 2004;19(8):1231–40.
29. American Society for Reproductive Medicine. Severity of energy-related menstrual disturbances increases in proportion to indices of energy conservation in exercising women. Fertil Steril. 2007;88(4):971–5.
30. Williams NI, Helmreich DL, Parfitt DB, et al. Evidence for a casual role of low energy availability in the induction of menstrual cycle disturbances during strenuous exercise training. J Clin Endocrinol Metab. 2001;86:5184–93.
31. Gamboa S, Gaskie S, Atlas M, et al. Clinical inquiries. What's the best way to manage athletes with amenorrhea? J Fam Pract. 2008;57(11):749–50.
32. Rose MZ, Lee TC, Maffulli N, et al. Special gynecological problems of the young female athlete. In: Maffuli N et al., editors. Sports medicine for specific ages and abilities. London: Churchill Livingstone; 2001. p. 139–47.
33. Hoch AZ, Papenek P, Szabo A, et al. Folic acid supplementation improves vascular function in professional dancers with endothelial dysfunction. PM R. 2011;3(11):1005–12.
34. Abraham SF, Beumont PJ, Fraser IS, et al. Body weight, exercise and menstrual status among ballet dancers in training. Br J Obstet Gynaecol. 1982; 89:507–10.
35. Dusek T. Influence of high intensity training on menstrual cycle disorders in athletes. Croat Med J. 2001;42:79–82.
36. Atlanta Reproductive Health Centre. Menstrual disturbances in female athletes. http://www.ivf.com/amenath.html. Accessed May 20, 2012.
37. Bennell K, Matheson G, Meeuwisse W, et al. Risk factors for stress fractures. Sports Med. 1999;28(2): 91–122.
38. Armstrong DW, Rue JP, Wilcken JH, et al. Stress fracture injury in young military men and women. Bone. 2004;35:806–16.
39. Shaffer RA, Rauh MJ, Brodine SK, et al. Predictors of stress fracture susceptibility in young female recruits. Am J Sports Med. 2006;34:108–15.

40. Miller KK, Lee EE, Lawson EA, et al. Determinants of skeletal loss and recovery in anorexia nervosa. J Clin Endocrinol Metab. 2006;91(8):2931–7.
41. Cumming DC, Cumming CE. Estrogen replacement therapy and female athletes: current issues. Sports Med. 2001;31:1025–31.
42. Misra M, Klibanski A. Anorexia nervosa and osteoporosis. Rev Endocr Metab Disord. 2006;7:91–9.
43. Lo B, Hebert C, McClean A. The female athlete triad no pain, no gain? Clin Pediatr (Phila). 2003;42:573–80.
44. De Souza MJ, Lee DK, VanHeest JL, et al. Severity of energy-related menstrual disturbances increases in proportion to indices to energy conservation in exercising women. Fertil Steril. 2007;88(4):971–5.
45. Gibson JH, Mitchell A, Reeve J, et al. Treatment of reduced bone mineral density in athletic amenorrhea: a pilot study. Osteoporos Int. 1999;10:284–9.
46. Hergenroeder AC. Bone mineralization hypothalamic amenorrhea and sex steroid therapy in female adolescents and young adults. J Pediatr. 1995;126:683–9.
47. Vescovi JD, Jamal SA, De Souza MJ. Strategies to reverse bone loss in women with functional hypothalamic amenorrhea; a systemic review of the literature. Osteoporos Int. 2008;19:465–78.
48. Barrack MT, Van Loan MD, Rauh MJ, et al. Physiologic and behavioral indicators of energy deficiency in female adolescent runners with elevated bone turnover. Am J Clin Nutr. 2010;92(3):652–9.
49. Hoch AZ, Dempsey RL, Carrera GF. Is there an association between athletic amenorrhea and endothelial cell dysfunction? Med Sci Sports Exerc. 2003;35:377–83.
50. Brooks-Gunn J, Burrow C, Warren MP. Attitudes toward eating and body weight in different groups of female adolescent athletes. Int J Eat Disord. 1988; 7:749–57.
51. Tortsveit MK, Rosenvinge JH, Sundgot-Borgen J. Prevalence of eating disorders and the predictive power of risk models in female elite athletes: a controlled study. Scand J Med Sci Sports. 2008;18(1): 108–18.
52. Movaseghi S, Dadgostar H, Dahaghin S, et al. Clinical manifestations of the female athlete triad among some Iranian athletes. Med Sci Sports Exerc. 2012;44(5): 958–65.
53. Thein-Nissenbaum JM, Rauh MJ, Carr KE, et al. Menstrual irregularity and musculoskeletal injury in female school athletes. J Athl Train. 2012;47(1): 74–82.
54. Pantano KJ. Strategies used by physical therapists in the U.S. for treatment and prevention of the female athlete triad. Phys Ther Sport. 2009;10(1):3–11.
55. Becker CB, McDaniel L, Bull S, et al. Can we reduce eating disorder risk factors in female college athletes? A randomized exploratory investigation of two peer-led interventions. Body Image. 2012;9(1):31–42.

# Screening for Disordered Eating and Eating Disorders in Female Athletes

13

Jennifer J. Mitchell and Jacalyn J. Robert-McComb

**Abstract**

Low energy availability appears to be the key etiologic abnormality central to the pathologies seen in the Female Athlete Triad. The energy deficit comes from either increased exertion or inadequate intake of nutrition, either through disordered eating (inadvertent or purposeful) or one of the pathologic eating disorders. It is important to educate female athletes about this concerning scenario as early intervention can limit morbidity and mortality from it. It is important for those interacting with female athletes, both recreational and competitive, to screen for disordered eating/eating disorders (DE/ED) which could lead to low energy availability. With proper education, screening can be done informally by virtually anyone who interacts with female athletes. In the formal setting, question-based tools are available to facilitate the process. Several of these tools are discussed in this chapter. Any athlete who screens positively for possible DE/ED should be referred to a physician and/or mental health provider for further evaluation.

**Keywords**

Low energy availability • Female athlete triad • Menstrual disorders • Eating disorders • Screening female athletes for the triad of disorders

## 13.1 Learning Objectives

Upon completion of reading this chapter, the reader will be able to:

- Understand how disordered eating and eating disorders impact energy availability and health in the female athlete.
- Become aware of the estimated prevalence of disordered eating and eating disorders in female athletes.

J.J. Mitchell, M.D., F.A.A.F.P. (✉)
Department of Family and Community Medicine, Texas Tech University Health Sciences Center School of Medicine, 3601 4th Street, MS8143, Lubbock, TX 79430, USA
e-mail: jennifer.mitchell@ttuhsc.edu

J.J. Robert-McComb, Ph.D., FACSM, ACSM P.D.
Department of Health, Exercise, and Sports Sciences, Texas Tech University, 3204 Main Street, Lubbock, TX 79409, USA
e-mail: jacalyn.mccomb@ttu.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_13, 

- List some of the sport-related and nonsport-related risk factors for disordered eating and eating disorders.
- Understand reasons for and methods of screening for disordered eating and eating disorders in the female athlete in both informal and formal settings.
- Become aware of screening tools that are utilized for disordered eating and eating disorders in general and specifically in athletes.

## 13.2 Introduction

There is no clearly defined standard of methods or timing for screening female athletes for disordered eating (DE) or eating disorders (ED). Several screening tools are available, but no consensus exists yet concerning the optimal tool for use with athletes. Various opportunities present themselves for screening athletes for disordered eating/eating disorders (DE/ED), but no single time has proven most advantageous. Screening may be performed in various ways, but it is optimal to gather as many objective pieces of evidence as possible, since denial by the athlete is often a large component of DE/ED. Ultimately, ideal screening is specific to each athletic level and entity, whether recreational or competitive. This chapter discusses various methods and timing of screening female athletes for disordered eating and eating disorders.

## 13.3 Research Findings

### 13.3.1 Energy Availability Related to Disordered Eating and Eating Disorders

The American College of Sports Medicine Position Stand on the Female Athlete Triad describes the interrelationships between energy availability, menstrual function, and bone mineral density. For each of these three areas, each athlete may be described along a continuous spectrum between health and disease. At the disease end of the spectrum lie eating disorders, functional hypothalamic amenorrhea, and osteoporosis. Low energy availability appears to impair both reproductive and skeletal health. These pathological conditions typically exist subclinically until a pathological event manifests [1]. An athlete may be at various points along the three spectra between health and disease and typically will not manifest all three disorders at the same time [2].

Low energy availability may occur from disordered eating (DE) or a true eating disorder (ED). Disordered eating encompasses various abnormal eating behaviors that are inadvertent, such as inadequate refueling, or they may be intentional. An eating disorder is a clinical mental disorder meeting diagnostic criteria as defined by the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). Eating disorders include anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified [3]. Binge-eating disorder will be included as a separate eating disorder in the DSM-V to be published in mid-2013.

Energy availability is the energy remaining for body functions after that used for exercise is subtracted from total energy intake. Continuous low energy availability with or without disordered eating can impair health. Ways an athlete's available energy may be reduced include: increased energy expenditure with excessive exercise, reduction of energy intake, abnormal eating behaviors such as binging and purging or use of laxatives or diuretics, or eating disorders which are often accompanied by other psychiatric illnesses [1].

### 13.3.2 Prevalence of Disordered Eating/Eating Disorders in Athletes

It is difficult to know the exact prevalence of DE/ED in female athletes as the majority of studies have various flaws including the use of nonstandard diagnostic procedures, small sample sizes, lack of or inadequate control group(s), inadequate statistics, and/or heterogeneous athlete population [1, 4].

There have only been two, well-controlled studies utilizing DSM-IV criteria for diagnosis of EDs. These were conducted with elite athletes

and demonstrated 31 % prevalence in athletes compared to 5.5 % in the control population for the first study and 25 vs. 9 % in the second study. Other studies have shown secondary amenorrhea to be as high as 69 % in dancers and 65 % in long distance runners versus 2–5 % in the general population [1].

The first study to look at the combined prevalence of disordered eating, menstrual dysfunction, and low bone mineral density in college females, demonstrated that the number of athletes suffering from all three disorders of the triad was small (1–3 athletes out of 112). However, a significant number suffer from the individual disorders of the female athlete triad [2].

A more recent systematic review of 65 studies evaluating the prevalence of the individual and combined components of the triad verified that initial study. It showed a relatively small percentage of athletes (0–15.9 %) exhibited all three components of the triad. The prevalence of any two triad conditions ranged from 2.7 to 27 %. The prevalence of any one condition was highest, from 16 to 60 %. The recommendation from that review is that additional research on the prevalence of the triad using objective and/or self-report/field measures is necessary to more accurately describe the extent of the problem [5].

### 13.3.3 Risk Factors for Disordered Eating/Eating Disorders

There are multiple risk factors, which predispose an athlete to DE or ED. This list includes four major groups of factors as in Table 13.1.

Further evaluation of the sport-specific factors seems warranted given conflicting data. Studies in 2002 (Byrne and Mc Lean) and 2006 (Sundgot-Borgen and Torstveit) indicate that EDs are more likely to occur in athletes in leanness sports such as gymnastics, cross country and figure skating compared with athletes in non-leanness sports and controls. This was again verified in 2008 by Torstveit, Rosenvinge, and Sundgot-Borgen in a study with 186 athletes compared to 145 controls. EDs were more common in athletes in leanness sports (46.7 %) compared to non-leanness sports (19.8 %) and controls (21.4 %) [7]. Beals provides evidence to question the long held belief that EDs are more common in athletes in leanness sports in the small study of 112 athletes mentioned above. She specifically notes that the percentages of individuals with DE and bone mineral density disorders, individually or in combination, were similar between lean build and non-lean build sports. The implication from this is that all female athletes, regardless of sport, should be screened for components of the female athlete triad and intervention should begin early to prevent development of the full triad [2, 8].

In any event, avoiding external pressure on the athlete to lose weight is essential to avert preoccupation with dieting as it is considered to be the number one trigger for EDs [1, 6].

**Table 13.1** Risk factors for disordered eating/eating disorders [1, 6]

| | |
|---|---|
| I. | Nonsport-related factors |
| | A. Biological factors |
| | 1. Pubertal status |
| | 2. Pubertal timing |
| | 3. Body mass index |
| | B. Psychosocial factors |
| | 1. Body image dissatisfaction |
| | 2. Mood disorders |
| | 3. Low self-esteem |
| | 4. Perfectionism |
| | 5. Family dysfunction |
| | C. Sociocultural factors |
| | 1. Perceived pressure to conform to an unrealistic standard of thinness |
| II. | Sport-specific factors |
| | A. Sports that emphasize |
| | 1. Appearance |
| | 2. Thin body build |
| | 3. Low body weight |
| | B. Sports that require weight classifications |
| | C. Early sport-specific training |

### 13.3.4 Screening for Disordered Eating/Eating Disorders

In the last 5 years, two position stances from major sports medicine associations have been

developed that address screening athletes for DE/ED, yet still no clear standard exists as to the optimal method and tool for screening [1, 6]. Screening can occur at a common entry point to athletic participation such as during pre-participation physical examinations (PPEs), but should also be an ongoing process throughout the span of an athlete's participation. This ongoing process is particularly applicable to recreational athletes who participate in exercise clubs, individual activities, and "weekend warrior" activities. The screening process should be viewed as a two-step process [9, 10]. Once an athlete screens positively or if a concern exists, the athlete should be referred for further medical and psychological or psychiatric evaluation.

#### 13.3.4.1 Reasons to Screen

The hope behind screening is to identify athletes who likely have significant levels of eating pathology and require further assessment. Screening can be a complex and challenging task, but the sports medicine team must keep in mind the reasons why screening is important. These include [1, 6, 11]:

- Prevention of DE/ED; the most effective way to decrease the incidence of eating disorders is to prevent them.
- Early intervention when DE/ED exists to minimize impacts on health and performance; the longer an ED is allowed to persist and progress without treatment, the greater the health and performance detriments.
- Athletes tend to deny or do not realize a problem exists.
- Athletes are unlikely to come forward on their own, so complications from low energy availability can go unrecognized until a major event such as a stress fracture occurs. There are several reasons why the athlete may not come forward including guilt, shame, fear of losing a scholarship, or fear of losing playing time [6].

#### 13.3.4.2 Screening Practices

With no clearly defined standard for screening athletes for DE/ED, practices fall across a wide spectrum that includes: no specific screening, a few general questions at the time of PPE, utilization of a self-report questionnaire screening tool (SRQST) at PPEs, or the use of a SRQST combined with an interview by a trained mental health provider.

A 2012 study evaluated PPE forms utilized at 257/347 NCAA Division I universities for efficacy in screening for the female athlete triad. It compared those forms to the 12 items recommended by the Female Athlete Triad Coalition for screening females for the triad [12]. Only 25 universities (9 %) had nine or more of the 12 recommended items on their forms [13].

Another study has shown that only 60 % of Division I schools, which responded to the study, screened for eating disorders during PPEs. Of those that did screen, <6 % used a standardized self-report questionnaire [14].

Because the goal of the PPE is to facilitate optimal performance for athletes while ensuring the best possible health for the athlete both today and in her future, it has been suggested to implement a separate supplemental health questionnaire specific to female athletes. It is felt that this method would allow health care providers to narrow in on female-specific issues. It might be implemented before, during, or shortly after PPEs on campus [15].

An interesting innovation in screening female athletes for DE/ED is the physiologic screening test consisting of 18 items (4 measurements and 14 questions). It has been validated and has the potential to be combined with one of the athlete-specific questionnaires (see Table 13.4) to create a two-step screening process in an attempt to minimize false positives and false negatives prior to psychological referral [8].

#### 13.3.4.3 Functionality of Screening Tools

What makes a screening tool useful is functionality as well as validity. SRQSTs seem more functional than interviewer applied tools. However, they are subject to report bias, as athletes tend to be not as forthright with these as in a one-on-one interview [2, 16]. The interview tools are more appropriate for an in-depth evaluation, in search of a specific diagnosis, but they are time intensive

and require education on the part of the interviewer. They are most useful as the second step in evaluation, once an initial screening tool is positive or when there is reason to suspect DE/ED in an athlete [10].

In the athletic arena, time is frequently an issue. It is essential for a screening tool to be focused and time limited. Formal screening tools are ideally brief, self-report questionnaires with simple cut-off scores that indicate a level of dysfunction concerning for pathology in the athlete [16].

One must be assured that the tool utilized, when interpreted as positive, truly indicates an issue for which further evaluation and time requirements will be needed. Once a screening tool is positive, the athlete should then have a more formal evaluation to determine whether true pathology exists or risk factors for pathology are present. This includes a detailed medical, nutritional, and reproductive history and physical examination with lab evaluation by a physician and referral to a psychologist or psychiatrist [12, 17]. The ideal tool for further evaluation is a structured interview. Eating disorders exam (EDE) has been identified as the gold-standard tool for identification of eating disorders in general [8, 16, 18]. During the one-on-one interview, the athlete must feel secure and not threatened [6].

In organized sports, the PPE is a common entry point for evaluation of athletes. Screening at that time is of utmost importance, but it is not the only opportunity to diagnose DE/ED. Screening female athletes for DE/ED needs to by a dynamic, ongoing process, throughout the span of recreational and competitive activity. It should not occur in a vacuum, only at the time of a PPE.

Recreational athletes can also fall into low energy availability from DE/ED. A less formal approach to screening may be applicable in their case.

### 13.3.5 Screening Settings

Screening for disordered eating or eating disorders (DE/ED) in athletes occurs in both informal settings, mostly by observation and interaction with athletic trainers and coaches, and formal settings, typically with a team physician or primary care provider or when referred to a psychologist or psychiatrist.

#### 13.3.5.1 Informal Settings

The informal setting occurs in the athlete's day-to-day routine while interacting with athletic trainers, coaches, administrators, teammates, teachers, family, and friends. For recreational athletes, informal screening may occur with personal trainers, group exercise leaders, and gym personnel. The ideal is for all individuals interacting with athletes to be educated on recognizing concerning patterns of behavior and exercise (nutrition issues, over-exercising, etc.). Once educated on what to look for, he/she can feel empowered to approach the athlete in an effort to assist her. Written policies on dealing with suspected eating disorders are recommended and adequate resources to assist the athlete are ideal [19, 20].

Each individual interacting with the athlete has the opportunity to informally screen the athlete for DE/ED. Whether they actually do, often depends upon their level of education and whether they are alert to a potential issue with the athlete [6, 21].

Direct questioning can be utilized, however the nature of eating disorders tends to be secretive. It is likely that the individual will not readily disclose the embarrassing symptoms of an eating disorder, such as vomiting or laxative use. The intensity of questioning has to be balanced between the relationship of the athlete with the person probing and the athlete's readiness to disclose her illness. Thus, the allied health professional sometimes must read between the lines and look for physical and behavioral characteristics that may signify an eating disorder.

Some physical findings include [1, 6, 17, 22]:

Poor exercise tolerance including dehydration, cramping, pre-syncope, and bradycardia

Hair, skin, or teeth changes including lanugo, alopecia, dry skin, callouses on hands and/or loss of tooth enamel from induction of vomiting

Gastrointestinal upset including bloating, diarrhea or constipation, abdominal discomfort

Complaints of menstrual irregularities

Once the athlete's trust is gained, a variety of questions can be utilized to attempt to further delineate behavioral characteristics. Areas to be explored include [1, 6, 17, 22]:

Eating behaviors such as binging, purging, eating in secret, recurrent dieting

A history of or current mood disorder to include sadness, depression, or anger

Use of extreme weight control measures to include starvation, use of diuretics, use of laxatives, use of saunas

People close to the athlete often contribute barriers to recognition of the issue. This is often inadvertent, but can also be intentional.

These barriers include [11]:

1. Coaches
   a. Lack of knowledge, experience, or resources to address the problem
   b. May not want to interfere with an athlete successfully training and performing, despite concerns about DE/ED
   c. Fear of being accused of creating or contributing to the DE/ED behaviors
2. Teammates
   a. Fear of breaking the trust of a team member
   b. May self-reflect upon her own DE, creating irrational fears that identifying a teammate's issue will expose her
3. Family/Parents
   a. Desire to see child succeed regardless of the consequences
   b. Feel unsure how to approach disordered eating behaviors
4. Administrators
   a. May feel they lack knowledge, experience, or resources to address the problem
   b. May fear feelings of inadequacy or challenges from others for not having been proactive in previously establishing resources or policies

In order to minimize barriers, it is critical to maintain an environment that promotes the clear expectation that DE/ED will be addressed with the intent to promote optimal health and performance for the entire team. This may minimize the concern for a "telltale" environment.

It is a responsibility of those who are close to the athlete to help recognize DE/ED and initiate further evaluation and assistance [11]. Once it is recognized that assistance is needed, screening becomes formalized in the clinical setting with the team physician or primary care provider.

#### 13.3.5.2 Formal Settings

The formal, structured setting occurs during pre-participation examinations and in the clinical setting. In the formal setting, SRQSTs are best utilized. A questionnaire tool is especially helpful as it can be difficult for the provider to remember the myriad of questions recommended for picking up on subtleties in order to discover DE or recognize an athlete attempting to hide an ED.

### 13.3.6 Pre-participation Examinations

The main benefit of screening during PPEs is that medical personnel are able to quickly review the responses to the tool utilized, and potentially, they can immediately refer the athlete who screens positively. All new athletes are required to have a PPE, so all would be screened, at least, in this format. The disadvantage of screenings during PPEs is that they often occur in a station-centered setting, such as in an athletic training room. This provides minimal privacy and confidentiality in completion of questionnaires and in further discussions with the individual athlete. Although, in order to enhance confidentiality and improve efficiency, some universities are shifting to having athletes complete health histories either before arrival on campus or on web-based sites [23]. As technology and the patient-centered medical home (PCMH) advance, a web-based data center in an electronic health record may become the standard. The PCMH promotes organizing care around patients, working in teams, and coordinating and tracking care over time [24].

Another negative aspect of screening at PPEs is that there are typically multiple other forms to complete. The athlete may then rush through the

DE/ED screening tool, not taking the time to answer accurately [11, 13].

Female athletes often feel uncomfortable discussing disordered eating during PPEs and are more likely to withhold information [11]. This is another reason why screening females with a supplemental form within the first few weeks of arrival on campus may be a better method.

An additional concern arises in settings where PPEs are only required at entry and not yearly. In a study of NCAA Division I universities, of the 257 (74 %) schools that participated, only 32 % require an annual PPE. In this case, if the athlete develops risk factors for DE/ED after her freshman year PPE, it may go undetected until a significant health event occurs, if at all [13].

### 13.3.7 Clinical Encounters

The other formal setting where the female athlete may be encountered is in the clinical setting when presenting for routine health care or for an acute illness or injury. The clinician then has the opportunity to screen for components of the female athlete triad, including those that set the athlete up for low energy availability (DE/ED). A full medical, reproductive, and skeletal health history should be taken as well as an appropriate physical examination looking for classic signs of eating disorders [12, 17].

Questions to be asked during the history should also include nutrition questions incorporating weight and dieting history, current exercise regimen looking for any recent changes in intensity or amount, and mood-related questions.

Physical complaints and findings such as amenorrhea, gastrointestinal disturbances, low body mass index, bradycardia, orthostatic hypotension, skin changes, and laboratory studies can help diagnose an eating disorder [25]. However, during the early course of an eating disorder, physical examination, and laboratory findings may be normal.

Again, there are time constraints in the clinical setting and the provider is likely to focus specifically on the illness, injury, or well woman examination at hand and not expand the history to include elements important in identifying ED/DE and female athlete triad disorders. Health providers (athletic trainers, team physicians, sports medicine fellows, physician assistants, nurse practitioners) working with female athletes need to remember to focus on their medical roots to complete an entire history and physical examination looking for symptoms and signs of DE/ED and female athlete triad disorders.

### 13.3.8 Tools

There are multiple screening tools for disordered eating and eating disorders in the literature. Some are specific to athletes, while others are general nutritional DE/ED screening tools. Most of the general tools are validated, but few of the tools specific to athletes have been validated in female athletic populations [1, 11].

A screening tool may save time obtaining the athlete's history either before or as a part of a PPE or in the setting of a clinical visit with the physician. Questions may be incorporated into the PPE form or a supplemental screening tool may be utilized. The American College of Sports Medicine Position Stand on the Female Athlete Triad and the National Athletic Trainers' Association Position Statement on Preventing, Detecting and Managing Disordered Eating in Athletes make the recommendation for screening during PPEs, but provide no guidance on any particular tool [1, 6]. It is generally felt that a supplemental tool directed specifically at female athletes may ultimately be the recommended ideal.

The SRQST is utilized as a first step. These tools are not designed to diagnose an eating disorder so athletes who screen positively, should then be further evaluated by a physician for medical evaluation and referred to a psychologist or psychiatrist. During that visit it is likely that one or more interview-based tools will be utilized to determine if the diagnosis of an eating disorder is appropriate.

**Table 13.2** General screening tools for disordered eating/eating disorders, not athlete specific

| Tool | Year | Key points | Validation |
|---|---|---|---|
| EAT-26 [10, 16] | 1982 revised from original EAT-40, 1979 | Most widely used standardized self-report measure of symptoms and concerns characteristic of EDs specifically<br>Web-based; easily accessible; free | Score of 20 or more—interview by a qualified professional to evaluate for diagnostic criteria for ED; concurrent validity; good discriminate validity ChEAT-children's version |
| SCOFF questionnaire [26, 27] | 1999 | 5 questions; 1–2 min to complete | Two or more + responses, 100 % sensitivity |
| Eating disorders exam-questionnaire (EDE-Q) [27–30] | 1994 | Self-completed, question form of EDE<br>Widely used measure of eating disordered behavior<br>36 items; 15 min to complete<br>Overestimates binge-eating frequency compared to EDE | Yes; criterion validity |
| Eating disorder invenory-3 (EDI-3) [9, 31–33] | 2011 | Developed from EDI (1983) and EDI-2 (1991)<br>91 questions; 12 subscales; 6 composite scores<br>20 min to complete<br>Cost associated | Clausen validating in 2011 EDI-C children's version |
| Eating disorder screen for primary care (ESP) [34] | 2003 | 4 questions; 1–2 min to complete | As effective as SCOFF |
| Bulimia test-revised BULIT-R [16, 35] | Revised 1991 | Bulimia nervosa screening; 28 question | Content construct criteria |
| NEDA screening program [36] | Yearly, March | Evaluates resources of colleges and universities; online screen for students | No |

Self-report questionnaire *screening tools*; not used to diagnose ed's

First step in a two stage process

### 13.3.9 General Screening Tools for Disordered Eating/Eating Disorders, Nonathlete Specific (Table 13.2)

#### 13.3.9.1 Self-Report Questionnaire Screening Tools

**EAT-26**

This is the most widely used standardized self-report measure of symptoms and concerns characteristic of eating disorders specifically. It has three subscales: dieting, bulimia and food preoccupation, and oral control. EAT-26 is a refinement of the original EAT-40 that was first published in 1979. This tool is easily accessible as it is web-based and free. Scoring instructions are included on the website. It can be administered in group or individual settings and does not have to be administered by a mental health or medical professional. A score of 20 or more should prompt referral for interview by a qualified professional to determine whether diagnostic criteria for an ED exist. It is valid and reliable. Ch-EAT is the version used in children [10].

**SCOFF Questionnaire**

This was developed in 1999 in Great Britain as a quick and easy to remember screening tool for clinicians. The use of a mnemonic with yes/no responses, similar to the CAGE questions for alcoholism, is intended to simplify screening. There are five questions, which take between 1 and 2 min to administer. In the original study, two or more positive answers provided 100 % sensitivity [26]. One question is written in Queen's English referring to weight in stones. An "Americanized"

version, with the value in pounds, was developed for use in research comparing SCOFF to another screening tool [27].

#### Eating Disorders Exam-Questionnaire (EDE-Q)

This tool was devised in 1994 and is a self-completed questionnaire form of the EDE, which is an interview-based tool administered by a qualified professional to diagnose eating disorders. It is a widely used measure of eating disordered behavior. The tool consists of 36 items and takes about 15 min to complete. It focuses on the past 28 days and is scored using a 7-point scale. The four subscales included are restraint, eating concern, weight concern, and shape concern. It has good criterion validity. Compared to the EDE, it does tend to overestimate binge-eating frequency [27–30].

#### Eating Disorder Inventory-3 (EDI-3)

This was developed in 2004 as an expansion and improvement upon Eating Disorder Inventory-2 (EDI-2) from 1991 and the original EDI in 1983. At the time, EDI-2 was already recognized as a standard self-report measure for ED assessment in the international health care community. EDI-3 evaluates for psychological traits and symptoms relevant to the development and maintenance of anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified. It consists of 91 items broken into 12 subscales (broken down into ED risk scales versus psychological scales) and provides 6 composite scores. On average, it takes about 20 min to complete. This tool can be accessed through the Internet, but there is a cost associated. EDI-C is available for use with children [9, 31–33].

#### Eating Disorder Screen for Primary Care (ESP)

This was developed in 2003 in Great Britain in an attempt to generate a short screening tool that could both rule in and rule out EDs. It consists of four questions and takes 1–2 min to complete. It is not validated. One study compared it directly to SCOFF and it was found to be equally effective [34].

#### Bulimia Test-Revised (BULIT-R)

This is a 28-question tool that is easy to score and is well validated. It is a revision from the original BULIT. This instrument has been shown to be a reliable and valid measure for identifying individuals who may suffer from bulimia nervosa both in clinical and nonclinical populations [16, 35].

#### National Eating Disorders Association (NEDA) Screening Program

This is an online eating disorder screening. There are two separate questionnaires; one for college students and one for the general population. It provides a free, anonymous self-assessment to gauge one's risk of an eating disorder. It takes only a few minutes and consists of a series of questions designed to indicate whether clinical help may be needed. After completing a screening, if indicated, participants will receive referral information through NEDA's Helpline for personal evaluation by a medical professional and treatment. This is considered a good resource for people who may need help or know someone who may need help and don't know where to begin. NEDA also provides the annual Collegiate Survey Project, each year in March. This is a compilation of responses from 165 colleges and universities concerning on-campus resources for eating disorder-related programs [36].

### 13.3.9.2 Interview-Based Tools

The clinical interview is the assessment tool of choice when diagnosing eating disorders as it allows for more detailed questioning. It is part of the second step in evaluation when a screening tool is positive (Table 13.3) [16].

#### Eating Disorders Exam (EDE)

This semi-structured interview is recognized as the method of choice for diagnosing eating disorders, specifically anorexia nervosa and bulimia nervosa. It was developed in 1987 and revised in 1993. The interviewer, not the subject, rates the severity of symptoms. It focuses on a 28-day time frame over the previous weeks. There are 62 items and it can take over an hour to administer. There are two behavioral indices (overeating and methods of extreme weight control) and four

**Table 13.3** Interview-based tools—administered by qualified professional; second stage after screening

| Tool | Year | Key points | Validation |
|---|---|---|---|
| Eating disorders exam (EDE) [16, 28, 29] | 1987 revised 1993 | Interview-based, semi-structured interview | Yes |
| | | Gold standard of eating disorder assessment, specifically AN and BN | Good criterion validity |
| | | 28 day time frame, prior 4 weeks | Questionable construct validity |
| | | 62 items; 2 behavioral indices; 4 subscales | Not in athletic population |
| | | 30–60 min to administer | |
| Interview for diagnosis of eating disorders (IDED)-IV [9, 16] | 1990 revised 1998 | Semi-structured interview | Not in athletic population |
| | | Specifically for diagnosing EDs, not DE; based on DSM-IV criteria | Good reliability and validity |

*AN* Anorexia nervosa, *BN* Bulimia nervosa

subscales (restraint, eating concern, shape concern, and weight concern). Administration is by a clinician with specific training in the use of this interview [16, 28, 29].

#### Interview for Diagnosis of Eating Disorders (IDED)-IV

This semi-structured interview was revised in 1998, after the original in 1990, for the purpose of discriminating between eating disorders and subthreshold syndromes, which it does. It has good reliability and validity. The rater uses severity scales on a diagnostic checklist that leads directly to the differential diagnosis using DSM-IV criteria. It is a reasonable alternative to EDE [9, 16].

If a generalized screening tool will be used, EAT-26 or EDE-Q are the most widely used self-report questionnaires. When time and resources are available or an athlete screens positively, the interview-based EDE is an ideal.

### 13.3.9.3 Self-Report Questionnaire Screening Tools, Athlete Specific (Table 13.4)

There are a limited number of tools, which have been designed specifically for female athletes. Some of the tools available screen both athletes and college students whether female or male. Another method of screening is through questions incorporated into a PPE form. In those, typically any nutrition questions will be directed at females and males. The following section of the form then has questions specific to females. Unless this is clearly delineated, this can be confusing for the athletes during completion of their history.

#### Female Athlete Screening Tool (FAST)

This tool was developed in 2001 to identify disordered eating and atypical exercise and eating behaviors among female athletes. It has 33 questions. It has internal reliability and concurrent validity to EDI and BULIT-R [37].

#### Health, Weight, Dieting, and Menstrual History Questionnaire (HWDMHQ)

This was the first study to assess the combined prevalence of all three components of the female athlete triad. The study showed that very few athletes demonstrate all three components, but a significant number suffer from the individual disorders of the triad. It was developed from the EDI symptom checklist and EDE-Q in 2002 and revised in 2006 [2].

#### Physiologic Screening Test

This tool was developed in 2003 to provide a physiologic screening test, specifically for collegiate female athletes competing at a high level, in order to detect DE/ED. It takes 15 min to complete and consists of 18 items: 14 questions and 4 physiologic measurements (percent body fat, waist:hip ratio, standing diastolic blood pressure, enlarged parotid glands). It outperformed the EDI-2 and BULIT-R on the false-negative rate, negative predictive value, yield, overall accuracy, and validity [8].

#### Female Athlete Triad Screening Questionnaire

This is a questionnaire available, free of charge, on the Internet. The Female Athlete Triad Coalition is

**Table 13.4** Self-report questionnaire screening tools, female athlete specific

| Tool | Year | Key points | Sex |
|---|---|---|---|
| Female athlete Screening tool FAST [37] | 2001 | 33 questions | F |
| | | To identify DE and atypical exercise and eating behaviors | |
| | | Internal reliability; concurrent validity to EDI and BULIT-R | |
| Health, weight, dieting, and menstrual history questionnaire HWDMHQ [2] | 2002 updated 2006 | First study to assess combined prevalence of all three components of female athlete triad | F |
| | | Developed from: | |
| | | EDI symptom checklist | |
| | | EDE-Q | |
| Physiologic screening test (PST) [8] | 2003 | 18 items: | F |
| | | Four Physiologic measurements | |
| | | 14 Questions | |
| | | 15 min to complete | |
| | | Validated; better than EDI-2 and BULIT-R | |
| Female athlete triad coalition screening questionnaire [12] | 2002 | Internet accessible | F |
| | | 12 questions: nutrition, 8; menses, 3; bone health, 1 | |
| | | If positive, follow by in-depth evaluation with detailed | |
| | | history of 19 questions and full medical evaluation | |
| Athletic milieu direct questionnaire AMDQ [9, 10, 38] | 2000 | 19 questions | F |
| | | Designed to assess DE/ED | |
| | | Compared to EDI-2 and BULIT-R, superior results on 7 of 9 | |
| | | epidemiologic analyses | |
| | | First instrument to operationalize the construct of DE | |
| | | Not validated in a clinical population | |
| ATHLETE [39] | 2005 | Female athletes at three division I universities | F |
| | | 6 subscales from EDI, modified to athletes | |
| | | Developed to assess psychological predictors of disordered | |
| | | eating in female athletes | |
| | | Construct validity confirmed by convergent and discriminate validity | |

sponsored by several sports medicine organizations and has existed since 2002.

The initial screen has 12 questions: nutrition, 8; menses, 3; bone health, 1.

If positive, an in-depth evaluation with a detailed history of 19 questions and a full medical evaluation are recommended [12].

#### Athletic Milieu Direct Questionnaire (AMDQ)

This was designed in 2000 to assess DE/ED in female athletes. It is the first instrument to operationalize the construct of DE. It consists of 19 questions evaluating behaviors relevant to weight management, diet, and exercise. It has not been clinically validated, but compared to EDI-2 and BULIT-R it has superior results on seven of the nine epidemiologic analyses [9, 10, 38].

#### ATHLETE

This tool was developed in 2005 to be administered to female athletes at three Division 1 universities. It is used to recognize psychological predictors of DE. There are six subscales from EDI, which were modified to athletes [39].

### 13.3.9.4 Non-gender-Specific Eating Disorder Tools (Table 13.5)

#### College Health Related Informational Survey (CHRIS)

This was developed in 2003 as a new screening instrument for college student athletes. It was based on the Juvenile Wellness and Health Survey. There are 32 questions broken down into four areas: mental health, 9; eating problems, 12; risk behaviors, 4; performance pressure, 6.

**Table 13.5** Self-report questionnaire screening tools, athlete specific

| Tool | Year | Key points | Sex |
|---|---|---|---|
| College health-related information survey CHRIS [40] | 2003 | College student athletes | F, M |
| | | Based on juvenile wellness and health survey (JWHS) | |
| | | 32 questions broken into four areas: mental health, 9; eating problems, 13; risk behaviors, 4; performance pressure, 6 | |
| | | Needs further validation | |
| Survey of eating disorders among athletes SEDA [41] | 1991 | 33 questions; self-reported eating pathology | F, M |
| | | Athletic environment-related risk factors | |
| | | Not validated in athletic population | |
| | | Student athletes and students | |
| De Palma [41] | 2001 | ID pathologic eating in college students and athletes | |
| | | 16 questions; 8 from SEDA and 8 from DSED-diagnostic survey EDs | |
| PPE monograph [42] | 2010 | 4 questions related to weight; 3 questions related to menses | F, M |
| International Olympics Committee screening [43] | | Athlete periodic health evaluation (PHE) form | F, M |
| | | 11 Nutrition questions for both sexes | |
| | | Female-specific questions: 6 menses, 2 bone health, 1 STI | |
| Stanford website [17] | | Questions as part of PPE questionnaire | F, M |

### De Palma

This was devised in 2001 as a discriminate analysis tool to identify college students and student athletes at low, moderate, or high risk of pathologic eating. It was not given any specific title so is referred to here by the first authors last name. It has 16 questions, 8 each from two different previously used instruments, diagnostic survey of eating disorders (DSED) and survey of eating disorders among athletes (SEDA). It takes about 2 min to complete and 2 min to score. The items are short and relatively nonconfrontational [41].

### Survey of Eating Disorders Among Athletes (SEDA)

This is a survey of collegiate females and males, who are both athletes and students. It consists of 33 questions related to self-reported eating pathology. It has not been validated in an athletic population [41].

Standardized PPE forms are directed at both female and male athletes. There are a myriad of those types of forms available. The following will discuss two of the more commonly used forms and an example of an Internet-based PPE form located online for the athlete to complete in advance of arrival for a PPE. Many collegiate athletic departments are beginning to utilize this technology. Given that the health care system is moving toward a PCMH, where patient information is stored electronically with ongoing updates, online storage of electronic data recorded in a PPE form may eventually be a recommended best practice. The National Committee for Quality Assurance is promoting the PCMH to allow for organizing care around the patient, working in health teams, and coordinating and tracking care over time [24]. The ability for health care providers to access the athlete's information electronically may improve the quality of care they receive and may make research related to athletes easier.

### Pre-participation Physical Evaluation, Fourth Edition

The latest revision of this form occurred in 2010. It has four questions concerning weight issues that are directed at both females and males. There are three questions related to menses [42].

### International Olympic Committee Periodic Health Evaluation of Elite Athletes

This form has 11 nutrition and weight-related questions for both females and males. There are nine questions directed at the female athlete's reproductive and/or skeletal health (6 menses, 2 bone health, 1 sexually transmitted infections) [43].

**First Year Varsity Athletics Pre-participation Medical Examination form for Stanford University Department of Athletics**

This is an 85 question PPE form for athletes entering into the Stanford University system. It is designed for female and male athletes. There are nine questions covering weight/nutrition/eating habits, two questions for males only concerning the reproductive system and five questions for females regarding menses [23].

When a self-report screening tool is utilized, the timing and setting for its use must be considered. The tools that appear to be most useful are FAST, AMDQ, and HWDMHQ. The Physiologic Screening Test appears to have potential. However, ongoing validation of these tools must continue to occur. If screening occurs during PPEs the use of a supplemental tool for female athletes is optimal.

## 13.4 Contemporary Understanding of the Issues

Screening female athletes for disordered eating and eating disorders is a complex issue. Those involved with active females need to encourage screening on multiple levels, both formally and informally, utilizing a combination of timing and methods (observation, standardized SRQST, interviews). Screening needs to occur as an ongoing process, not only occurring as an isolated event during pre-participation examinations. Education of those involved with female athletes, on all levels, will help in ongoing informal recognition of signs and symptoms of disordered eating and eating disorders. This is essential because the longer that low energy availability is allowed to exist, the greater the health and performance impairments that occur and the more difficult they are to treat.

For competitive athletes, formal screening should occur, either immediately before, during, or immediately after, PPEs. A national standard should be encouraged, with a supplemental SRQST specific to females rather than questions incorporated in the PPE form that is utilized for both males and females. The athletic trainer or team physician or both should then review the tool. Those athletes who screen positively should be evaluated, beyond the standard pre-participation exam, by the team physician or primary care provider. This evaluation should include a detailed medical, reproductive and skeletal history, physical examination incorporating examination for findings specific to those with eating disorders and appropriate labs and additional studies. A referral to a mental health professional should also occur for further evaluation and diagnosis whether an eating disorder exists. The mental health provider is likely to utilize a battery of tests evaluating various risk factors for eating disorders including mood screening. The gold standard for evaluation of an eating disorder is the interview-based EDE.

Physicians need to be reminded to screen female athletes for risk factors for low energy availability during routine health visits and those for acute illness or injury. The supplemental self-report questionnaire should be specific to the female athlete. Validated tools include FAST, HWDMH, and PST [2, 8, 37].

Those that are female athlete specific, but have not been validated include AMDQ, Female Athlete Triad Coalition Questionnaire, and ATHLETE.

If for some reason a female athlete-specific questionnaire cannot be utilized, consideration should be given to a tool to screen for eating disorders in a general population that is inexpensive and easily accessible such as EAT-26.

The ideal will be to have a consensus on a nationally or internationally recognized SRQST with validation of that tool. This will help with standardized early recognition and treatment for disordered eating and eating disorders in an effort to optimize energy availability, health and performance in female athletes.

## 13.5 Future Directions

The sports medicine community can serve its female athletes well by developing a consensus related specifically to screening for disordered eating and eating

disorders. A standardized form/tool that the athlete completes prior to PPE or a physician's visit for a health issue would be ideal. The challenge has been twofold: to find a consensus about questions that need to be asked on a survey tool and to achieve validation of any such tool. A supplemental form specific to the female athlete could prove to be the most functional. A method of rapid assessment of that form would then allow the provider to determine whether further referral should be made the same day, or if ongoing monitoring may be needed. Incorporation of physiologic variables into a screening tool shows promise and should be further evaluated.

Simplified education programs for all people who interact with athletes should be developed and distributed nationally in an effort to identify disordered eating and eating disorders early. Methods of screening for these conditions should be covered in these programs so that screening will become an ongoing process in both informal and formal settings where female athletes are encountered. Further, programs of education and screening should be expanded into junior high and high schools to identify issues of low energy availability as early as possible.

A PCMH designed specifically for athletes may prove to enhance the overall care provided throughout the career of a competitive athlete. This would include ongoing updates of the athlete's sport specific and basic medical health data in an electronic format. This allows for organizing care around the athlete, working in health teams, and coordinating and tracking care over time as recommended for all Americans by the National Committee for Quality Assurance.

## 13.6 Concluding Remarks

Low energy availability, as a consequence of disordered eating or eating disorders in female athletes, is a significant health concern. It is a key component of the female athlete triad and can lead to menstrual disorders and changes in bone health. The athletic health care community needs to address this health concern beginning in junior high school and high school and continuing through the lifetime of the active female. The best method of management is through a combined approach with screening both informally through observation and formally during preparticipation examinations and other interactions of female athletes with health care providers, in order to prevent the consequences of disordered eating and eating disorders. Eating disorders can lead to significant health complications including death.

For formal screening, the best tool is one that is confidential, inexpensive, readily accessible, and validated in a female athlete population. A promising step forward will be a nationally agreed upon standardized self-report supplemental questionnaire specific for female health concerns related to low energy availability.

## 13.7 Questions

1. The American College of Sports Medicine Position Stand on the Female Athlete Triad describes the interrelationships between
   (a) Energy availability
   (b) Menstrual function
   (c) Bone mineral density
   (d) All of the above
2. Energy availability is the energy remaining for body functions after that used for exercise is added to total energy intake.
   (a) True
   (b) False
3. One way an athlete's available energy may be reduced is
   (a) Decreased energy expenditure with reduced exercise
   (b) Increasing energy intake
   (c) Abnormal eating behaviors
   (d) None of the above
4. Screening for disordered eating and eating disorders can occur at a common entry point to athletic participation.
   (a) True
   (b) False
5. Once an athlete screens positively for possible low energy availability or if a concern exists, the athlete should
   (a) Celebrate his/her positive screening
   (b) Continue his/her normal training routine

(c) Increase energy expenditure while decreasing energy intake
(d) Be referred for further medical and psychological or psychiatric evaluation.

6. What makes a screening tool useful is
   (a) Functionality
   (b) Validity
   (c) Reliability
   (d) All of the above
   (e) None of the above
7. A more formal screening evaluation for disordered eating/eating disorders should include:
   (a) Detailed medical, nutritional, and reproductive history
   (b) Physical examination with lab evaluation
   (c) Referral to a psychologist
   (d) All of the above
   (e) None of the above
8. The informal settings where athletes may be screened for disordered eating/eating disorders occur:
   (a) While interacting with personal trainers, family, and friends
   (b) When filling out PPEs
   (c) In clinical settings
   (d) None of the above
9. Physical complaints that can help diagnose an eating disorder include:
   (a) Amenorrhea
   (b) Bradycardia
   (c) Skin changes
   (d) Low body mass index
   (e) All of the above
10. The clinical tool that is recognized as the assessment of choice for diagnosing an eating disorder is:
    (a) Eating Disorders Interview (EDI)
    (b) EAT-26
    (c) Eating Disorders Exam (EDE)
    (d) Female Athlete Screening Tool (FAST)
    (e) SCOFF

## 13.8 Answer Key

1. D
2. B
3. C
4. A
5. D
6. D
7. D
8. A
9. E
10. C

## References


1. Nattiv A, Loucks AB, Manore MM, et al. The female athlete triad. Med Sci Sports Exerc. 2007;39(10): 1867–82.
2. Beals KA, Hill AK. The prevalence of disordered eating, menstrual dysfunction, and low bone mineral density among US collegiate athletes. Int J Sport Nutr Exerc Metab. 2006;16(1):1–23.
3. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed. Washington, DC: American Psychiatric Association; 2000. Text Revision.
4. Byrne S, Mc LN. Eating disorders in athletes: a review of the literature. J Sci Med Sport. 2001;4(2):145–59.
5. Gibbs JC, Williams NI, De Souza MJ. Prevalence of individual and combined components of the female athlete triad. Med Sci Sports Exerc. 2013;45(5):985–96.
6. Bonci CM, Bonci LJ, Granger LR, et al. National athletic trainers' association position statement: preventing, detecting, and managing disordered eating in athletes. J Athl Train. 2008;43(1):80–108.
7. Torstveit MK, Rosenvinge JH, Sundgot-Borgen J. Prevalence of eating disorders and the predictive power of risk models in female elite athletes: a controlled study. Scand J Med Sci Sports. 2008;18:108–18.
8. Black DR, Larkin LJS, Coster DA, et al. Physiologic screening test for eating disorders/disordered eating among female collegiate athletes. J Athl Train. 2003; 38(4):286–97.
9. Garner D. This week's citation classic; self-report measures for eating disorders. Curr Contents. 1993;8.
10. Garner D. Eating attitudes test (EAT-26) self-test, scoring and interpretation. Eating attitudes test (EAT-26) website. http://www.eat-26.com/. Accessed 4 May 2013.
11. Beals KA. Disordered eating among athletes: a comprehensive guide for health professionals. Champaign, IL: Human Kinetics; 2004. p. 117–30.
12. Mountjoy M, Hutchinson M, Cruz L, et al. Female athlete triad coalition position stand on female athlete triad pre participation evaluation. 2008. Available at: http://www.femaleathletetriad.org. Accessed 5 May 2013.
13. Mencias T, Noon M, Hoch AZ. Female athlete triad screening in national collegiate athletic association division 1 athletes: is the preparticipation evaluation form effective? Clin J Sport Med. 2012;0(0): 1–4.

14. Beals KA. Eating disorder and menstrual dysfunction screening, education, and treatment programs. Phys Sportsmed. 2003;31(7):33–8.
15. Rumball JS, LeBrun CM. Preparticipation physical examination selected issues for the female athlete. Clin J Sport Med. 2004;14(3):153–60.
16. Anderson DA, Lundgren JD, Shapiro JR, Paulosky CA. Assessment of eating disorders review and recommendations for clinical use. Behav Modif. 2004;28(6):763–82.
17. Lebrun CM. The female athlete triad: what's a doctor to do? Curr Sports Med Rep. 2007;6:397–404.
18. Fairburn CG, Beglin SJ. Assessment of eating disorders: interview or self-report questionnaire? Int J Eat Disord. 1994;16(4):363–70.
19. Kakaiya D. Eating disorders among athletes. IDEA Fitness Journal. March 2008;5(3):12 pages. http://www.ideafit.com/fitness-library/eating-disorders-among-athletes. Accessed 4 May 2013.
20. Vaughan JL, King KA, Cottrell RR. Collegiate athletic trainers' confidence in helping female athletes with eating disorders. J Athl Train. 2004;39(1):71–6.
21. Sherman R, Thompson R. Athlete special interest group of the academy of eating disorders. NCAA coaches handbook, managing the female athlete triad. National collegiate athletic association.
22. Beals KA, Meyer NL. Female athlete triad update. Clin Sports Med. 2007;26:69–89.
23. http:/www.stanford.edu/dept/sportsmed/visitors/visitors98.html. Accessed 4 May 2013.
24. National Committee for Quality Assurance website. Patient centered medical home. http://www.ncqa.org/Programs/Recognition/PatientCenteredMedicalHomePCMH.aspx. Accessed 4 May 2013.
25. Pritts S, Susman J. Diagnosis of eating disorders in family care. Am Fam Physician. 2003;67(2):297–304.
26. Morgan JF, Reid F, Lacey JH. The SCOFF questionnaire: assessment of a new screening tool for eating disorders. BMJ. 1999;319:1467–8.
27. Mond JM, Myers TC, Crosby RD, et al. Screening for eating disorders in primary care: EDE-Q versus SCOFF. Behav Res Ther. 2008;46:612–22.
28. Berg KC, Peterson CB, Frazier P, Crow SJ. Psychometric evaluation of the eating disorder examination and eating disorder examination-questionnaire: a systematic review of the literature. Int J Eat Disord. 2012;45(3):428–38.
29. Fairburn CG, Beglin S. Appendix II, eating disorder examination questionnaire (EDE-Q 6.0). In: Fairburn CG, editor. Cognitive behavior therapy and eating disorders. New York, NY: Guilford Press; 2008.
30. Mond JM, Hay PJ, Rodgers B, Owen C. Eating disorder examination questionnaire (EDE-Q): norms for young adult women. Behav Res Ther. 2006;44:53–62.
31. Garner D, Olmstead MP, Polivy J. Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. Int J Eat Disord. 1983;2(2):15–34.
32. Clausen L, Rosenvinge JH, Friborg O, et al. Validating the eating disorder inventory-3 (EDI-3): a comparison between 561 female eating disorder patients and 878 females from the general population. J Psychopathol Behav Assess. 2011;33(1):101–10.
33. Cumella EJ. Review of the eating disorder inventory-3. J Pers Assess. 2006;87(1):116–7.
34. Cotton M, Ball C, Robinson P. Four simple questions can help screen for eating disorders. J Gen Intern Med. 2003;18:53–6.
35. Thelan MH, Farmer J, Wonderlich S, Smith M. A revision of the bulimia test: the BULIT-R. Psychol Assessment. 1991;3:119–24.
36. National Eating Disorders Association (NEDA) Screening program. http://www.nationaleatingdisorders.org/. Accessed 30 May 2013.
37. McNulty KY, Adams CH, Anderson JM, Affenito SG. Development and validation of a screening tool to identify eating disorders in female athletes. J Am Diet Assoc. 2001;101(8):886–92.
38. Nagel DL, Black DR, Leverenz LJ, Coster DC. Evaluation of a screening test for female college athletes with eating disorders and disordered eating. J Athl Train. 2000;35(4):431–40.
39. Hinton PS, Kubas KL. Psychosocial correlates of disordered eating in female collegiate athletes: validation of the ATHLETE questionnaire. J Am Coll Health. 2005;54(3):149–56.
40. Steiner H, Pyle RP, Brassington GS. The college health related information survey (CHRIS-73): a screen for college student athletes. Child Psychiatry Hum Dev. 2003;34(2):97–109.
41. DePalma MT, Koszewski WM, Romani W, et al. Identifying college athletes at risk for pathogenic eating. Br J Sports Med. 2002;36:45–50.
42. Bernhardt DT, Roberts WO, editors. PPE preparticipation physical evaluation. 4th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2010. p. 120–30. 154–156.
43. Ljungqvist A, Jenoure P, Engebretsen L, et al. The international olympic committee (IOC) consensus statement on periodic health evaluation of elite athletes. J Athl Train. 2009;44(5):538–57.

# Evidence-Based Disordered Eating Prevention Programs for Active Females

14

Jacalyn J. Robert-McComb and Anna M. Tacón

**Abstract**

In this chapter, the authors discuss the role that self-concept plays as a modulator in disordered eating. While there are many models for self-concept, all models recognize that the development of positive self-esteem is multidimensional and an individual's perception of self can be affected by the environment in both positive and negative ways. Effective prevention and intervention programs must recognize the importance of enhanced self-esteem and embrace the development of this concept in their programs for positive health behavior change. Numerous theoretical frameworks have been proposed to explain and predict the process of health behavior change. The Transtheoretical Model (TTM) developed by Prochaska and DiClemente as a model of intentional behavior change is highlighted in this chapter. Targeted educational programs to prevent disordered eating for female athletes are presented and contact information for more details for research based effective programs are provided in a summary format.

**Keywords**

Prevention programs for eating disorders • Transtheoretical model • Enhanced self-esteem

## 14.1 Learning Objectives

After completing this chapter, you should have an understanding of:

- The importance of self-esteem in disordered eating
- The transtheoretical model in the process of health behavior change
- General treatment principles and medical criteria for hospitalization
- Program examples
- Eating disorder resources

J.J. Robert-McComb, Ph.D., FACSM, ACSM P.D. (✉)
A.M. Tacón, Ph.D.
Department of Health, Exercise and Sport Sciences, Texas Tech University, Lubbock, TX, USA
e-mail: jacalyn.mccomb@ttu.edu; anna.tacon@tu.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_14, 

## 14.2 Introduction

Low self-esteem, accompanied with perfectionism, is a well-recognized trait of those with disordered eating and could be a precipitating factor in the development of eating disorders [1, 2]. This characteristic likely increases females' vulnerability to disordered eating when combined with recently documented genetic and neurobiological findings: the heritability of eating disorder symptoms increases from zero risk before puberty to ≥50 % during and after puberty; also, fMRI studies show decreased activity in brain areas involved in self-regulation and impulse control [3, 4]. Self-esteem, as a component of self-regulation, frequently triggers behavioral and cognitive strategies to maintain or enhance sense of sense [5]. The terms self-concept, self-worth, and self-image are used interchangeably with self-esteem and are all based on self-perception. Self-esteem can defined as the extent to which a person feels positive about himself or herself [6]. Often, self-esteem is described as dichotomous, with a person possessing either high or low self-esteem. People with low self-esteem feel positive about themselves when they encounter affirmative experiences. Conversely, when they face negative experiences, they are disapproving of themselves. People with high self-esteem embrace and benefit from positive experiences and have developed strategies to mollify negative feedback. In short, they have learned how to *offset* negative experiences [7, 8].

Effective prevention and intervention programs must recognize the importance of enhanced self-esteem and embrace the development of this concept in their programs. Education to prevent disordered eating can only be effective if the individual understands and accepts herself, even her limitations. Many times, physically active women have a heightened awareness of the body and its limitations [8]. These limitations may contribute to low self-esteem (losing a race, finishing last, etc.). Rosenberg [9] describes self-esteem as consisting of three major components: (a) social identities (how an individual defines him or herself in society), (b) personal dispositions (perceptions of traits, preferences, response tendencies), and (c) physical characteristics (height, weight, body fat distribution, attractiveness, etc.). While there are many models for self-concept, all models recognize that the development of positive self-esteem is multidimensional and an individual's perception of self can be affected by the social, emotional, and physical involvement in sport and exercise.

## 14.3 Research Findings

### 14.3.1 The Transtheoretical Model in Health Behavior Change

Numerous theoretical frameworks have been proposed to explain and predict the process of health behavior change. One frequently used is the transtheoretical model (TTM) developed by Prochaska and DiClemente as a model of intentional behavior change [10]. The TTM assumes that individuals vary in motivation and readiness to change their behavior, and as well, realistically acknowledges that relapse is normal under situations that involve such significant behavior change. Four related concepts considered central to health behavior change are included within this model: stage of change, self-efficacy, decisional balance, and processes of change.

The five stages of change are precontemplation (PC, no intention to change health behaviors within the next 6 months), contemplation (C, seriously considering behavioral change within the next 6 months), preparation (P, still lack commitment to change, but investigating the possibility of change within the next 30 days), action (A, actively modifying problematic behavior within the last 6 months), and maintenance (M, self-control of the behavior established more than 6 months ago).

In the PC stage, information needs to be provided about the behavioral and potential medical problems associated with the behavior. In the C stage, health professionals must help individuals assess the pros and cons of the behavior change so that they will make a commitment to change. Health professionals must encourage initial small steps to initiate change, no one can force someone

to change, and individuals must begin to place greater significance on the benefits of behavior change in the P stage. The A stage occurs when benefits outweigh the costs. Initiating a new health behavior change is bound to be fraught with relapse; health professionals should act to reinforce an individual's self-confidence along with their decision to change. The M stage occurs when individuals are able to continue the new behavior. Conceptually, progression through these stages during attempts at behavioral change is expected to be linked to differences in self-efficacy, decisional balance, and the processes of change [11].

Self-efficacy and decisional balance represent beliefs about behavior that are common to many social cognition models. Specifically, self-efficacy refers to an individual's confidence in his or her ability to perform a specific behavior, which is expected to increase as an individual moves through the stages [12]. Indeed, self-efficacy for health behavior change reliably predicted stages of change; precontemplators and contemplators had the lowest efficacy, while those in the maintenance stage exhibited the highest efficacy [13].

Decisional balance relates to the pros (benefits) and cons (costs) of the behavior; for example, eating gives me more energy, yet it might make me fat. Individuals who change their behaviors have positive decisional balance because the positive beliefs about the behavior outweigh the negative ones. Additionally, the pros increase, while the cons decrease across the stages of change.

Lastly, the TTM also includes processes of change and specifically define a process of change as a "type of activity that is initiated or experienced by an individual in modifying affect, behavior, cognition or relationships" [10]. Health professionals can assist the process of change and the maintenance of the new behavior by providing follow-up support [14].

### 14.3.2 Athletes at Risk Program

Targeted educational programs for female athletes can be effective at any stage of change. The Athletes@Risk® program is a preventative program for female athletes in both recreational and competitive sport who are at risk of developing the female athlete triad that involves disordered eating, amenorrhea, and osteoporosis (Table 14.1). This licensed prevention educational program was developed by Dr. Julia Alleyne, a medical director for Sport CARE and a clinical professor of medicine at the University of Toronto.

There are five interactive workshop units of this program: understanding health consequences (of the athlete triad), healthy eating habits (food as fuel), positive self-esteem and body image (my body, my sport), safe training practices (getting strong, getting fit), and stress management (life skills and wellness) [15]. The program has been designed for health professionals who interact with female athletes who are at risk for developing the female athlete triad, who may be prone to anxiety disorders, and who have frequent muscular injuries, low self-esteem, and problems coping with lifestyle stressors. This program also can be used as a summer camp for children at a sponsoring facility. For further information, contact Women's College Hospital, Toronto, ON, M5S 1B2, toll free at 1-800-363-9353or visit the website (http://www.womenscollegehospital.ca/programs-and-services/fitness-and-exercise/athletesrisk-program423).

### 14.3.3 General Treatment Principles

Treating eating disorders is both a science and an art [16]. Even though the treatment goals for anorexia nervosa and bulimia nervosa are well defined (Table 14.2), the method of achieving these goals is less certain [17]. Also, treatment is less likely to occur without proper screening in terms of medical criteria used for hospitalization (Table 14.3) [18–20].

Achieving and maintaining a normal weight and nutritional status is essential for recovery for both anorexia nervosa (AN) and bulimia nervosa (BN). Perfectionist attitudes, low self-esteem, unrelenting pursuit of thinness, intolerance of mood fluctuations, and poor coping skills are problematic for both AN and BN. Recovery is unlikely without a fundamental change in these attitudes.

**Table 14.1** Overview of the components in the Athletes@Risk® program at the University of Toronto

| | |
|---|---|
| *Session #1* | Injury prevention and treatment principles |
| Understanding the health consequences | Female-specific issues in physical activity |
| The female athlete triad | *Interactive tools* |
| Prevention, recognition, and treatment | Strength training on the go |
| The continuum between disordered eating and an eating disorder | Heart smart learning |
| Prevention and physical consequences | Performance aids challenge |
| Exploring osteoporosis in the younger woman | Injury management game |
| Identification and decreasing the risk | Female-specific issues crossword |
| Menstruation and the female athlete | *Session #4: food as fuel* |
| *Interactive tools* | Nutrition: the basics |
| Are you at risk? | Using the food guide for good nutrition |
| Commonly asked questions | Getting enough fuel for activities |
| Word search | How much is enough? |
| *Session #2: my body, my sport* | What happens if needs are not met? |
| Body image and self-esteem | Eating for performance: before, during, and after |
| The genetics of body shape | Why do we eat? |
| Body image and shape through history and culture | *Interactive tools* |
| The truth about body composition testing | Healthy choices at fast food restaurants |
| Body image and injury: dealing with the scars | Food and water log |
| Healthy self-esteem strategies for teens | Analyze a food label |
| *Interactive tools* | Nutrition jeopardy |
| Body contour rating | Olympic quiz |
| Determining body esteem | *Session #5: life skills and wellness* |
| Body image diary | Stress management |
| Developing self-talk: being positive and realistic | Coping with stress |
| Athletes response to injury | Perception and control |
| Simple relaxation techniques | Self-esteem and stress management |
| *Session #3: getting strong, getting fit* | Developing healthy sexuality |
| Stretch and strength: keeping balanced | Harassment and abuse |
| How do we get stronger | Boundary setting |
| Women and strength training | Physical, social, and sexual boundaries |
| The seasons of strength training | Assertiveness training |
| Additional benefits of getting stronger | Interactive tools |
| Getting fit | Are you stressed? |
| Aerobic, anaerobic, and interval training | Simple relaxation techniques |
| Performance aids: what is safe and what is not? | Are you non-assertive? |
| Overtraining: signs and symptoms | Developing assertive behavior |

Athletes@Risk® is a licensed preventive education program

Research has shown that the therapist should function in the therapeutic encounter as a parent, teacher, guide, and coach and that the personality of the therapist is a major therapeutic element in the treatment of patients with AN [17]. The therapist must make every effort to engage the family, especially if treating patients with AN under 18 years of age [21]. Family therapy has shown to be very effective in younger patients with AN of shorter duration (less than 2 years).

Although most patients with BN can be treated in an outpatient setting, the first issue a clinician must decide with AN is the treatment setting. Most clinicians will recommend inpatient treatment for a patient who weighs less than 75 % of average weight, has severe metabolic disturbances, if feeling suicidal, or has failed to

**Table 14.2** Treatment goals for anorexia nervosa and bulimia nervosa

| Anorexia nervosa | Bulimia nervosa |
|---|---|
| Restore weight and improve eating habits | Identify the factors and processes that maintain the binge-purge-starve cycle |
| Change dysfunctional attitudes | |
| Treat concomitant medical complications | Help the individual identify strategies to overcome the disturbed eating pattern |
| Work with the family | Change dysfunctional thoughts |
| Pharmacotherapy to treat depression, moderate obsessive perfectionism, to target certain neurotransmitters, or to treat concomitant complications | Build coping skills |
| Prevent relapse (most difficult task) | Note: Cognitive behavioral therapy (CBT) has been found to be most effective. However, self-help (SH), using a written manual based on the principles of CBT, has gained wide appeal since the patient may use it with or without the guidance of a therapist |

improve after a period of outpatient or partial program treatment [20]. Again, refer to Table 14.3 as to the medical criteria for hospitalization provided by multiple sources [18–20].

Traditionally, inpatient treatment is continued until a patient reaches a reasonable healthy body weight. Discussing a target weight is one of the most important initial tasks of weight restoration. Currently, most clinicians use a body mass index of 18.5 kg/m$^2$ as the minimal healthy weight for a patient older than 16 [17].

The treatment of BN is usually conducted in an outpatient setting. Cognitive behavioral therapy (CBT) is the treatment of choice and is effective whether conducted individually [22, 23] or in a group setting [24]. The most widely used CBT is the version developed by Fairburn and is implemented over a period of 18 weeks [25]. In the first stage, behavioral techniques are used to replace binge eating with a stable pattern of regular eating. In the second stage, the goal is to eliminate dieting; the focus is on the thoughts, beliefs, and values that reinforce dieting. The third stage is focused on the maintenance of these new healthy behaviors and thought patterns.

Self-help (SH), using a written manual based on the principles of CBT, has been shown to be effective for BN patients and is more accessible than CBT [26]. The patient may use it without any guidance or with the help of a therapist. Under guidance, the program usually consists of 7 sessions and is conducted over a period of 12 weeks. The most widely used manual, by Fairburn, provides a step-by-step discussion of implementing the program [27].

### 14.3.4 Dissonance-Based Eating Disorder Prevention Program

Almost 10 % of teenage girls and young women experience threshold or subthreshold anorexia nervosa, bulimia nervosa, or binge eating disorders [28, 29]. A meta-analytic review [30] found that only five from dozens of randomized controlled efficacy trials investigating disordered eating prevention programs produced significant reductions in disordered eating symptoms among intervention participants compared to controls that extended at least 6-month post-intervention [31, 32]. One program, cognitive dissonance-based prevention or DBP, which utilizes the theory of cognitive dissonance, is accumulating data as to its empirical effectiveness with EDs [33].

Dissonance theory posits that having inconsistent cognitions creates psychological discomfort which motivates people to change their cognitions for the sake of consistency.

Dissonance may be activated by having individuals act in ways that are not consistent with their beliefs; dissonance can then be reduced by altering the behavior or belief. Recent research indicates that DBP interventions can help athletes with disordered eating by reducing negative thoughts and feelings [34–38].

Smith and Petrie [35] tested the effectiveness of a cognitive dissonance-based intervention compared to a psychoeducationally based healthy

**Table 14.3** Sample hospitalization criteria

| | Adults | Children and adolescents |
|---|---|---|
| Heart rate | <40 bpm or >110 bpm | <50 bpm daytime; <45 bpm nighttime arrhythmia |
| Blood pressure | <90/60 mmHg or orthostatic hypotension (pulse increase of >20 bpm or drop in BP of >10–20 mmHg/min from lying to standing) | <80/50 mmHg; orthostatic blood pressure changes (>20 bpm increase in heart rate or >10–20 mmHg drop in blood pressure) |
| Glucose | <60 mg/dL | <60 mg/dL |
| Electrolytes or metabolic function | Potassium<3 meq/L; electrolyte imbalance; dehydration; metabolic abnormalities | Hypo- or hypernatremia, hypophosphatemia, hypokalemia, hypomagnesemia; serum chloride concentration <88 |
| Gastrointestinal | Hematemesis; esophageal tears; intractable vomiting | Hematemesis; esophageal tears; intractable vomiting |
| Temperature | <97.0 °F; inability to sustain body core temperature | <96.0 °F, dehydration |
| Hepatic, renal, or cardiovascular | Organ comprise requiring acute treatment | Organ compromise requiring acute treatment; arrhythmias |
| Weight and body fat | <75 % of healthy body weight or acute weight decline with food refusal | Acute weight decline with food refusal even if not <75 % of healthy body weight; body fat <10 % |
| Motivation to recover | Very poor to poor; preoccupied with egosyntonic thoughts | Very poor to poor; preoccupied with egosyntonic thoughts; failure to respond to outpatient treatment |
| Comorbid psychiatric Disorders | Any existing disorder that would require hospitalization such as suicidal risk and depression | Any existing disorder that would require hospitalization such as suicide risk and depression |
| Purging behavior (laxatives and diuretics) | Needs supervision during and after all meals and in bathrooms | Needs supervision during and after all meals and in bathrooms |
| Ability to care for self; ability to control exercise | Complete role impairment; structure required to keep patient from compulsive exercising | Complete role impairment; structure required to keep patient from compulsive exercising |
| Stress and support | Severe family conflict, lack of structured treatment in home; inadequate support | Severe family conflict, lack of structured treatment in home; inadequate support |

Multiple sources: adapted from American Psychiatric Association. Practice Guidelines for the Treatment of Patients with Eating Disorders, 2nd edition. Washington, DC: American Psychiatric Press; 2000;57:5–56; Halmi, K. (2009). Salient components of a comprehensive service for eating disorders. *World Psychiatry*, *8*, 150–155; Rosen, D. and the Committee on Adolescence. (2010). Identification and management of eating disorders in children and adolescents. *Pediatrics*, *126*: 1240–1254

weight and a wait-list control to determine their relative effectiveness in reducing body dissatisfaction, negative affect, dietary restriction, and internalization of the sociocultural ideal. The sample of 29 self-identified disordered eating female athletes engaged in exercises that questioned the thin ideal body type so as to produce dissonance. Possibly due to low power, no treatment effects were found; however, exploratory post hoc analyses suggested that the cognitive dissonance intervention provided some positive effects, that is, decreases in sadness/depression, internalization of a physically fit body type, and increases in body satisfaction.

Another study focused on the issue of education in the female athlete triad. Specifically, Becker and colleagues evaluated whether two exploratory peer-led interventions could have a positive effect on athletes at risk for an eating disorder [34]. Athletes were randomly assigned to either an athlete-modified dissonance prevention or a healthy weight intervention (AM-HWI); ED risk factors were assessed pre/posttreatment, at 6-week and 1-year follow-up. The results ($N=157$) indicated that both interventions reduced dietary restraint, thin ideal internalization, bulimic pathology, shape/weight concern, and negative affect at 6 weeks. Bulimic pathology, shape concern, and negative affect were

reduced at 1-year follow-up. Also, qualitative results suggested that AM-HWI may be the more preferred intervention by athletes.

### 14.3.5 Eating Disorder Organizations and Resources

Resources exist for physicians in treating eating disorders (Table 14.4). Moreover, education programs at exercise facilities can help to prevent triad disorders in girls and young women by alerting them and caring individuals such as family members. Education materials can be displayed on bulletin boards, web pages, newsletters etc. Information packets can educate parents and children, even if the facility is restricted to adults. An information packet about eating disorders is the BodyWise Handbook at http://www.maine.gov/education/sh/eatingdisorders/bodywise.pdf. The Female Athlete Triad Coalition web page (http://www.femaleathletetriad.org/) contains additional helpful information that exercise professionals can use as educational tools.

Workshops related to improving body image, healthy eating habits, and coping with stressors during puberty can also be provided by fitness facilities. These workshops do not have to be put on by employees but can be hosted for the facility's own clients, as a community service and as advertising for the sponsoring facility. A professional workshop for increasing the self-esteem of girls is GirlPower (https://www.urstrong.com/). There are licensed GirlPower facilitators in Canada, the United States, and Australia. Table 14.5 lists additional educational resources for the prevention and treatment of eating disorders and other related mental illnesses. For more information about the National Mental Health Association or additional resources, please call 1-800-969 NMHA (6642) or visit their website at http://www.nmha.org/infoctr/index.cfm.

**Table 14.4** Resources for physicians for the treatment of eating disorders

| |
|---|
| *The Academy for Eating Disorders (AED)* |
| 3728 Old McLean Village Dr |
| McLean VA 22101 |
| Phone: (847) 498-4274 |
| Fax: (847) 480-9282 |
| Website: http://www.aedweb.org |
| E-mail: info@aedweb.org |
| Under the eating disorders information, click on research-practice guidelines. This will bring up the latest in research and practice initiatives |
| *American Psychiatric Association (APA)* |
| 1400 K St, NW |
| Washington, DC 20005 |
| Phone: 1-888-35-PSYCH (toll free) |
| Website: http://www.psych.org |
| E-mail: apa@psych.org |
| There is a section entitled Mental Health. Under this tab, there is a list of key topics, one of which is eating disorders. A discussion of the disorders as well as treatment is listed |
| *Internet Mental Health (IMH)* |
| 601 W Broadway Suite 902 |
| Vancouver, BC |
| Canada, V5Z4C2 |
| Phone: (604) 876-2254 |
| Fax: (604) 876-4929 |
| Website: http://www.mentalhealth.com |
| This site features descriptions of eating disorders by going to the Index and selecting Eating Disorders. Also, under the area of Content, website links are provided |

(continued)

**Table 14.4** (continued)

| |
|---|
| *National Association of Anorexia and Associated Disorders (ANAD)* |
| National Association of Anorexia Nervosa and Associated Disorders, Inc.®<br>750 E Diehl Road #127<br>Naperville, IL 60563<br>Helpline: (630) 577-1330<br>Website: http://www.anad.org<br>E-mail: anadhelp@anad.org<br>This website has abundant resources regarding disorders information, treatment and support with a helpline, and listings of treatment facility partners associated with this organization. The site also contains an area dedicated to legislative news relevant to mental health and eating disorders. A list of referrals and support groups is available by calling the helpline |
| *National Eating Disorders Association (NEDA)* |
| 165 West 46th Street<br>New York, NY 10036<br>Phone number:(212) 575-6200 or<br>1-800-931-2237 (toll-free information and referral hotline)<br>Fax:(212) 575-1650<br>Web: http://www.edap.org<br>E-mail: info@NationalEatingDisorders.org<br>This comprehensive website offers multiple resources and guidance that begins with online eating disorder screening, an information and referral helpline, as well as a requested listing of practitioners and facilities in one's local area and insurance resources. Support groups and research studies as well as parent, family, and friends' networks are also available |
| *Something Fishy* |
| Phone number: (866) 690-7239<br>Website: http://www.something-fishy.org<br>News, descriptions of eating disorders, and a treatment finder are all offered on this extensive site. Online support through chat rooms, message boards, and American Online Instant Messaging (AIM) is also available. Sections on dangers associated with eating disorders, helping loved ones, recovery, and cultural issues provide useful information not always seen on other websites. "Doctors and Patients" is a section that provides a practical discussion of medications as well as blood and lab tests. An area entitled "Tips for Doctors" dispenses functional advice about what patients fear most. Their concerns, for example, include not being taken seriously by a physician or that the physician will notify their parents. The additional resources area lists organizations, other websites, and hotline numbers; recommends written material; and provides links to research |

Source: adapted from Patient Care. Identifying and managing eating disorders. November 30, 2001 (http://www.patientcareonline.com/patcare/). With permission

**Table 14.5** Eating disorder organizations and resources

| |
|---|
| *Anorexia Nervosa and Related Eating Disorders, Inc. (ANRED)*<br>*Internet*: http://www.anred.com/<br>ANRED's mission is to provide easily accessible information on anorexia nervosa, bulimia nervosa, binge eating, and other food and weight disorders. ANRED, a nonprofit organization, distributes materials on topics about recovery and prevention of weight related disorders |
| *Soy Unica! Soy Latina!*<br>*Internet*: http://latinasunidas.org/mybody/default.htm<br>An excellent bilingual website for young Latinas with a good section on eating disorders |
| *Eating Disorder Information and Referral Center*<br>*Internet*: http://www.EDreferral.com<br>This website is a resource for information and treatment options for all forms of eating disorders. It includes referrals to local treatment centers nationwide |

(continued)

**Table 14.5** (continued)

| |
|---|
| *Harvard Eating Disorders Center (HEDC)*<br>WACC 725<br>15 Parkman Street<br>Boston, MA 02114<br>Tel: (617) 236-7766<br>*E-mail*: info@hedc.org<br>*Internet*: http://www.hedc.org/<br>The Harvard Eating Disorders Center is a national nonprofit organization dedicated to research and education and gaining new knowledge of eating disorders, their detection, treatment, and prevention to share with the community at large. The website includes information about eating disorders, help for family and friends, resources, and a listing of events and programs |
| *Overeaters Anonymous (OA)*<br>World Service Office<br>PO Box 44020<br>Rio Rancho, NM 87174-4020<br>Tel: (505) 891-2664<br>*E-mail*:info@overeatersanonymous.org<br>*Internet*: http://www.overeatersanonymous.org/OA is a nonprofit international organization that provides volunteer support groups worldwide. Modeled after the 12-step Alcoholics Anonymous program, the OA recovery program addresses physical, emotional, and spiritual recovery aspects of compulsive overeating. Members are encouraged to seek professional help for individual diet and nutrition plans and for any emotional or physical problems |
| *The Renfrew Center Foundation*<br>475 Spring Lane<br>Philadelphia, PA 19128<br>Tel: 1-800-RENFREW<br>*E-mail*: foundation@renfrew.org<br>*Internet*: http://www.renfrew.org/<br>The Renfrew Center Foundation is a tax-exempt, nonprofit organization promoting the education, prevention, treatment, and research of eating disorders. The Renfrew Center Foundation is funded by private donations and by the Renfrew Center, the nation's first freestanding facility committed to the treatment of eating disorders |

## 14.4 Contemporary Understanding of the Issues

Theoretical perspectives differ in the treatment of eating disorders, and the interplay among the many treatment variables is very complex and not well understood. Furthermore, many ED specialists in the sport world consider female athletes to represent a uniquely challenging population with which to work [34]. CBT, the Athletes@Risk®, and dissonance-based programs are three intervention strategies aimed at providing further understanding of treatments for ED. The later DBP has been supported to the extent that it meets the American Psychological Association's (APA) criteria for an efficacious intervention (i.e., DBP outperformed no treatment control groups, an alternative intervention, and findings have been replicated by independent laboratories/researchers), which is rare for ED prevention programs ([34], p. 4). Additionally, DPB effects appear to be long lasting with data showing reductions in ED risk factors continuing at 2- and 3-year follow-up periods [36]; DBP has been shown to reduce the onset of EDs by 60 % compared to an assessment only control [34].

## 14.5 Future Directions

Practice guidelines for the treatment of patients with eating disorders have been developed by psychiatrists who are in active clinical practice and are available on the web at http://www.psych.org/psych_pract/treatg/pg/eating_revisebook_index.cfm?pf=y. These guidelines were approved by the American Psychiatric Association in 1999 and published in 2000 [22]. These guidelines are not intended to serve as a standard of medical care but rather provide recommendations for treating patients with eating disorders.

## 14.6 Concluding Remarks

Theoretical perspectives differ in the treatment of eating disorders, and the interplay among treatment variables is very complex. Despite the fact that treatment goals for anorexia nervosa and bulimia nervosa are well defined, a decisive therapeutic approach with which to achieve these goals remains unclear. Regardless of theoretical perspectives, however, the personality of the health professional and the therapeutic relationship developed between the health professional and patient are important elements in recovery [17].

## References


1. Silverstone PH. Is chronic low self-esteem the cause of eating disorders? Med Hypotheses. 1992;39:311–5.
2. Thompson RA, Sherman RT. Helping athletes with eating disorders. Champaign, IL: Human Kinetics; 1993.
3. Klump KL, Perkins PS, Alexandra BS, McGue M, Iacono WG. Puberty moderates genetic influences on disordered eating. Psychol Med. 2007;37:627–34.
4. Klump KL, Keel PK, Sisk C, Burt SA. Preliminary evidence that estradiol moderates genetic influences on disordered eating attitudes and behaviors during puberty. Psychol Med. 2010;40:1745–53.
5. Rhodewalt F, Tragakis MW. Self-esteem and self-regulation: toward optimal studies of self-esteem. Psychol Inq. 2003;14:66.
6. Sonstroem RJ. Exercise and self-esteem. In: Terjung RL, editor. Exerc Sport Sci Rev, vol. 12. Lexington, MA: The Collamore Press; 1984. pp. 123–155.
7. Brown JD, Mankowski TA. Self-esteem, mood, and self-evaluations: changes in mood and the way you see you. J Pers Soc Psychol. 1993;64(3):421–30.
8. Lindeman A. Self-esteem: its application to eating disorders and athletes. Int J Sport Nutr. 1994;4:237–52.
9. Rosenberg M. Society and the adolescent self-image. Princeton, NJ: Princeton University Press; 1965.
10. Prochaska J, DiClemente C. Toward a comprehensive model of change. In: Miller WE, Heather N, editors. Treating addictive behaviors. London: Plenum Press; 1998. p. 3–27.
11. Prochaska J, Marcus B. The transtheoretical model: applications to exercise. In: Dishman RK, editor. Advances in exercise adherence. Champaign, IL: Human Kinetics; 1994. p. 161–80.
12. Marcus B, Selby V, Niaura R, et al. Self-efficacy and the stages of exercise behavior change. Res Q Exerc Sport. 1992;63:63–6.
13. Marcus B, Owen N. Motivational readiness; self-efficacy and decision making for exercise. J Appl Soc Psychol. 1992;22:3–16.
14. Bass M, Turner L, Hunt S. Counseling female athletes: application of the stages of change model to avoid disordered eating, amenorrhea, and osteoporosis. Psychol Rep. 2001;88:1153–60.
15. Rocci N. Athletes at risk: preventive education for active women. Nat Eat Disorder Info Cent. 2002. http://www.nedic.ca/knowthefacts/documents/athletesatrisk.pdf.
16. Lucas AR. Demystifying anorexia nervosa. New York, NY: Oxford University Press; 2004.
17. Hsu LK. Eating disorders: practical interventions. Womens Health. 2004;59:113–24.
18. Halmi K. Salient components of a comprehensive service for eating disorders. World Psychiatry. 2009;8:150–5.
19. Rosen D, The Committee on Adolescence. Identification and management of eating disorders in children and adolescents. Pediatrics. 2010;126:1240–54.
20. American Psychiatric Association. Practice guidelines for eating disorders revision. Am J Psychiatry. 2000;157(1 Suppl):1–39.
21. Lock J. Treating adolescents with eating disorders in the family context. Child Adolesc Psychiatr Clin N Am. 2002;11:331–42.
22. American Psychiatric Association. Practice guidelines for the treatment of patients with eating disorders. 2nd edn. Washington, DC: American Psychiatric Press; 2000. vol. 57. pp. 5–56.
23. Agras WS, Walsh BT, Fairburn CG, et al. A multicenter comparison of CBT and IPT for patients with BN. Arch Gen Psychiatry. 2000;57:459–66.
24. Chen E, Touyz SW, Beumont PJ, et al. Comparison of group and individual CBT for patients with BN. Int J Eat Disord. 2003;33:241–54.
25. Fairburn CG, Marcus MD, Wilson GT. CBT for bulimia nervosa: a treatment manual. In: Fairburn CG, Wilson GT, editors. Binge eating. New York, NY: Guilford Press; 1993. p. 361–404.
26. Kaye WH, Nagata T, Weltzin TE, et al. Double blind placebo controlled administrates of fluoxetine in restrictive type AN. Br J Psychiatry. 2000;57:459–66.
27. Fairburn CG. Overcoming binge eating. New York, NY: Guilford Press; 1995.
28. Lewinsohn PM, Striegel-Moore RH, Seeley JR. Epidemiology and natural course of eating disorders in young women from adolescence to young adulthood. J Am Acad Child Adol Psychiatry. 2000;39:1284–92.
29. Stice E, Marti CN, Shaw H, Jaconis M. Prevalence, incidence, duration, remission, diagnostic progression, and diagnostic crossover of threshold and subthreshold eating disorders in a community sample. J Abnorm Psychol. 2009;118(3):587–97.
30. Stice E, Shaw H, Marti CN. A meta-analytic review of eating disorder prevention programs: encouraging findings. Ann Rev Clin Psychol. 2007;3:233–57.

31. McVey G, Tweed S, Blackmore E. Healthy schools-healthy kids: a controlled evaluation of a comprehensive eating disorder prevention program. Body Image. 2007;4:115–36.
32. Stice E, Shaw H, Burton E, Wade E. Dissonance and healthy weight eating disorder prevention programs: a randomized efficacy trial. J Consult Clin Psychol. 2006;74:263–75.
33. Stice E, Shaw H, Becker CB, Rohde P. Dissonance-based interventions for the prevention of eating disorders: using persuasion principles to promote health. Prev Sci. 2008;9:114–28.
34. Becker CB, McDaniel L, Bull S, et al. Can we reduce eating disorder risk factors in female college athletes? A randomized exploratory investigation of two peer-led interventions. Body Image. 2012;9(1):31–42.
35. Smith A, Petrie TA. Reducing the risk of disordered eating among female athletes: a test of alternative interventions. J Appl Sport Psychol. 2008;20: 392–407.
36. Stice E, Marti N, Spoor S, et al. Dissonance and healthy weight eating disorder prevention programs: long-term effects from a randomized efficacy trial. J Consult Clin Psychol. 2008;76:329–40.
37. Stice E, Presnell K, Gau J, Shaw H. Testing mediators of intervention effects in randomized controlled trials: an evaluation of two eating disorder prevention programs. J Consul Clin Psychol. 2007;75:20–32.
38. Green M, Scott N, Diyankova I, et al. Eating disorder prevention: an experimental comparison of high level dissonance, low level dissonance, and no-treatment control. Eat Disord. 2005;13:157–69.

# Part III

# Prevention and Management of Common Musculoskeletal Injuries in Active Females

# Prevention and Management of Common Musculoskeletal Injuries in Preadolescent and Adolescent Female Athletes

15

Mimi Zumwalt and Brittany Dowling

**Abstract**

Females transitioning from childhood to adolescence undergo a dramatic change in their body. In fact, this transitional period is where males and females start to diverge in terms of body composition, muscular strength, and bone mass. With the start of menses, female hormones begin to take an even more significant role on the body by greatly affecting the development/function of skeletal, muscular, and nervous systems. This rapid physiologic change during menarche exposes the female athlete's body to musculoskeletal injury, i.e., tendons, ligaments, muscles, and bones are all at risk. The skeleton in particular, is at greater risk due to the presence of open physes or "growth plates" at the ends of growing long bones. The young female athlete is therefore more prone to the multitude of sports-related injuries, and in fact at higher risk for certain types of trauma than their adult counterparts. Heightened awareness and a certain level of precaution need to be taken to help prevent potential injury. However, if trauma does occur then appropriate steps need to be taken to treat as well as protect the injured site for optimal healing and recovery.

**Keywords**

Adolescence • Menses • Menarche • Physes or "growth plate"

## 15.1 Learning Objectives

After completion of this chapter, you should have an understanding of:

- The important changes from prepubescence to postpubescence in females as far as anatomy and physiology of the musculoskeletal system are concerned

M. Zumwalt, M.D. (✉)
Department of Orthopaedic Surgery and Rehabilitation, Texas Tech University Health Sciences Center, Lubbock, TX, USA
e-mail: mimi.zumwalt@ttuhsc.edu

B. Dowling, M.Sc.
Department of Health, Exercise, and Sports Sciences, Texas Tech University, Colorado Springs, CO, USA

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_15, 

- The pertinent differences AND similarities between young females and males in terms of anatomy, physiology, and biomechanics of the musculoskeletal system
- The occurrence of various common musculoskeletal injuries more unique to young female athletes, especially during the adolescent growth spurt
- Several measures for prevention of athletic injuries from occurring in preadolescent and adolescent females
- Different methods of orthopaedic treatment for musculoskeletal injuries incurred by young female athletes, including specific recommendations for a regular conditioning program once healing is complete in order to enhance their physical fitness profile

## 15.2 Introduction

The pubescent growth spurt, especially the period transitioning from late childhood to adolescence, represents a time of tremendous transformation. Generally speaking, growth, development, and maturation play such an important role during these ever so important teenage years. Specifically, growth refers to a size increase of the body or part(s) thereof, while development is the functional alterations occurring along with anatomical/structural growth. Maturation is reached when the body has achieved its full potential in terms of growth and development, i.e., attainment of the adult form. Specific bodily units involved in this early stage of life alteration include the reproductive/sexual, physiological, and musculoskeletal systems. Unique musculoskeletal conditions can occur while the body is growing, and they need to be recognized and addressed appropriately. In addition, with the increased emphasis in youth sports and female involvement with athletic activities, this comes with a higher risk/frequency of orthopaedic injuries [1]. This chapter focuses on several of the more common musculoskeletal concerns of the young female athlete, plus prevention and treatment for these orthopaedic issues.

## 15.3 Research Findings and Contemporary Understanding of the Issues

### 15.3.1 The Important Changes from Prepubescence to Postpubescence in Females as Far as Anatomy and Physiology of the Musculoskeletal System Are Concerned

After the initial rapid increase in height during the first 2 years of life (when 50 % of adult height is attained) and up until the onset of puberty, a female's body grows steadily in terms of height and weight. Once puberty starts, growth velocity rapidly rises causing a dramatic increase in height and weight, with peaking around the age of 12. In girls, final adult stature is reached between the ages of 16 and 17. It is an established fact that regular exercise, along with an appropriate diet, is essential for proper bone growth in terms of width, density, and strength by mineral (calcium among others) deposition into the skeletal matrix [1–3]. Alongside the bony framework providing structural support and protection for the body, musculotendinous and ligamentous attachment to the skeleton help synergistically by providing dynamic and static restraints while allowing motion/movement of the head, trunk, and limbs, respectively. Muscle mass also increases steadily (from hypertrophy or enlarging fiber size) in response to hormonal influence, reaching its peak between the ages of 16 and 20 years in females. As a corollary to the increase in muscle mass, an accompanied gradual rise in muscle strength occurs in conjunction as well, and reaches a maximum by 20 years of age in young female adults [1]. Prior to puberty, girls and boys are comparable in terms of muscular strength. Then around the age of 15 or 16, adolescent females are only about 75 % as strong as pubescent males. This strength difference is more marked in the upper versus the lower extremities [4].

In addition to longitudinal growth and gaining in body size/weight, morphologically the

young female body shape is further transformed during the adolescent growth spurt, partly in preparation for later childbearing [1, 4]. Under the influence of estrogen, fat deposition increases which more than doubles the percentage of total body weight present at birth (25 % rather than 10 to 12 %). Both sex specific (surrounding breasts and hips) and subcutaneous tissue fat accumulate throughout the body of a young woman. The mechanism of adipose tissue storage, unlike that of muscle, stems from both hypertrophy (increase in size of fat cells) and hyperplasia (increase in number of fat cells). The latter process can continue throughout one's life span, dependent on diet and activity which is among other factors. With continued enhancement from the developing neuromuscular and endocrine systems, motor control continues to develop; however, this process starts to plateau at the onset of pubescence in young females. Girls tend to slow down physically as a whole during adolescence, partly due to greater fat deposition [1].

### 15.3.2 The Pertinent Differences and Similarities Between Young Females and Males in Terms of Anatomy, Physiology, and Biomechanics of the Musculoskeletal System

Before puberty, both growth and development in females and males parallel each other. During this childhood stage, prepubescent females and males are similar in stature and have the same relative muscle mass and strength. However, at the onset of puberty, due to the differing levels of secreted sex hormones, namely, the ratio of estrogen to testosterone, body composition changes start to separate young girls and boys in terms of maturation of the musculoskeletal system. Females have earlier onset of puberty than males; however, males undergo puberty for a longer time period, specifically 4 years as compared to 3 years [5]. Puberty for females starts around age 10–13 and ends at 15–16, whereas for males puberty begins at age 12–15 and ends at 17–18 years [1, 5, 6]. Because males grow for a longer period of time than females, they are on average 10 % taller and 17 % heavier than females [6].

Likewise, under the influence of a sudden ten times increase in testosterone production during puberty, boys markedly gain muscle mass at an accelerated rate, resulting in 40 % of total body weight as compared to the 25 % present at birth. However, muscle mass does not reach its peak quantity until 18–25 years of age. Females do not undergo a significant change in muscle mass with puberty, resulting in only minor changes of muscular strength. Around age 12 or so, muscular strength in boys, and to lesser extent, in girls, starts to improve along with an increase in muscle mass. However, the rate of growth is compounded in males at a faster rate, stronger proportion, and longer duration, peaking between the ages of 20 and 30 [1, 7, 8]. Before puberty, males and females have similar muscular strength. However, at ages 11–12 females are 90 % as strong as males, then at age 13–14 females are 85 % as strong as males, and by age 15–16, females are only 75 % as strong as males [9]. This muscular strength difference between the sexes can be accounted for by differences in body composition. Adult males have muscle mass comprising 40 % of body weight, whereas females have only 23 % of total muscle mass [9]. This difference is due to estrogen, which increases adipose tissue and has been shown to have a slight reducing affect on lean tissue [6]. In contrast, due to lower estrogen levels as well as greater levels of androgens, males have an increase in lean tissue and do not tend to accumulate a large quantity of fat. Body fat in physically unconditioned males ultimately reaches 15 % of total body weight, whereas unconditioned females average about 25 % total body fat. For a conditioned male athlete, the percentage is lower at 7 % and females around 10–15 % [6, 9].

The difference in muscle mass and adipose tissue between females and males also accounts for difference in ability, coordination, and learning of motor skills starting between the ages of 9 and 12 for both sexes. After age 12, the physical performance in males continues to accelerate and far exceeds that of adolescent females during puberty due to more muscle mass/strength and

less fat accumulation [1, 7]. Along the same lines, development of speed also favors adolescent males, which ultimately results in higher fitness levels as compared to girls. In fact, the sprint velocity increases yearly starting at 5 years old in both sexes, but maxes out in females between the ages of 13 and 15, yet does not peak in boys until the age of 16. Two phases encompass the phenomenon of speed development; the first begins at about age eight in both girls and boys, most likely attributed to improved coordination aided by the maturing nervous system. The second phase occurs about age 12 in females, and anywhere from age 12 to 15 in males as a natural progression from the larger body size, muscle mass and, along with these changes, speed, strength, power, and endurance [7]. Morphologically, pubescent boys maintain their body structure similar to prepubescent girls, again affected by the ratio of testosterone to estrogen. Interestingly, recent studies have demonstrated that neuromuscular control and preference, especially in the lower limbs of adolescent females during certain sporting activities, differs from that of male athletes, potentially putting these young girls much more at risk for knee injuries [10–13].

### 15.3.3 The Occurrence of Various Common Musculoskeletal Injuries More Unique to Young Female Athletes Especially During the Adolescent Growth Spurt

As previously outlined, the transition from prepubescence to postpubescence in females brings on a multitude of bodily changes, the majority of which involves the musculoskeletal system. While the skeleton is growing rapidly, muscles, tendons, ligaments, and other connective tissues must also grow to accommodate this accelerated bony growth. As a matter of fact, under endocrine and neural influence, locomotion and movement must be coordinated and adapted to the transforming skeletal framework in order to carry out life tasks and other physical endeavors, such as recreational athletics or competitive sports. Consequently, teenage female athletes involved in certain sporting activities are exposed to a higher risk of orthopaedic injuries. This is partly due to a marked rise in the number of school-aged females playing sports, from one female participating in competition compared to 27 male athletes in 1972, to the ratio of one female athlete competing to three males 30 years later. Half of all children aged 5–18 years in the USA are thought to participate in organized sports; this is equivalent to a total of 30 million children [5].

Susceptibility to injury results from both intrinsic and extrinsic factors. Intrinsic factors include age, gender, strength, flexibility, and previous injury. Extrinsic factors include biomechanics of movement skills, equipment, environment, training schedule, and intensity of activity. Both intrinsic and extrinsic factors can work together or against each other to provide a mechanism of trauma or prevention of injury. For example, the repetitive nature of throwing and the acquired high velocity are coupled to cause chronic injury over time. Trauma can result from direct impact (such as a collision with another player) or an indirect impact (such as a force transmitted through an extremity causing injury at a different site) (Fig. 15.1).

Aside from acute orthopaedic trauma occurring by sudden explosive episodes, more gradual, chronic bouts of repeated force over and over can also cause damage to the musculoskeletal system, resulting in overuse injuries and can eventually progress to stress fractures [13, 14].

Inflammation is the body's natural response to tissue trauma, which can develop in response to an acute injury or from repeated, chronic irritation. The primary functions of this physiologic reaction are to protect the body from harmful released histamines, dispose of dead/dying tissue, and promote the regeneration of new tissue. The first step of inflammation is an increase in blood flow and capillary permeability at the site of injury due to release of chemicals by injured tissue cells. This causes swelling or edema with a subsequent rise in osmotic pressure, in turn causing an increase in movement of proteins, white blood cells, and fluid to the site of injury.

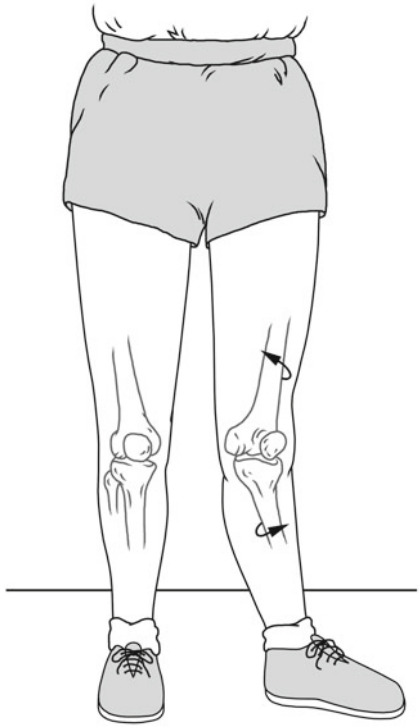


**Fig. 15.1** Risky limb landing attitude

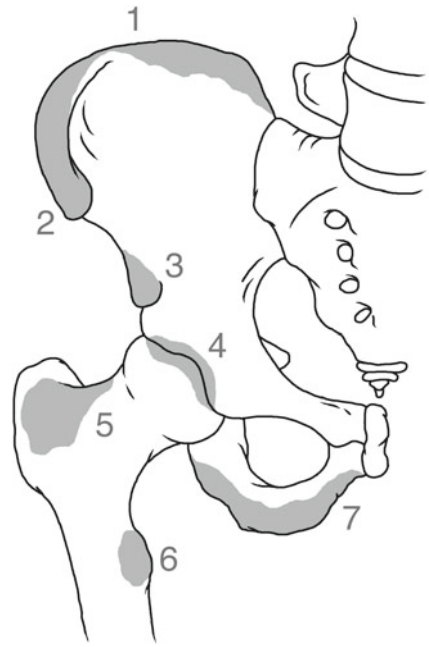



**Fig. 15.2** Pelvic apophyses

Edema and associated chemicals heighten the sensitivity of pain receptors, causing an increase in pain of the involved area. The next step is coagulation, causing fluid to be trapped at the injury site. This captured fluid, also known as exudate, dilutes and inactivates toxins, provides nutrients for cells, and contains antibodies. The final phase of inflammation is the breakdown of coagulation with a decrease in osmotic pressure causing chemicals, toxins, and dead cells to leave the injured site [13].

The greatest difference between the immature skeleton and the adult skeleton is the presence of physes, or open growth plates. The physis is comprised of cartilage cells that proliferate to create longitudinal growth. Apophysis is a similar structure but differs in that it is in close proximity to the tendinous attachment on bone (Fig. 15.2).

As discussed in Chap. 7, there is an inherent weakness of the physis during the adolescent growth spurt because the peak velocity height is obtained prior to peak bone mass. These growing anatomical structures are also at more risk of injury when exposed to excessive force because they are inherently weaker than the surrounding ligaments and tendons [9]. The enveloping tissue, including capsules (connective tissue around joints), has sufficient strength and therefore can resist an application of force better than the weaker physeal plate plus adjacent cartilage and bone. Consequently, any stress of sufficient magnitude to tear ligaments in adults tends to disrupt the physeal cartilage in bone of younger children, resulting in growth plate injuries or avulsion fractures (Fig. 15.3).

The latter results from sudden violent muscular contractions transmitted through various tendons inserting onto bone. Physeal injuries account for about 15 % of all fractures in children; with girls more prone to injury from ages 9 to 12 years and boys from ages 12 to 15 years [15]. Physiologic fusion of growth plates begins during the preteen years and is completed by the early twenties, occurring several years earlier in girls than boys. Prior to completion of the fusing process, any physeal damage incurred while the body is changing may lead to temporary or even permanent growth disturbance, which could

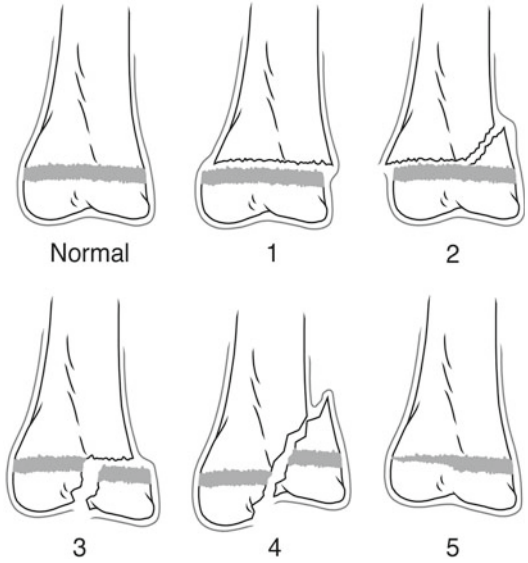



**Fig. 15.3** Growth plate injuries

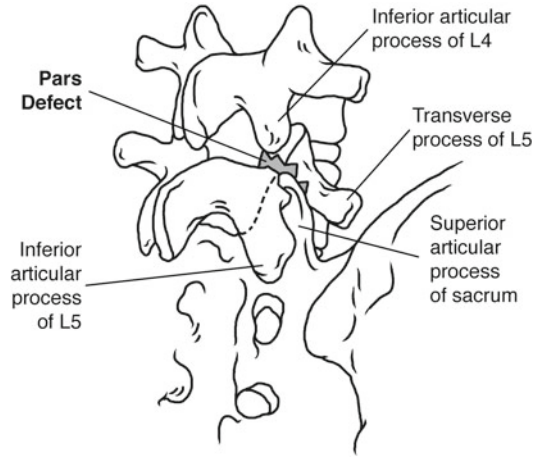



**Fig. 15.4** Spondylolysis

result in leg length discrepancy or angular deformity of the affected limb [9, 15].

Bone of the immature skeleton is more porous and not as dense as adult bone due to the fact it is more vascular with less mineral content, which translates to being more flexible but structurally weaker. The offset of this characteristic is that the periosteum or envelope surrounding growing bones is thicker, stronger, and biologically more active; thus, greatly promotes the process of healing at a much faster rate [9, 15].

The most common musculoskeletal injury found in young athletes involves chronic, repetitive, submaximal mechanical load applied to the same area, causing microtrauma. Continued microtrauma culminates in bursitis of soft tissues, strains of tendons, sprains of ligaments, and stress reactions or even frank fractures of bone. This type of orthopaedic injury tends to occur more often in the lower rather than upper extremities and results primarily from overtraining. The anatomical sites that are more prone to fatigue fractures depend on the types of movement performed in specific athletic activities. For example, in the lower extremity, volleyball and basketball players are at increased risk of stress injury to the tibia due to repeated jumping/landing. Long distance runners, especially those athletes engaged in training over 20 miles weekly, can suffer stress fractures to their femoral neck or fibula from excessive impact from running. In a similar fashion, because ballerinas have to be "en pointe" while dancing on the tips of their toes, they tend to sustain stress fractures of the second metatarsal more readily [14].

This does not mean that the upper extremities are exempt from microtrauma due to overuse. Stress fractures are commonly seen in the ulna from participation in tennis and fast-pitch softball players due to the nature of their racquet striking the ball or mechanism of pitching with their forearms, respectively. Along the same lines, the risk of radial fatigue fractures is higher in gymnasts as a result of repetitive load-bearing maneuvers on their wrists. Although quite rare, swimmers can injure their humerii from repeated overhead striking the water [14]. Furthermore, other athletic activities involving repetitive back extension maneuvers, i.e., dance, skating, and gymnastics can potentially place excessive load on the lumbar spine, resulting in a stress fracture of the pars interarticularis (spondylolysis) [4] (Fig. 15.4).

Additional variables contributing to other overuse type of injuries include environmental factors such as inadequate playing equipment, difficult/uneven surface terrain, faulty footwear, and inappropriate technique. The bodily internal environment also contributes to overuse injuries such as genetics, anatomic structure, and prior injury. Females in particular have altered internal

milieu due to monthly hormonal changes (as discussed in Chap. 7). Varying hormones in the circulation have different effects on soft tissue, timing of muscular contraction, and stability of joints. For instance, with the upper extremity, structural differences in young female athletes place them more at a mechanical disadvantage and, therefore, at an increased risk for certain types of overuse injuries. This stems from a shorter humerus compared to total arm length, less muscular strength, and more joint laxity. Consequently, sports involving excessive overhead activity such as throwing and swimming can contribute to rotator cuff tendinosis and shoulder subluxation [4].

The "miserable malalignment syndrome" is quite unique to young female athletes and is a constellation of lower extremity anatomical structures being misaligned. This syndrome is associated with primary quadriceps neuromuscular dominance, placing an increased risk for the knee, especially anterior cruciate ligament (ACL) injuries during various sporting activities [7, 11, 13]. The structural malalignment consists of a widened pelvis, genu valgum, increased internal tibial torsion, and pes planus. The Q-angle is accentuated, contributing to patellar maltracking which can cause anterior knee pain as well [4] (Fig. 15.5).

Additionally, adolescent female athletes tend to recruit their quadriceps prior to their hamstrings (the reverse occurs in males) when they land from a jump, along with a decrease in hip and knee flexion and an elevated valgus knee moment. The resulting misalignment of the lower body places a higher anterior shear force on the tibia which puts more strain on the knee, leading to one of the theories behind a several fold increased incidence of ACL tears in females involved in certain sports, i.e., basketball, soccer, handball, and softball [16, 17] (Fig. 15.6).

Osgood–Schlatter disease is commonly seen in young athletes aged 10–15 years. The patella tendon transfers high amounts of force generated by quadriceps contraction to the tibial tuberosity. Muscular contractions from sprinting and jumping in particular create high forces irritating the tibial tubercle, causing it to become tender and painful with subsequent quadriceps contraction. Increased force can also stimulate bone growth,

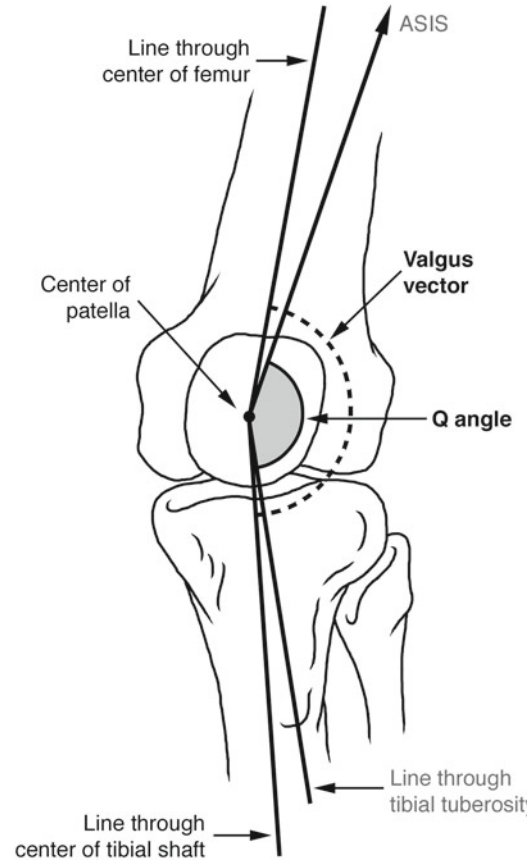



**Fig. 15.5** Alignment/forces across the knee joint

increasing the size of the tuberosity. Prominence of the tibial tubercle s is seen around the adolescent growth spurt. Treatment for Osgood–Schlatter disease is conservative with stretching, ice, anti-inflammatory drugs, and rest. Knee symptoms begin to abate, then sequester after the adolescent growth spurt, and the disease disappears with the closure of the physis [5, 18].

Collateral ligaments (medial and lateral) are at high risk of injury in adolescent athletes because they originate and insert into the inherently weak physis of the femur and tibia. The distal femoral physis is susceptible to any varus or valgus stress. The collateral ligaments are able to withstand higher amounts of tensile strength compared to the physis; therefore, the physis will fail first with sufficient force. When an injury has occurred to the collateral ligaments, evaluation of the physis needs to be done as well. With isolated collateral ligament injury, treatment can be initiated with

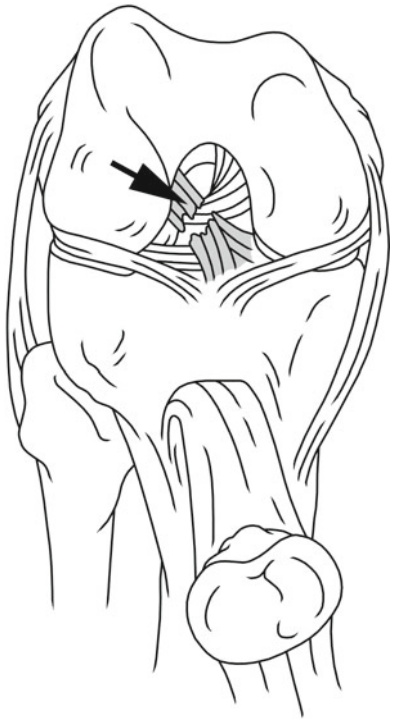


**Fig. 15.6** ACL (anterior cruciate ligament) tear

bracing, ice, rest, and, only in rare instances, surgical repair of high-grade tears [5, 18].

Anterior cruciate ligament (ACL) injuries are not seen in high numbers in adolescent athletes, occurrence of ACL ruptures is rare before age 11 but the incidence increases with age. Female athletes experience ACL rupture at least three to seven times more often than males, most likely due to sex-specific hormones, ligament strength and size, anatomical alignment, and landing differences (discussed further in Chap. 16). Injury to the ACL is seen in deceleration, change in direction (cutting), and in hyperextension during simple leg landing. Issues arise in terms of ligament surgery because most surgical techniques for reconstruction involve crossing the physis. Alterations of the physeal plate create concern for premature closure of the physis. Therefore, delay in ligament reconstruction is common in adolescent athletes until the growth plates have closed. However, delaying surgical treatment can cause an increase in knee injuries to cartilage due to the subsequent decline of joint stability [9]. Nonsurgical intervention can include knee bracing and reduction of activity to ensure there is no more tissue damage (especially the meniscus). Studies have found that injured adolescents treated with knee bracing alone have higher rates of further meniscal damage and in the long term, earlier osteoarthritis [19]. In fact, young athletes treated 12 weeks after ACL injury are four times more likely to develop medial meniscal tears and 11 times more likely to have lateral compartment chondral injuries at the time of surgery [20].

Dislocation of the patella is most common in females aged 14–18 years, most often occurring laterally. The mechanism of injury is most often due to internal rotation of the femur with a fixed foot causing the quadriceps to pull the patella laterally. A less common mechanism is a medial blow to the knee causing lateral dislocation. The dislocated patella, in general, usually spontaneously reduces; however, if it does not reduce, slow extension of the knee with medial force on the lateral patella will cause reduction [9]. The occurrence of subsequent dislocations is between 15 % and 44 %, with higher rates being associated with younger age [21].

Patellofemoral pain syndrome (PFPS) is most common in adolescent female athletes characterized by anterior knee pain. Pain is caused by activities such as repetitive knee flexion, jumping, climbing stairs, and sitting for long periods of time [9]. Treatment for PFPS includes strengthening the quadriceps (especially the vastus medialis oblique), restraint bracing, and patellar taping. Bizzini et al. found that, for adolescent female athletes, the use of orthotics for excessive pronation can help to relieve knee pain [22]. In extreme cases, surgery might be needed to correct PFPS such as lateral release, proximal patellar realignment, and medial tibial tubercle transposition [21] (Fig. 15.7).

Lateral ankle sprains are among the most common adolescent sports-related injury. This injury usually results from inversion of a plantarflexed foot injuring the anterior talofibular ligament (ATFL) and the calcaneofibular ligament (CFL). Symptoms of an ankle sprain include swelling, bruising, tenderness, and decreased range of motion. Studies have shown that bony tenderness in adolescent sprains is correlated

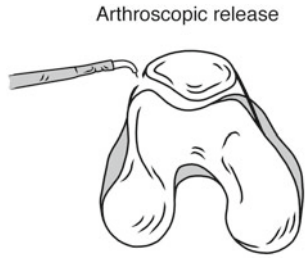



**Fig. 15.7** Surgical treatment for patellofemoral joint incongruency

with fracture; therefore, any ankle injury with extreme pain and difficult weight bearing should be radiographed to detect fractures. Treatment for ankle sprains consists of rest, ice, elevation, and compression [9].

The radius and the ulna comprise the bones of the forearm. The relative length difference between these two bones is known as ulnar variance (UV). When the ulna is longer than the radius, UV is positive; and, vice versa, when the radius is longer than the ulna, UV is negative. Variation in UV is determined by age, gender, ethnicity, and loading history. While the wrist is not designed to be a load bearing joint, in sports such as gymnastics, a great amount of force in applied to the wrist then transmitted to both the ulna and radius, with the radius receiving 80 % of the load. Particularly in those young athletes with immature bones, repetitive loading of the wrist can cause premature closing of the distal radial growth plate. As the athlete matures, the ulna continues to grow, thus causing a positive UV. Continual loading of the wrist causes other issues such as ulnar impaction syndrome, with progressive degeneration of the fibrocartilage and bones of the ulnar-sided carpus [9].

Injuries to the mid clavicle are one of the most common fractures seen in adolescent athletes. These fractures generally occur from a fall on the shoulder or from direct impact on the bone itself [18]. Surgical fixation of a clavicle fracture is not necessary for healing; therefore, treatment usually consists of bracing, ice, and pain medicine. Another common place for upper extremity fracture is the distal radius, accounting for one sixth of all fractures [9]. Symptoms of a fracture to the radius consist of tenderness, swelling, and inability to move the wrist [18]. The most common treatment is casting typically for 4–6 weeks, depending on the severity of the fracture and age of the patient [5].

Rotator cuff injuries are uncommon in adolescent athletes. However, injury to the shoulder is common in overhead sports. For example, little league's shoulder causes pain in the proximal humerus due to repetitive throwing. This is thought to be an overuse injury involving the physis and is not limited to baseball players but is also seen in swimming, gymnastics, volleyball, and tennis [5]. Treatment consists of rest, ice, and anti-inflammatory medications. Even though traumatic shoulder dislocations are rare in adolescents, it is the most common dislocated large joint involving athletes in general [9]. Dislocation usually results from an indirect force caused by landing on an abducted externally rotated arm [9]. After a shoulder dislocation, the issue of recurrence arises especially in young athletes. One study found recurrence rates of at least 75 % in athletes younger than 20 years of age [23].

Common back injuries that affect a large portion of adolescents and athletes are spondylolysis and spondylolisthesis involving the lumbar vertebrae, particularly at L4-L5 and L5-S1 levels. Spondylolysis is characterized by a defect in the lamina between the superior and inferior articular facets; whereas spondylolisthesis is the translational motion between adjacent vertebral bodies. There are different classifications of injury for these two mechanisms; however, for the young athlete, repetitive axial loading or hyperextension force causes microfractures and eventually progresses to complete bone failure [9, 24]. Treatment for both of these lumbar spine injuries consists of back bracing and hamstring stretching. Acute trauma to the back and neck (cervical spine) can result from high impact collisions and acrobatic sports. While it is uncommon to see fractures of the thoracic spine (since it is protected by the ribcage), when this traumatic injury does occur, it can result in devastating consequences [24].

### 15.3.4 Several Measures for Prevention of Athletic Injuries from Occurring in Preadolescent and Adolescent Females

The first step in musculoskeletal injuries for adolescent female athletes is prevention. As discussed above, there are different types of orthopaedic trauma incurred by these females during various sporting activities. Acute injuries while playing team sports are difficult to control since other players are involved. However, preventive measures should include employing appropriate safety equipment and avoiding extreme surrounding environmental conditions during actual competitions. In terms of the more chronic type of overuse injury, consider instituting and/or modifying the six S's: altered Structure or anatomic malalignment; Shoe wear pattern or status; Surface type or irregular topography; Stretching for flexibility; Strengthening of imbalanced muscle groups; and avoiding too much Speed too soon. As an example, for those participating in track and field events, correctly fitted footwear, along with correct running stride mechanics is extremely important in the prevention of uneven force distribution and excessive stress transmission through the feet with regard to shoe–ground interaction [25]. Beyond these external variables, the main factors which can be modified through training are: correct technique/appropriate skill level, altered parameter of play, and physical condition of the athlete [10, 26, 27]. For example, at the beginning of the regular sports season, athletes should not increase the volume of their workouts more than 10 % weekly to avoid overtraining [13].

The other very important variable to consider as far as prevention of musculoskeletal trauma in adolescent female athletes is their individual fitness profile. Studies have shown that preseason (several weeks) workouts, i.e., strength, endurance, conditioning, and plyometrics programs can increase physical fitness and help in decreasing the risk of knee/ACL injuries [27–29]. A previous study has shown that even only 8 weeks of training in preadolescents can result in increased muscle strength of ~75 % [8]. Since the period of training is relatively short, these regimens represent the initial physiologic adaptations involving the neuromuscular system, incorporating resistance/flexibility exercises, jumping/landing drills, and speed/agility maneuvers; all of these measures contribute toward improving muscular strength/endurance, anaerobic power, and sprint velocity [1, 30]. One of the most crucial elements involved with typical conditioning programs is teaching an athlete how to land with the lower limbs in a "safer" position to help protect against potentially devastating ACL injuries [11, 29] (Fig. 15.8).


**Fig. 15.8** Risky versus safe landing positions

### 15.3.5 Different Methods of Orthopaedic Treatment for Musculoskeletal Injuries Incurred by Young Female Athletes, Including Specific Recommendations for a Regular Conditioning Program Once Healing Is Complete in Order to Enhance Their Physical Fitness Profile

Once a female athlete has sustained an acute or overuse type of injury, proper treatment is imperative for a full recovery. Conservative steps can

be implemented if the injury is acute. First, strive to follow PRICE: pain control and Protect the injured part from further harm; relative Rest for the limb (not absolute immobility since the surrounding joints will become stiff) while maintaining gentle ROM (range of motion); apply intermittent Ice for inflammation (20–30 min every 4–6 h); use Compression wraps for swelling; and Elevate the limb above heart level to limit edema. This is basic first-aid type treatment for any kind of soft tissue trauma. If a fracture occurs, whether due to a sudden force or from gradual, repeated stress application, then add the following precaution: no weight bearing on the injured extremity and, if deemed severe enough, strict immobilization with external splinting/bracing or casting. For more specialized management, proceed toward orthopaedic consultation. If the fracture displacement is of sufficient magnitude, it could ultimately require surgical internal fixation [13].

Administration of medications may help with pain and speed up recovery of the injured body part. Over-the-counter nonsteroidal anti-inflammatory (NSAID) drugs can be used for swelling and pain; however, caution needs to be taken as not to remove the body's natural protective mechanism (masking effect). Another consideration is the medication's potential toward a delayed healing response due to blunting of the body's natural process of inflammation. Administration of vitamin C has been shown as a strong anti-inflammatory supplement plus aids in the process of bone building and scar tissue formation. Vitamin C is also an anti-oxidant, which helps in the repair of tissues. However, results are contradictory as far as the role of vitamin C in exercise recovery. In a similar fashion, recent studies have shown both anti-inflammatory as well as anabolic affects of omega-3s, seen particularly in fish oil. Like NSAIDs, caution needs to be taken when consuming these minerals as not to remove the body's response to inflammation and subsequent healing [13].

After an injury has occurred, modification of the training regimen must be modified as to not add further insult to the already injured extremity. The first step is reduction in the levels of training, i.e., magnitude, intensity, duration, and/or frequency to allow for full recovery after tissue healing. Cross training with a low or no impact type of activity (such as swimming or stationary bicycling) can be beneficial to keep from losing cardiorespiratory fitness. Administration of a formal physical therapy rehabilitation program to regain joint range of motion plus muscular strength/endurance is begun and aimed to progress towards sports-specific exercises after the initial period of recovery. Once functional testing of the affected extremity documents the restoration of objective measures of strength, girth, flexibility, endurance, among other parameters, demonstrating that an athlete is indeed physically recovered back to pre-injury status and again ready to participate in sports, clearance can be granted for gradual return to play [13].

Of utmost importance is the prevention of another injury to the same site; therefore, a conditioning program should be instituted and continued throughout the sports season, as well as extended through the off-season to help maximize performance and minimize injury [12, 13]. Warming up before any exercise by activating muscles around the involved joints appears to afford some protection from risk of injury due to improved proprioception of surrounding soft tissue restraints. However, there is debate on what type of warm-up is most beneficial in terms of maximizing performance and injury prevention. Recent movements in the athletic world have steered away from any type of passive, static stretching. In fact, studies have found that static passive stretching can inhibit neural muscular feedback and decrease peak muscular force up to 120 min after the stretch session [31]. Another study looked at the differences between static and dynamic stretching in 200-m race times; they found that runners who performed dynamic stretches before the race ran faster than those who stretched passively [32]. Contradiction exists in research regarding stretching as injury prevention; some studies have found stretching has a minimal prophylactic protective effect [33, 34], while others have shown better protective results [35]. Several studies have demonstrated the beneficial effects of strength training in children and adolescents in terms of enhancing athletic performance;

with minimal risk of trauma or growth disturbance as long as these programs are well supervised and submaximal exercises are being performed [5, 8, 12, 36, 37]. Specific guidelines regarding a resistance program for building muscular strength include the following: begin at age 7 or 8; add warm-up and cool down periods prior to and after exercise; use own body weight, light medicine balls or dumbbells; perform workouts that last between 20 and 30 min no more than 2–4 days/week (with rest days in between); initially, start with 1 set of 10–15 repetitions 2 times a week; progress to 1–3 sets of 6–15 repetitions concentrating on 6–8 different exercises, focusing on major muscle groups with mild tension/resistance while learning the correct technique of lifting; finally, sequentially accelerate training by small increments of weight (2–5 pounds), or 5–10 % increase in the exercise load weekly once the amount lifted is no longer challenging/fatiguing. For those young athletes who desire to maximize performance in various competitions, they should also practice sports-specific moves/skills as well. Additionally, engaging in any type of aerobic or other endurance type training is also well advised to enhance overall cardiac health in the long term [5, 8, 26, 27, 36]. The only caveat to this principle is that once training stops, regression or detraining occurs after a few to several weeks of inactivity, so the athlete must continue with the conditioning program to retain the highest level of fitness benefits. For all adolescent athletes, power-lifting type maneuvers/drills (i.e., single maximal lifts) should be avoided in order to avoid the potential of growth plate injury to their limbs [5, 26, 27].

## 15.4 Future Directions and Concluding Remarks

In summary, young females, during the time span from childhood to adolescence is accompanied by a myriad of bodily alterations, some of which resemble the changes experienced by males, while others are totally different. The main components involved with this life transition period of puberty include reproductive, hormonal, nervous, and musculoskeletal systems. The latter undergoes an extreme makeover to involve morphological and neuromuscular transitions. Growth, development, and then eventually maturation into the adult form is faced with an inherent risk, mainly orthopaedic trauma from soft tissues (ligament and musculotendinous structures) lagging behind the fast growing skeletal system (bones). Acute and chronic injuries may occur in preadolescent and adolescent female athletes participating in certain sporting activities. It is important to recognize these unique patterns of musculoskeletal trauma in order to protect the athlete and prevent injury, plus treat the offending problem if need be. When in doubt or if the injury is severe, seek appropriate orthopaedic consultation. The bottom line lies in helping to maximize gains and minimize risks by involving these young females in a well-supervised maintenance conditioning program for the musculoskeletal system. As such, they can compete and perform their best, while adapting to their ever-changing body during their teenage years and even further beyond that into adulthood.

## References

1. Wilmore J, Costill D. Growth development and the young athlete: chapter 17. Special populations in sport and exercise. Physiology of sport and exercise: human kinetics. Champaign, IL: Human Kinetics; 1994. p. 401–21.
2. Rogol A, Clark P, Roemmich J. Growth and pubertal development in children and adolescents: effects of diet and physical activity. Am J Clin Nutr. 2000; 72:521S–8.
3. Schoenau E, Frost HM. The “muscle-bone unit” in children and adolescents. Calcif Tissue Int. 2002 ;70: 405–7.
4. Timmerman M. Medical problems of adolescent female athletes. Wis Med J. 1996;95(6):351–4.
5. Cassas KJ, Cassettari-Wayhs A. Childhood and adolescent sports-related overuse injuries. Am Fam Physician. 2006;73(6):1014–22.
6. Brooks GA, Fahey TD, Baldwin KM. Exercise physiology: human bioenergetics and its applications. 4th ed. New York, NY: McGraw-Hill; 2005.
7. Borms J. The child and exercise: an overview. J Sports Sci. 1986;4:3–20.
8. Bencke J, Damsgaard R, Saekmose A, Jorgensen P, Klausen K. Anaerobic power and muscle strength characteristics of 11 years old elite and non-elite

boys and girls from gymnastics, team handball, tennis and swimming. Scand J Med Sci Sports. 2002; 12:171–8.
9. Whiting WC, Zernicke RF. Biomechanics of musculoskeletal injury. Champaign, IL: Human Kinetics; 1998.
10. Ahmad C, Clark M, Heilmann N, Schoeb S, Gardner T, Levine W. Effect of gender and maturity on quadriceps-to-hamstring strength ratio and anterior cruciate ligament laxity. Am J Sports Med. 2006; 34(3):370–4.
11. Barber-Westin S, Noyes F, Galloway M. Jump-land characteristics and muscle strength development in young athletes a gender comparison of 1140 athletes 9 to 17 years of age. Am J Sports Med. 2006;34(3): 375–84.
12. Hewett T, Ford K, Myer G. Anterior cruciate ligament injuries in female athletes. Part 2, a meta-analysis of neuromuscular interventions aimed at injury prevention. Am J Sports Med. 2006;34(3):490–8.
13. Withrow T, Huston L, Wojtys E, Ashton-Miller J. The relationship between quadriceps muscle force, knee flexion, and anterior cruciate ligament strain in an in vitro simulated jump landing. Am J Sports Med. 2006;34(2):269–74.
14. Verma R, Sherman O. Athletic stress fractures: part II. The lower body part III. The upper body—with a section on the female athlete. Am J Orthop. 2001;30: 848–60.
15. Ogden JA. Anatomy and physiology of skeletal development. In: Ogden JA, editor. Skeletal injury in the child. 2nd ed. Philadelphia: Saunders; 1990. p. 42.
16. Hewett T, Zazulak B, Myer G, Ford K. A review of electromyographic activation levels, timing differences, and increased anterior cruciate ligament injury incidence in female athletes. Br J Sports Med. 2005;39:347–50.
17. Powell J, Barber-Foss K. Sex-related injury patterns among selected high school sports. Am J Sports Med. 2000;28(3):385–91.
18. Kaeding CC, Whitehead R. Musculoskeletal injuries in adolescents. Adolesc Med. 1998;25(1):211–23.
19. Maffulli N, Del Buno A. Anterior cruciate ligament tears in children. Surgeon. 2012. doi:10.1016/j.surge.2012.02.003.
20. Henry J, Chotel F, Chouteau J, Fessy MH, Berard J, Moyen B. Rupture of the anterior cruciate ligament in children: early reconstruction with open physes or delayed reconstruction to skeletal maturity? Knee Surg Sports Traumatol Arthrosc. 2009;17:748–55.
21. Cofield RH, Bryan RS. Acute dislocation of the patella: results of conservative treatment. J Trauma. 1977;17:526–31.
22. Bizzini M, Childs JD, Piva SR, Delitto A. Systematic review of the quality of randomized controlled trials for patellofemoral pain syndrome. J Ortho Sports Phys Ther. 2003;33:4–20.
23. Deitch J, Mehlman CT, Foad SL. Traumatic shoulder dislocation in the adolescents. Am J Sports Med. 2003;31:758–63.
24. Patritti B. Running shoe cushioning impacts footground interface. Biomechanics. 2004: 57–67.
25. Benjamin H, Glow K. Strength training for children: risks versus benefits. Illinois chapter AAP. Illinois pediatrician sports medicine articles. http://www.illinoisaap.org/sportsarticles.htm.
26. Faigenbaum A, Kang J. Youth strength training: facts, fallacies and program design considerations. Am Coll Sports Med. 2005;15(4):5–7.
27. Faigenbaum A., Chu D. Plyometric training for children and adolescents. Am Coll Sports Med. 2001.
28. Myer G, Ford K, McLean S, Hewett T. The effects of plyometric versus dynamic stabilization and balance training on lower extremity biomechanics. Am J Sports Med. 2006;34(3):445–55.
29. Balabinis C, Psarakis C, Moukas M, Vassiliou M, Behrakis P. Early phase changes by concurrent endurance and strength training. J Strength Cond Res. 2003;17(2):393–401.
30. Bernhardt D. Strength training by children and adolescents. Am Acad Pediatr. 2001;107(6):1470–2.
31. Nelson AG, Driscoll NM, Landin DK, Young MA, Schexnayder IC. Acute effects of passive muscle stretching on sprint performance. J Sports Sci. 2005; 23(5):449–54.
32. Bartlett MJ, Warren PJ. Effects of warming up on knee proprioception before sporting activity. Am J Sports Med. 2002;36(2):132–4.
33. Herbert R, Gabriel M. Effects of stretching before and after exercising on muscle soreness and risk of injury: systematic review. BMJ. 2002;325:1–5.
34. McHugh MP, Cosgave CH. To stretch or not to stretch: the role of stretching in injury prevention and performance. Scand J Med Sci Sports. 2010; 20:169–81.
35. Izquierdo M, Hakkinen K, Gonzalez-Badillo J, Ibanez J, Gorostiaga E. Effects of long-term training specificity on maximal strength and power of the upper and lower extremities in athletes from different sports. Eur J Appl Physiol. 2002;87:264–71.
36. Guy J, Micheli L. Strength training for children and adolescents. Am Acad Orthop Surg. 2001;9(1): 29–36.
37. Power K, Behm MD, Cahill F, Carroll M, Young W. An acute bout of static stretching: effects on force and jumping performance. Med Sci Sports Exerc. 2004;36(8):1389–96.

# Prevention and Management of Common Musculoskeletal Injuries in the Adult Female Athlete

16

Mimi Zumwalt and Brittany Dowling

**Abstract**

As the female athlete transitions into an adult, her body is again under a multitude of changes, primarily from the effects of estrogen. This sex-specific hormone causes the change in body composition, muscular strength, neuromuscular firing, and bone composition. This bodily alteration opens the female athlete up to face different musculoskeletal injuries, with overuse trauma being the most common. However, injury to the knee in general and anterior cruciate ligament in particular is more of a concern for young adult female athletes, with tear rates exceeding male athletes several fold, especially for those participating in soccer and basketball. Current research continues to focus on ACL injury in females, looking at a wide array of potential contributing factors. Prevention of musculoskeletal trauma is crucial in keeping the female athlete healthy, and proper training/conditioning programs can help to reduce injury risk.

**Keywords**

Estrogen • Sex-specific hormones • Anterior cruciate ligament • Soccer and basketball • ACL injury in females • Training/conditioning programs

## 16.1 Learning Objectives

After completion of this chapter, you should have an understanding of:

- The fundamental differences in the anatomy, physiology, and body composition between adolescent and adult females in terms of the musculoskeletal system
- The relative similarities and pertinent differences between adult males and females concerning the anatomy, body composition, and biomechanics of the musculoskeletal system

M. Zumwalt, M.D. (✉)
Department of Orthopaedic Surgery and Rehabilitation, Texas Tech University Health Sciences Center, Lubbock, TX, USA

B. Dowling, M.Sc.
Department of Health, Exercise, and Sports Sciences, Texas Tech University, Colorado Springs, CO, USA
e-mail: dowlingb86@gmail.com

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_16, 

- Various more common types of orthopaedic injuries sustained by adult females involved in certain athletic activities
- Several measures for prevention of musculoskeletal injuries incurred by adult female athletes
- Different modes of treatment for orthopaedic injuries sustained by adult females participating in certain sporting activities

## 16.2 Introduction

As growth proceeds from childhood through adolescence then finally culminates into adulthood, the developing female body carries along with it a multitude of changes. Not only does a mature woman have to face morphologic challenges, she must also adapt to structural, hormonal, and metabolic alterations as well. Indeed, the musculoskeletal system is certainly no exception to this growing rule throughout the different life stages. Although it is healthy to be engaged in a regular exercise program, participating in multiple athletic competitions and sporting activities can leave the adult female more vulnerable to orthopaedic injuries; especially if she is not sufficiently fit to perform her best in extremely demanding levels of physical play.

This chapter focuses on several anatomical features unique to the adult female athlete; different types of musculoskeletal trauma which tend to be more common for women participating in sports, along with methods of injury prevention and how to go about treating these orthopaedic issues once they do occur.

## 16.3 Research Findings and Contemporary Understanding of the Issues

### 16.3.1 The Fundamental Differences in Anatomy, Physiology, and Body Composition Between Adolescent and Adult Females in Terms of the Musculoskeletal System

To recap some of the similarities and differences between young females and adult women, the following anatomical changes are found to be fairly consistent: breast tissue development, fat deposition around the hips, thighs, and buttocks, plus broadening of the pelvis, all of which herald the onset of puberty under the influence of the female sex hormone, estrogen [1, 2] (Fig. 16.1).

This transition stage usually occurs between the ages of 12 and 14. Estrogen also brings about longitudinal skeletal growth, with the final bone length achieved between 2 and 4 years after pubescence. A woman's body composition continues to fluctuate during the teenage years, then at some point in the mid-20s, more adipose tissue is accumulated and muscle mass starts to decline, at a rate of about 3 kg per decade (greater than ½ pound per year). The rise in total body fat along with loss in the quantity of fat free mass is due to lower levels of physical activity, relative lack of testosterone, and consuming the same quantity of caloric intake [2]. In terms of skeletal density and therefore bony integrity, calcium, among other essential minerals, continues to be deposited into

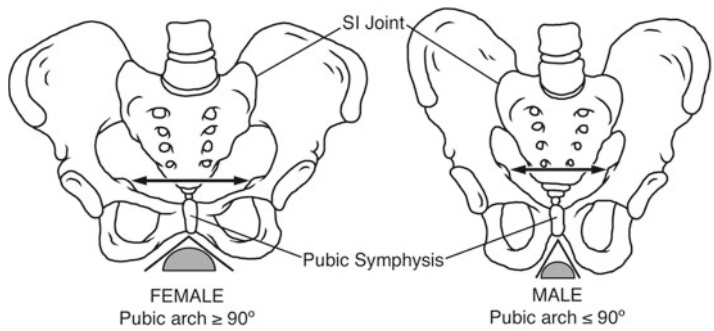



**Fig. 16.1** Pelvis morphology

the skeleton to build up bone as the body is steadily growing. This process of calcium deposition into the bone bank thus increasing the quantity of bone gained, becomes accelerated during the adolescent growth spurt of puberty spanning the teenage years, and then reaches its peak in terms of bone mass acquisition in the early 20s. After this period of very rapid bone deposition, bone loss begins to occur gradually during the late 20s or early 30s depending on both nutritional and hormonal status, along with the amount of mechanical stimuli applied [3] (see Chap. 7) (Fig. 16.2).

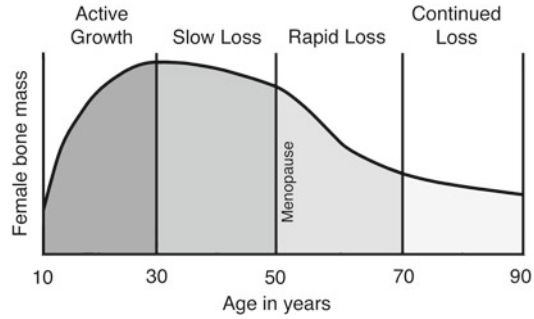



**Fig. 16.2** Rate of bone loss through a woman's lifetime

### 16.3.2 The Relative Similarities and Pertinent Differences Between Adult Males and Females Concerning the Anatomy, Body Composition, and Biomechanics of the Musculoskeletal System

Up until the onset of puberty, anatomical structure and body composition between males and females are fairly similar in terms of height, weight, girth, bone width, and subcutaneous fat. The body build, shape, and size of both sexes begin to diverge once the endocrine system starts to undergo changes. Two sex-specific hormones, estrogen and testosterone, begin to take over to influence the development of adolescent features, separating teenage girls and boys in terms of anatomy as well as functions of the musculoskeletal system [1, 2]. During the adolescent growth spurt females tend to deposit more fat around the breasts, hips and thighs. This gender related adipose tissue deposition, in addition to that surrounding internal organs, is termed essential fat. Essential fat in females composes about 9–12 % of total body weight, as compared to only 3 % in males. The other type of adipose tissue is storage fat, which is comparable in both sexes, comprising about 15 % of body weight [2, 4–6]. At physiological maturation, the average adult female non-athlete carries between 18 and 26 % of adipose tissue, whereas mature men only contain about 12–16 % body fat on average [4, 5]. However, for female athletes engaged in excessive exercise, especially long distance runners who train and compete over 100 miles per week, their percentage of body fat can be reduced to well below 10 % [4, 6]. In fact, some elite endurance athletes can eventually attain body fat of only around 6–8 %. It is estimated that body fat below 11 % in women is enough to cause amenorrhea [2].

Males tend to carry more adipose tissue in the abdomen/flank and upper body versus females, who store fat mostly around their hips and lower body [2]. The different body fat distribution in men and women tends to affect athletic performance. For example, because females have greater fat content they have increased buoyancy in water. This is deemed advantageous by decreasing drag and lowering energy expenditure by 20 %. However, the higher fat percentage is thought to hinder physical performance in many endurance sports from having to carry the excess body weight (though hard evidence is somewhat lacking) [4–6].

Generally speaking, between 11 and 12 years of age, girls are about 90 % as strong as boys. By the time they are 15–16 years old, females are about 75 % as strong as males [4, 6]. When adjustments are made for body mass, men are at least one-third stronger than women [5]. Specifically, females are only 50 % as strong in the upper and 75 % in the lower body as males [6]. When muscle mass is taken into account, relative leg strength is similar in both sexes, but upper extremity strength in females still lags behind that of males [1, 4, 6]. This is due to men having more muscle mass relative to body weight than women, 40 % as compared to 23 %.

The lack of muscle strength stems from women having decreased fiber size (15–40 % less), making the cross sectional area about 60–85 % smaller as well [4, 6]. In fact, this relative difference in fat free mass tends to be lower in females from age 7 until 25 years of age. After the mid-20s, lean muscle mass begins to decline in both sexes, at a rate of ¼ to ½ pound yearly [2]. In correlation to a greater amount of lean muscle mass in men, the skeletal framework in males is larger as well; they also mature later and attain more final height (taller) and weight (heavier) than females from undergoing a longer period of growth [6]. However, under estrogen's effect, growth rate of the female skeleton is greatly accelerated, culminating in the final bone length to be achieved earlier only a few years after the onset of puberty. In other words, females cease to grow faster—their body and bones reach a plateau after 2–4 years of fairly rapid growth. Therefore, men reach final skeletal maturity about 21 or 22 years old, rather than 17–19 years of age in women [2].

In terms of bony and articular differences, females' bones are smaller as well and therefore their joints have less surface area. Women have shorter and smaller limbs relative to body length as compared to men. The length of lower extremities in women is 51.2 % of total height versus 56 % in men [7]. Additionally, men have broader shoulders, larger chests, and narrower hips; women, on the other hand, have wider pelves, more varus hip, and higher knee valgus angles (Fig. 16.3).

This anatomical difference, combined with an overall smaller stature and wider pelvis, gives women a lower center of gravity. The increased angular inclination in the lower limbs of females causes an asymmetrical force distribution/transmission through the extremity's overall alignment from the hips to the ankle, and subsequently can contribute to a myriad of overuse type of musculoskeletal injuries [1, 2, 4–6].

Neuromuscular recruitment, primarily in the lower extremities in women, differs from that of men after puberty, especially during landing tasks [4, 8–12]. This altered neuromuscular recruitment has been linked to increased risk for injury during athletic activities. Women also have increased joint laxity, which is partially attributed to less lean muscle mass that can restrain excessive joint motion [13]. Joint laxity differences are most profound in the knee, ankle and elbow joints. Studies have shown this increase in joint laxity along with differential neuromuscular recruitment may be responsible for the greater incidence of ACL injury in females compared to males [12] (Fig. 16.4).

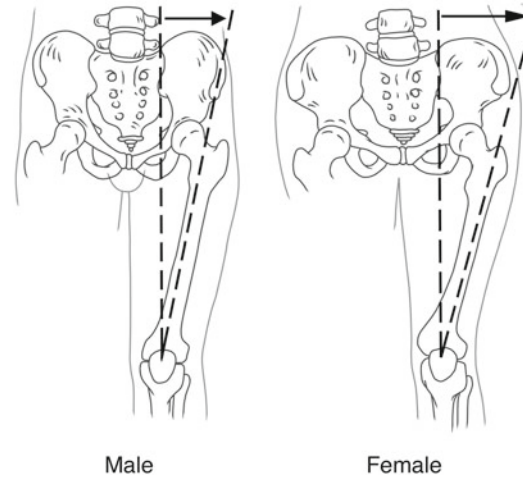



**Fig. 16.3** Hip–knee angular difference

### 16.3.3 Various More Common Types of Orthopaedic Injuries Sustained by Adult Females Involved in Certain Athletic Activities

The involvement of women in athletic activities, ranging from mild exercise to elite performance, has skyrocketed over the past 100 years. This comes from a change in social norms and attitudes towards women as well as legislature ensuring equal opportunity for females. Subsequently, over the past 30 years, the role of women in athletic competition has been revolutionized, from sitting in the stands to standing on the sidelines, and of course playing in sporting events. Alongside the tremendous rise in female sports participation, comes with that visible gender


**Fig. 16.4** Risky versus safe landing positions

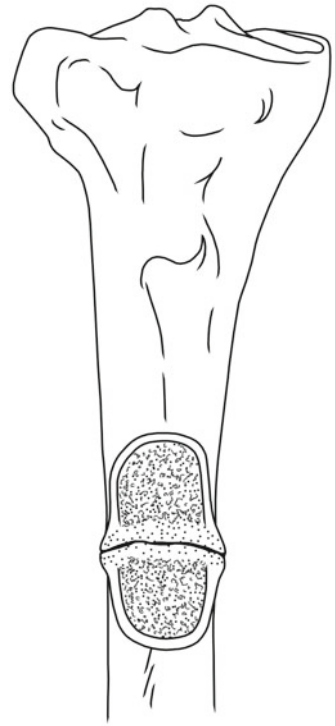


**Fig. 16.5** Tibial stress fracture

differences between men and women, especially in the different types of musculoskeletal injuries incurred among other unique orthopaedic issues occurring in athletics [3–6, 14–16].

The following depicts musculoskeletal conditions more commonly seen in female athletes that may vary in types and incidence according to different sporting activities. Even recreational events such as running can bring about 25–65 % of injuries severe enough to keep women away from training. In fact, about half of these female runners must seek medical care for their musculoskeletal problems. Other modes of physical activity, which portend a similar rate of injury risk, include group exercise participants, soccer players, basketball players, and handball players [14].

As discussed in Chap. 15, overuse injuries are extremely common in the female athlete. The rise in overuse injuries can be contributed to the relative rise in the number of females involved in athletics as well as an increase in intensity along with year-round participation. Stress fractures are seen in both men and women, with the most common bone fractured being the tibia (33–55 %) (Fig. 16.5).

However, fractures to the femoral neck, tarsal navicular, metatarsal, and pelvic area are more commonly seen in the female athlete [17]. Specific sites of stress fracture are also dependent on the type of sport; for example, high jumpers most commonly fracture their medial malleolus and tarsal navicular [18]. Associated risk factors can be due to internal body structure/function, including muscle imbalance, limb malalignment, and fluctuating hormones; or attributed to external factors such as the type of footwear, running style, and training errors (i.e., exercise mode/frequency/intensity/volume). Additional contributors to the increased risk of stress fractures can involve psychological and medical issues, including inadequate/inappropriate nutrition, disordered eating, osteopenia, and hormonal irregularity [1, 4, 5, 8, 19]. The presence of stress fracture shows itself as gradual onset of pain induced by activity as well as tenderness to the bony site when palpated. Radiographic imaging should be utilized to determine if a stress fracture is present. General treatment for a stress fracture

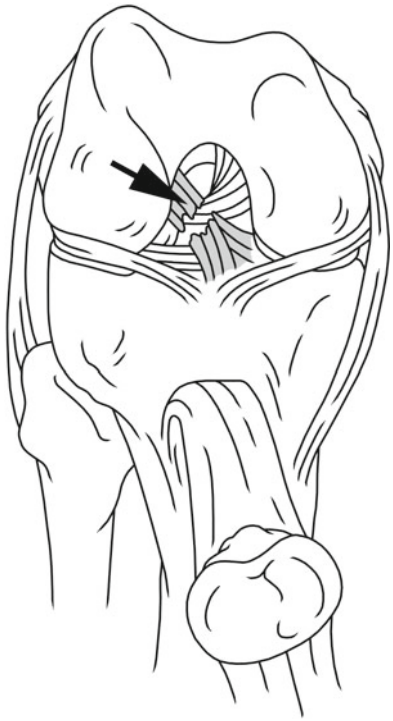


**Fig. 16.6** ACL (anterior cruciate ligament) tear

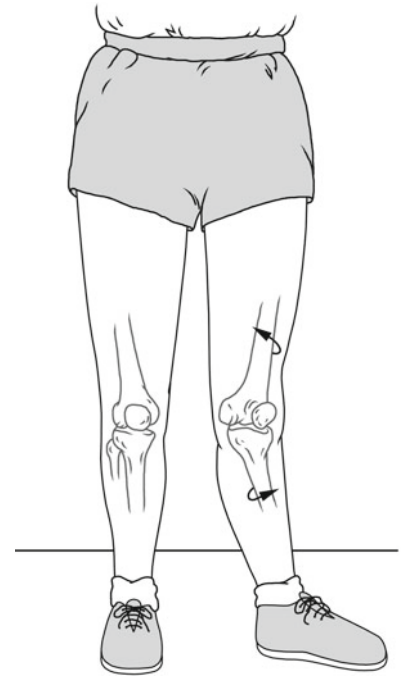


**Fig. 16.7** Risky limb landing attitude

is reduction in activity and relative rest; however, if the stress fracture does not respond to resting or casting, a surgical procedure to fix the problem may be necessary. Non-impact exercise can be beneficial during the "rest" period to help maintain cardiovascular fitness. For example, cross-training such as swimming, water running, and bicycling can also be beneficial as well [17–19].

One of the most common injuries in female athletes is trauma to the anterior cruciate ligament (ACL). The incidence of injury in women competitors can range anywhere from two to ten times higher than male athletes [3–6, 8, 12–14] (Fig. 16.6).

Specific sports portend different rates of ACL tear/injury. For example, collegiate female soccer players are 2.6 times more likely to sustain an ACL injury as compared to males, whereas basketball players are 5.75 times more likely to have an ACL tear [20]. Injuries to the ACL are also commonly seen in gymnastics, downhill skiing, field hockey, team handball, and lacrosse. While it has not been clearly defined as to why there is an increased risk in ACL injury in women, the etiology of this gender specific phenomenon is theorized to be multifactorial, including anatomical, hormonal, biomechanical, neuromuscular, and environmental. Other contributing factors may include skill level, physical condition, imbalanced thigh musculature (especially hamstring weakness as compared to the quadriceps), increased flexibility, and decreased proprioceptive capability [4, 5, 9]. Risky positions when landing have been previously alluded to and usually involve an off-balance body position especially on one limb. Injury in women occurs most often in response to a valgus load with hip internal rotation and external tibial rotation as seen in cutting maneuvers, and can be exacerbated if an external lateral force is applied to the knee [20, 21]. The other, less common mechanism for ACL injuries occurs during knee hyperextension with external tibial rotation while landing, which tend to be more predominant in basketball and gymnastics [20] (Fig. 16.7).

Joint stiffness and stability can be maintained by active contractile soft tissues. Stiffness is the resistance of bodily structures in response to a force, and active joint stability can be controlled

by voluntary muscle contraction. Wojtys et al. measured anterior tibial translation and muscular contraction to an externally applied stress, finding that translation was decreased with increased muscular co-contraction. However, they found that women naturally have decreased joint stiffness, indicating a reduced ability to protect the knee from external forces [21].

Recent studies have looked into the possibility of joint laxity due to fluctuating hormones during the menstrual cycle. It has been proposed that the rate of ACL injury increases during the initial and late follicular phase, accompanied by a surge in estrogen (see Chap. 7) [22]. However, there has been no consensus as to whether hormonal changes during menses directly cause the increased risk of ACL injury; thus more research needs to be conducted. As discussed previously, the timing of neuromuscular firing is different in women during landing tasks and can be linked to ACL injury. Huston and Wojtys found female athletes initially activate their quadriceps for knee stabilization, whereas males recruit their hamstrings first. They also found female athletes took longer to generate maximum hamstring muscle torque as compared to males [23]. More recently, research trends have shifted from looking at hamstring and quadriceps to analyzing the effect of activation and strength of hip musculature in relation to ACL injuries. When the knee is loaded, biomechanically, the hip abductors and external rotators are activated preventing hip adduction and internal rotation, which causes a valgus force at the knee [24]. Failure of the hip abductors and external rotators to fire causes an increase in the Q-angle which subsequently adds more load to the knee joint [25]. Brent et al. found a decrease in hip abduction strength in females during cutting maneuvers, suggesting a decrease in muscular contraction at the hip can lead to an ACL injury at the knee [26].

Contributing factors to anterior knee pain and dysfunction stem from static structural misalignment from the pelvis to the feet, dynamic imbalance of limb muscular strength, excessive soft tissue pliability or extreme stiffness, or a combination thereof [8]. Specifically speaking, this

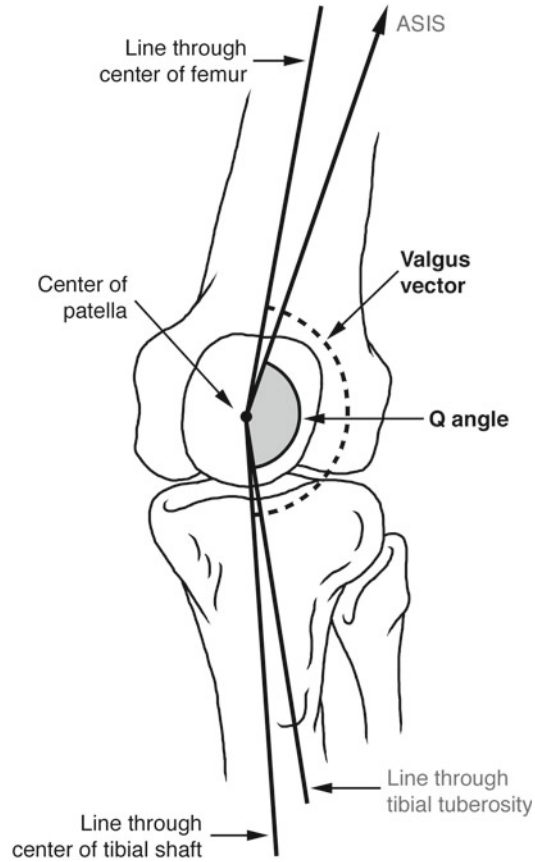



**Fig. 16.8** "Miserable malalignment syndrome"

entity is often named the "Miserable Malalignment Syndrome," associated with an increase in hip external rotation and femoral anteversion coupled with a higher Q-angle, genu valgum, tibia vara, and a hypermobile patella [1, 4, 6, 16] (Fig. 16.8).

The body, as an attempt to center the knee under the hips and above the ankle, compensates by internally rotating the femur, along with externally rotating the tibia and pronating the foot, all of which asymmetrically loads the patellofemoral joint [25, 27].

Patellofemoral pain syndrome (PFPS) is very common in female athletes, especially runners, occurring in 20 % of females as compared to 7.4 % in males [28]. The higher rates of PFPS in women are thought to be due to structural alignment as well as biomechanical and hormonal differences. However, the exact mechanism for the


**Fig. 16.9** Patellofemoral joint incongruency

injury and pain is still unclear and seems to vary between athletes. Three major factors contributing to the development of PFPS are lower extremity and/or patellofemoral malalignment, quadriceps muscle imbalance and/or weakness, plus physical overload of the patellofemoral joint [27]. Malalignment can be caused by increased pressure from excessive tightness of the lateral patellar retinaculum (soft tissue restraint) or from a shallow trochlear grove of the distal femur and/or high or low riding patella, all of which can contribute to PFPS and anterior knee pain [1, 6, 16] (Fig. 16.9).

Furthermore, as females flex their knees from full extension to about 30°, the femoral condyles do not fully support the patella, contributing to a more lateral riding position. Sports such cycling or running on hills, or those involving repeated strikes to the anterior knee region (volleyball) tend to place more stress across the patellofemoral joint, in turn causing an increased incidence of experiencing painful knee symptoms [4]. Malalignment can also contribute to increased risk for subluxation or frank dislocation of the patella. A common mechanism of patellar dislocation stems from internal rotation of the femur with a fixed foot causing the quadriceps to pull the patella laterally which may be a result of an acute, traumatic episode or from chronic, repetitive type of activity [1, 4, 20]. The dislocated patella, in general, usually spontaneously reduces; however if it doesn't reduce, slow extension of the knee with medial force on the lateral patella will cause patellar reduction [20].

Iliotibial band syndrome (ITBS) is one of the most common overuse injuries in female athletes. The incidence of ITBS are sports dependent and most commonly seen in long distance runners at a rate of 4.3–7.5 %. Iliotibial band syndrome is a result of inflammation and irritation of the distal attachment of the iliotibial band that runs alongside the lateral femoral condyle. Inflammation is exacerbated with repetitive flexion and extension motions, as seen in running and bicycling. Anatomical factors that are thought to increase the risk of developing ITBS are excessive tibial internal rotation, genu varum, and increased foot pronation. It has also been suggested that weakness in the hip abductor muscles can also contribute to the development of ITBS. Treatment of ITBS is generally conservative and consists of rest, heat and ice to decrease inflammation, stretching of the hamstrings, quadriceps, and ITB, along with physical therapy for strengthening. In extreme intractable cases, surgical intervention to increase the length of or partially release the tendinous band might be required [4, 16].

Other lower extremity conditions more prevalent in athletic females involve the foot, especially if special shoe wear is deemed necessary for sports participation. The other factor playing a contributory role toward female foot problems lies in the type of sports consisting of frequent starts, decelerations, and sudden stops such as basketball, causing excessive forward and backward movements of the foot inside the shoe and friction around the lesser toes. Again, this overuse condition is exacerbated by wearing athletic shoes originally designed for males, not taking into account that a female's foot is shaped wider in front (forefoot) and narrower in the back (hindfoot) [1, 4, 6]. Bunion (or hallux valgus) formation results from bursitis overlying the first metatarsal head of the great toe. Bunions are commonly seen in female athletes and in fact exceed the incidence in males by about nine times. More than half the women in the USA have a bunion. This condition is due to a wide forefoot shape combined with wearing a narrow shoe toe box, causing excessive pressure and therefore more frictional wear, resulting in inflammation and pain. This inflammatory entity is aggravated by midfoot pronation or flat arches and wearing high heels [1, 4, 16]. Treatment for bunion pain is first and foremost properly fitting shoes. Shoes designed with a wide toe box are ideal for women suffering from bunions. Orthotics can also be worn to alleviate pain and provide extra comfort, support, and

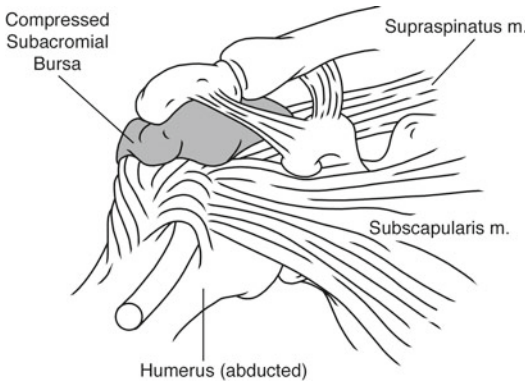



**Fig. 16.10** Impingement syndrome

protection. Other modalities for pain relief are bunion shields, splints, and bandages. However, if the pain becomes unbearable, a surgical procedure can be performed to correct the bony protuberance. Bunionectomy will remove the bony outgrowth plus realign the bones and soft tissues [16].

The shoulder girdle joint seems to be more susceptible in females in terms of orthopaedic injuries, especially when engaging in higher risk athletic activities such as gymnastics, swimming, diving, throwing/pitching maneuvers, tennis, and volleyball. Again, etiology of this upper limb trauma could also be acute or chronic, with anatomical malalignment, structural imbalance, poor posture, muscular weakness, soft tissue inflexibility or excessive laxity, all contributing to the incidence and severity of injury. Biomechanically speaking, female athletes performing in sports requiring overhead activities tend to place undue stress on the soft tissue stabilizers (both static *and* dynamic) surrounding the shoulder girdle articulations, resulting in bursitis and rotator cuff tendinosis, leading to inflammation and pain (impingement syndrome) (Fig. 16.10).

For example, female swimmers, since their bodies and arm lengths are generally shorter; they must utilize more strokes across the water to cover the same distance as men, increasing the number of repetitive insults placed across the shoulder joint. Another cause of shoulder problems in overhead female athletes is due to increased laxity of the joint capsule, allowing the humeral head to "ride" out and back in over the glenoid, making the dynamic stabilizers such as the rotator cuff muscles work that much harder to keep the "ball" centered in the "socket", again causing painful symptoms [1, 4, 6] (Fig. 16.11).

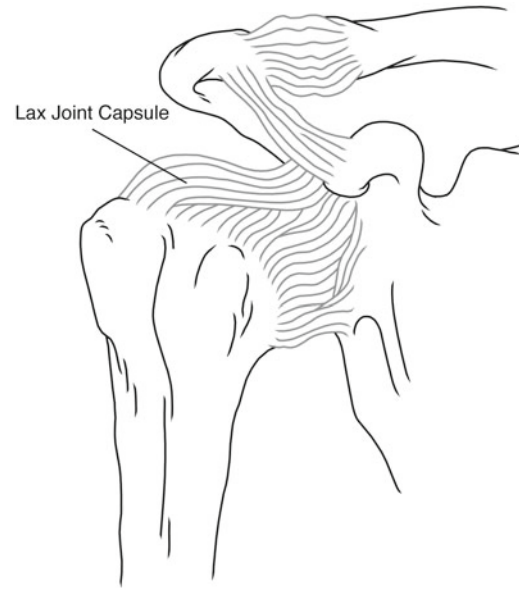



**Fig. 16.11** Shoulder instability

Similarly for throwing mechanics, since humeral length is less in women but forearm length is the same as men, along with having narrower shoulder girdles, the lever arm is shorter as well, making overhead winding motions more difficult, placing differential forces on the surrounding supportive musculature [6].

The elbow is also more susceptible to "wear and tear" in females, with an incidence of 10 % versus 1–5 % in males primarily between 42 and 46 years of age. Lateral epicondylitis, also known as tendinosis or tennis elbow, is degeneration of the tendons attaching to the lateral humeral epicondyle. This overuse condition is due to repetitive forearm rotation affecting the extensor muscle(s). Symptoms consist of pain and tenderness to touch at the lateral epicondyle and treatment is very limited. Generally, rest, ice, and bracing are recommended initially, then steroid injections, with surgery in only recalcitrant cases [4].

Spondylolysis is a common back injury seen in female athletes, particularly affecting the fourth and fifth lumbar vertebrae (L4–L5). Injury usually occurs at the junction between the superior and inferior process or pars interarticularis. Injury rates

are higher in women participating in gymnastics, skating, and dance. The presenting symptom is unilateral lower back pain, exacerbated by lumbar extension. Confirmation of this type of stress fracture is obtained through X-ray, although sometimes radiographs cannot pick up the injury site. In these cases, other imaging modalities such as an MRI may be ordered to reveal the damaged area. Treatment for spondylolysis is conservative, primarily with rest and/or bracing, with surgery only in extremely rare cases [1, 4, 6].

### 16.3.4 Several Measures for Prevention of Musculoskeletal Injuries Incurred by Adult Female Athletes

As previously stated, as compared to males, both intrinsic and extrinsic factors contribute to the increased incidence and varied types of orthopaedic injuries incurred and musculoskeletal conditions occurring in adult female athletes. Intrinsic factors include sex specific changes in musculoskeletal anatomy and physiology affecting relative limb length, lower extremity alignment; body composition, muscle mass/strength, and neurological recruitment. Extrinsic factors involve physical fitness, proprioception, playing level, competitive environmental conditions, sporting equipment, athletic wear, especially shoes, along with interaction/friction between footwear and ground surfaces [1, 2, 5, 6, 29]. Generally speaking, altering intrinsic conditions is virtually impossible because this is inherent within the athlete. However, neuromuscular control can be modified through training and conditioning to affect the manner by which muscles are recruited and the force they produce. This can affect the way the athlete lands during certain athletic activities, thus potentially decreasing the risk of lower extremity injury. Additionally, strengthening exercises for the hip (gluteus extensors/abductors/adductors), thigh (quadriceps/hamstrings), and leg (gastrocsoleus) muscles can be instituted to help support key joints of the lower limb (hip–knee–ankle), respectively. The importance of physical training lies in balancing the anterior and posterior musculature to optimize muscular contraction with neurological activation while playing sports. Furthermore, other aspects of the workout program should be considered, such as stretching for flexibility, ipsilateral stance exercises for balance/proprioception, core conditioning for trunk stabilization, plyometrics for anaerobic power, and agility drills for speed/coordination. As discussed in Chap. 15, the different types of warm-up, in particular static stretching, to potentially prevent injury and enhance performance, is being highly debated. Trends are now moving towards actively recruiting muscles as opposed to passively stretching them in preparation for activity [6].

To be extremely effective, training programs must be at least 6 weeks long, and sessions must be more frequent than once a week [5, 6, 30, 31]. Sports specific exercises should also be integrated into the workout regimen to ensure that correct and strict technique is maintained. Similar conditioning programs can also be applied to the upper body, enhancing physical fitness ability and minimizing injurious mechanisms. The training program needs to focus on deficits in range of motion and strength of the shoulder girdle region, especially the rotator cuff muscles. Other musculotendinous units surrounding this upper limb articulation, such as the pectorals (major and minor), latissimus dorsi, deltoids, triceps, and biceps brachii, should also be strengthened for added limb support [1, 4, 6].

Control of external factors can contribute to the prevention of injury. Modification of environmental conditions can be done to a certain extent, ensuring that playing equipment is adjusted to body size, and making sure that athletic gear, especially shoes, are well padded/fitted to the foot will also decrease risk of trauma. As far as prevention of stress injury is concerned, avoidance of excessive, repeated bouts of impact activity will help to reduce this and other types of overuse orthopaedic conditions. Cling to the principle of gradual moderation when increasing training intensity, duration, and frequency in order to optimize athletic performance and minimize musculoskeletal injury [1, 6, 14, 16].

In short, the majority of extrinsic factors contributing to adult female musculoskeletal conditions can be modified to decrease the incidence of orthopaedic injury. The mainstay of

this prevention strategy lies in physical conditioning programs, training women athletes to keep their body in balance as far as strength, flexibility, and proprioception, along with optimizing other factors such as speed/agility, coordination, and power. However, the exercise regimen must be individualized to match the female athlete's fitness profile and progression should be gradual in order to minimize overtraining, attrition, and injury rates [5, 29, 30, 32].

### 16.3.5 Different Modes of Treatment for Orthopaedic Injuries Sustained by Adult Females Participating in Certain Sporting Activities

Proper management of a musculoskeletal injury should be done in order for optimal recovery in a timely manner. The following general principles (PRICE) hold when addressing orthopaedic issues: *P*rotection from further harm to the injured extremity; relative *R*est for the affected limb(s) to maintain range of motion (ROM) to minimize stiffness; *I*ce to aid in soft tissue inflammation; even/equal distribution *C*ompression wrap (not to tight) to help with edema; and *E*levation above heart level as much as possible to control swelling. In other words, utilize appropriate first aid measures initially to hopefully halt the progression of the injurious process and reduce painful symptoms. As far as treatment for specific injuries is concerned, tendon strains, muscle "pulls," and ligament sprains, dependent upon the degree, will usually respond to avoidance of provocative maneuvers and first aid treatment initially [31].

Administration of medications may help with pain and speed up recovery. Over-the-counter nonsteroidal anti-inflammatory drugs (NSAID) can be used for inflammation and pain; however, caution needs to be taken as not to remove the body's natural protective mechanism by masking symptoms. Another consideration is the medication's potential toward a delayed healing response due to blunting of the body's natural process of inflammation. Administration of vitamin C has been shown to provide a strong anti-inflammatory effect and aids in the process of bone formation and scar tissue healing. Vitamin C is also an antioxidant, which also aids in the repair of tissues. However, results are contradictory as far as the role of vitamin C in terms of helping with exercise recovery. Similarly, other studies have shown anti-inflammatory as well as anabolic affects of omega-3 fatty acids, particularly seen in fish oil. Like NSAIDs, however, caution needs to be taken when consuming these minerals as not to remove the body's natural inflammatory response and subsequent tissue healing [6].

Along the same line, electrical stimulation devices may also be used, but only as an adjunct to neuromuscular retraining. In terms of orthotics for the feet, the custom molded ones tend to be better than those off the shelf as far as helping to distribute load more evenly inside the shoe [4, 16]. As for bracing of the limbs, use these judiciously as well, since muscles can become deconditioned if braces are worn too frequently, and no studies thus far have shown conclusive evidence that they can prophylactically prevent knee injuries [1, 6, 16]. Moreover, recent studies have found that improper bracing can, in fact, increase the risk of injury because lack of motion in one joint will cause load transfer to be distributed to the adjacent joint. Therefore, caution needs to be taken when using limb braces as protective or preventative measures. Similarly, athletic taping, although helpful in terms of joint stability, tends to take away the protective proprioceptive capability of soft tissues surrounding the joint. In addition, the tape loosens within about 20 min of play, rendering taping completely ineffective after that length of time [4, 6, 16].

Extreme trauma to the limb warrants absolute immediate immobilization plus radiologic studies are definitely needed to quickly rule out displaced fractures. Musculoskeletal insults of this severity require an orthopaedic consultation and could culminate eventually in surgical fixation if the injury is severe enough [5, 29]. As far as nonoperative management of musculoskeletal conditions in the adult female is concerned, as soon as the athlete has a pain free extremity and joint ROM is regained, progression of activities should

be instituted to include therapeutic rehabilitation exercises. The rehab program should encompass conditioning workouts previously outlined to include strength, flexibility, power, speed, and agility. In addition, other elements including coordination, balancing/proprioception should also be integrated as part of the physical training regimen. Furthermore, faulty body biomechanics should be adjusted when engaging in movement/motion to minimize physical stress as part of the kinetic chain adding insult to the already injured limb. Finally, sports specific drills need to be added once prior physical milestones have been mastered. From this point on, functional exercises are emphasized in this phase of recovery in order to return the athlete to her pre-injury physical status and level of play [5, 6, 14, 29, 32].

## 16.4 Future Directions and Concluding Remarks

In summary, the adult female athlete, through the growing years of development into more mature stages in life, has to face not only morphologic changes of the entire musculoskeletal system, but she also has to deal with sex-specific hormonal alterations influencing body composition as well. Prior to the early 1970s, involvement of women in sports was sparse until congress intervened, mandating equality for females participating in academic/athletic competitions. Paralleling the exponential rise in female sporting events, the rate of orthopaedic injuries/conditions also became much more prevalent and exceeding the incidence in males, affecting especially the knee joint and namely the ACL. Different factors, both intrinsic and extrinsic, contribute to the increased occurrence of musculoskeletal injury in athletic women. What appears to be fairly effective in preventing certain orthopaedic conditions in female athletes consists of engaging in physical conditioning programs to institute strength, flexibility, agility, speed, proprioception, coordination, plyometrics, and power. Once musculoskeletal injuries have occurred, however, it is prudent to recognize/diagnose the offending problem early in order to protect the affected athlete from further harm. In addition, initial first-aid type measures can be instituted to help decrease symptoms plus enhance healing and recovery. Physical rehabilitation exercises with individualized gradual progression as tolerated can be added once the athlete responds to these first-line modalities. However, if the injury becomes recalcitrant to conservative management, orthopaedic consultation should be sought for further evaluation and treatment. The ultimate goal for any injured athlete is to provide aid promptly and hasten rapid recovery as much as possible in order to return her to play, whether it is in elite competitions or recreational sports.

## References

1. Arendt E. Orthopaedic issues for active and athletic women. Athletic Wom. 1994;13(2):483–503.
2. Wilmore J, Costill D. Chapter 19: gender issues and the female athlete. Physiology of sports and exercise. Champaign, IL: Human Kinetics Publishers; 1994.
3. Cassels J, Magelssen D. Chapter 18: general physiology the female athlete. Sports medicine for the primary care physician. In: Richard B. Birrer, editor. CRC Press; 1994.
4. Beim G. Sports injuries in women: how to minimize the increased risk of certain conditions. Womens Health. 1999;2(1):27–33.
5. Gill J, Miller S. Chapter 4: female athletes. Sports medicine. Philadelphia, PA: Lippincott Williams & Wilkins; 2006.
6. Nattiv A, Arendt E, Hecht S. The female athlete. Principles and practice of primary care sports medicine. Philadelphia, PA: Lippincott Williams & Wilkins; 2001.
7. Hale RW. Factors important to women engaged in vigorous physical activity. In: Strauss R, editor. Sports medicine. Philadelphia, PA: W.B. Saunders; 1984. p. 250–69.
8. Griffin L, Hannafin J, Indelicato P, Joy E, Kibler W, Lebrun C, Pallay R, Putukian M. Female athlete issues for the team physician: a consensus statement. Med Sci Sports Exerc. 2003.
9. Ahmad C, Clark M, Heilmann N, Schoeb S, Gardner T, Levine W. Effect of gender and maturity on quadriceps-to-hamstring strength ratio and anterior cruciate ligament laxity. Am J Sports Med. 2006;34(3):370–4.
10. Chappell J, Yu B, Kirkendall D, Garrett W. A comparison of knee kinetics between male and female recreational athletes in stop-jump tasks. Am J Orthop Soc Sports Med. 2002;30(2):261–7.
11. Hass C, Schick E, Tillman M, Chow J, Brunt D, Cauraugh J. Knee biomechanics during landings: comparison of pre- and postpubescent females. Med Sci Sports Exerc. 2005;37(1):100–7.

12. Hewett T, Ford K, Myer G. Anterior cruciate ligament injuries in female athletes. Part 2, a meta-analysis of neuromuscular interventions aimed at injury.
13. Keros P, Pećina M, editors. Functional anatomy of the locomotory system [in Croatian]. Zagreb: Naklada Ljevak. Croat Med J. 2007;48(6). www.ncbi.nlm.nih.gov.
14. Gilchrist J. CDC recommendations for preventing exercise-related injuries in women. Womens Health. 2000;3(3):90–2.
15. Hasbrook C. Chapter 15: gender: its relevance to sports medicine. Principles and practice of primary care sports medicine. Lippincott Williams & Wilkins; 2001.
16. Teitz C, Hu S, Arendt E. The female athlete: evaluation and treatment of sports-related problems. J Am Acad Orthop Surg. 1997;5(2):87–96.
17. Bennell KL, Brukner PD. Epidemiology and site specificity of stress fractures. Clin Sports Med. 1997;16:179–96. doi:10.1016/S0278-5919(05)70016-8.
18. Zeni AI, Street CC, Dempsey RL, Staton M. Stress injury to the bone among women athletes. Phys Med Rehabil Clin N Am. 2000;11:929–47.
19. Milner C, Ferber R, Pollard C, Hamill J, Davis I. Biomechanical factors associated with tibial stress fracture in female runners. Med Sci Sports Exerc. 2006;38(2):323–8.
20. Whiting WC, Zernicke RF. Biomechanics of musculoskeletal injury. Champaign, IL: Human Kinetics; 1998.
21. Wojtys EM, Ashton-Miller JA, Huston LJ. A gender-related difference in the contribution of the knee musculature to sagittal-plane shear stiffness in subjects with similar knee laxity. J Bone Joint Surg Am. 2002; 84A:10–6.
22. Ardent EA, Bershadsky B, Agel J. Periodicity of noncontact anterior cruciate ligament injuries during the menstrual cycle. J Gend Specif Med. 2002;5(2):19–26.
23. Huston LJ, Wojtys EM. Neuromuscular performance characteristics in elite female athletes. Am J Sports Med. 1996;24:427–36.
24. Bandholm T, Thorborg K, Andersson E, Larsen T, Toftdahl M, Bencke J, et al. Increased external hip-rotation strength related to reduced dynamic knee control in females: paradox or adaptation? Scan J Med Sci Sports. 2011;21:215–21. doi:10.1111/j.1600-0838.2010.01255.x.
25. Ireland ML, Willson JD, Ballantyne BT, Davis IM. Hip strength in females with and without patello-femoral pain. J Orthop Sports Phys Ther. 2003;33: 671–6.
26. Brent JL, Myer GD, Ford KR, Hewett TE. The effect of gender and age on isokinetic hip abduction. Med Sci Sports Exerc. 2006;38:225.
27. Witvrouw E, Lysens R, Bellemans J, Cambier D, Vanderstraeten G. Intrinsic risk factors for the development of anterior knee pain in an athletic population. A two-year prospective study. Am J Sports Med. 2000;28:480–8.
28. DeHaven KE, Lintner DM. Athletic injuries: comparison by age, sport, and gender. Am J Sports Med. 1986;14:218–24. doi:10.1177/036354658601400307.
29. Locke R, Warren M. How to prevent bone loss in women with hypothalamic amenorrhea. Womens Health. 2000;3(3):82–8.
30. Myer G, Ford K, McLean S, Hewett T. The effects of plyometric versus dynamic stabilization and balance training on lower extremity biomechanics. Am J Sports Med. 2006;34(3):445–55.
31. Rich B. Chapter 57: PRICE: management of injuries. Principles and practice of primary care sports medicine. Lippincott Williams & Wilkins; 2001.
32. Hewett T, Zazulak B, Myer G, Ford K. A review of electromyographic activation levels, timing differences, and increased anterior cruciate ligament injury incidence in female athletes. Br J Sports Med. 2005;39:347–50.

# Prevention and Management of Common Exercise-Related Musculoskeletal Injuries During Pregnancy

17

Mimi Zumwalt and Brittany Dowling

**Abstract**

Pregnancy causes additional alterations to the female athlete's body that she must be aware of. The hormonal, bodily, and biochemical changes that accompany the pregnant state can incite new symptoms and may cause additional avenues for musculoskeletal trauma. Precaution does need to be taken by the pregnant female when participating in certain physical activities. However, in general the female athlete can be very active up until delivery if she's careful to protect the fetus. An already existing injury can be exacerbated by pregnancy, although in most cases symptoms tend to cease shortly after delivery. On the other hand, maintaining an active lifestyle while pregnant helps the mother as well as the fetus to be healthier in the long run.

**Keywords**

Pregnancy • Delivery • Fetus

## 17.1 Learning Objectives

After completion of this chapter, you should have an understanding of:

- The pertinent female anatomy involved in pregnancy and how the surrounding musculoskeletal structures are affected by the gravid uterus and growth of other essential organs
- The accompanying physiological alterations along with other associated systemic changes affecting the pregnant woman's ability to exercise
- The anatomical and structural issues contributing to or detracting from performing different types of exercises while pregnant

M. Zumwalt, M.D. (✉)
Department of Orthopaedic Surgery and Rehabilitation, Texas Tech University Health Sciences Center, 3601 4th Street, Lubbock, TX 79430-9436, USA
e-mail: mimi.zumwalt@ttuhsc.edu

B. Dowling, M.S.
Department of Health, Exercise, and Sports Sciences, Texas Tech University, 1545 Shadowtree Court, Colorado Springs, CO 80921, USA
e-mail: dowlingb86@gmail.com

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_17, 

- Different prevention measures for common musculoskeletal injuries incurred with exercising while being pregnant, to include current recommended standard medical guidelines
- Various modes of treatment for common musculoskeletal injuries which can be exacerbated or sustained by a woman while exercising in the pregnant state

## 17.2 Introduction

Pregnancy is a time of tremendous change for a woman's body, not only involving the anatomical structures but also physiologically. As a matter of fact, these bodily changes persist for the entire duration of pregnancy (generally 9 months) and may continue even beyond childbirth. These morphological, hormonal, and biochemical alterations may incite new symptoms and predispose the pregnant woman to a myriad of injuries involving the musculoskeletal system, or even aggravate previously preexisting orthopaedic conditions. Therefore, the expecting female must try to protect her own body from physical harm, and, at the same time, she also needs to make sure that the fetus she is carrying remains safe from any injury. This does not mean that she must feel completely confined and give up exercising altogether. However, she has to be cognizant of the fact that she cannot and should not train as hard, or intensely, as prior to conception. She must also modify the manner by which she exercises and avoid certain athletic activities, due to the potential increased risk of trauma to herself and/or the fetus. Beyond these specific considerations, a pregnant woman can engage in various forms of physical activity as long as she remains strict in following current standard medical guidelines, takes measures to prevent musculoskeletal insult, and seeks appropriate orthopaedic care if she ends up sustaining injuries from physical training [1–5].

This chapter will focus on different potential musculoskeletal injuries and conditions which can occur and/or become exacerbated with exercising while being pregnant, along with various methods of prevention and management of these more common orthopaedic injuries.

## 17.3 Research Findings and Contemporary Understanding of the Issues

### 17.3.1 The Pertinent Female Anatomy Involved in Pregnancy and How the Surrounding Musculoskeletal Structures Are Affected by Organ Growth

The important anatomical female structures involved in pregnancy include the main organ, the uterus, which is connected to a pair of ovaries by two fallopian tubes (Fig. 17.1).

This female sexual complex is completely contained within the bony pelvis and totally surrounded/supported by soft tissues. Sex differences exist in terms of the female pelvis morphology in general. Although anatomically and structurally similar to the male pelvis, the female pelvis is configured differently. The female pelvis is built larger in all dimensions to accommodate a fetus and allow for childbirth. The basic shape of the female pelvis is wider, rounder, and deeper. Specifically, the pelvis is composed of four main bones: the ilium, the pubis, the ischium, and the sacrum. Two of these large bones join anteriorly at the pubic symphysis in the midline and posteriorly via the two sacroiliac (SI) joints (Fig.17.2).

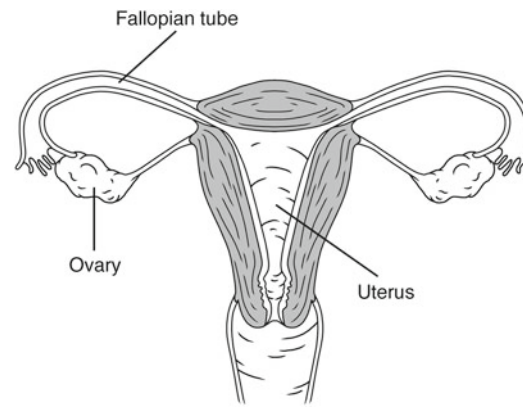



**Fig. 17.1** Female reproductive unit

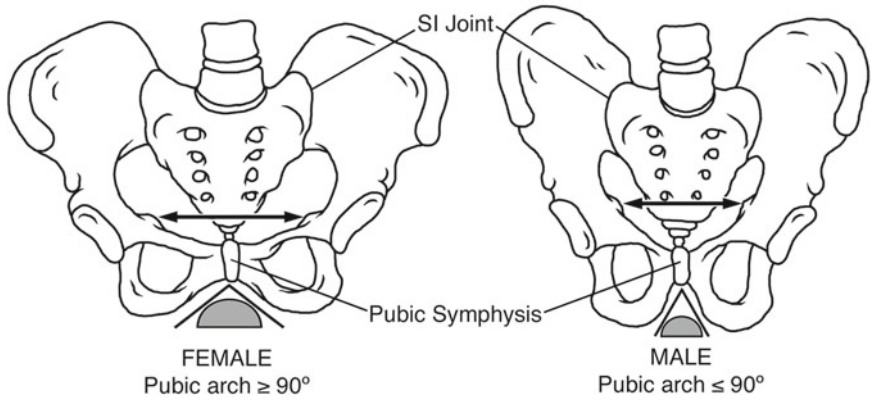



**Fig. 17.2** Pelvis morphology

The formation of the pelvic basin thus consists of bony structures strengthened by ligaments, partially covered by muscles, and entirely lined by fascia. The anterior wall of the pelvis forms from the two pubic bones connected in the midline by the symphysis. Each of the lateral walls is formed by the ilium, obturator foramen, sacrospinous/sacrotuberous ligaments, and obturator internus muscles. The posterior wall is composed of the sacrum, coccyx, and piriformis muscle. Finally, the pelvic floor is made up of the levator ani and coccygeus muscles, along with associated fascial covering. The latter muscle group, however, can only be palpated internally through a rectal exam [2, 6].

In addition, various ligaments which attach from the spine to the pelvis, along with different musculotendinous units originating from the pelvis and inserting onto the lower extremities, also become affected by the gravid uterus, especially over time as the fetus grows in size through the three trimesters. Specifically speaking, the iliolumbar ligaments, the iliopsoas tendon, the rectus abdominis, internal/external obliques, the quadratus lumborum, the erector spinae, and the pelvic walls/floor musculature are the main contributors to the "core" group of supporting structures surrounding the fetus, which inevitably will be affected gradually by fetal and other organ growth within the expanding uterus [2, 6] (Fig. 17.3).

Furthermore, other structures which also play a role in changing a woman's anatomy after conception are the mammary glands. As the breast tissue enlarges in preparation for hormone secretion and milk production, the surrounding skeletal framework, i.e., rib cage, thoracic spine, chest, and upper back, along with their attached muscles, must accommodate accordingly (consequently receiving more load) to support these ever-enlarging breasts [7, 8] (Figs. 17.4 and 17.5).

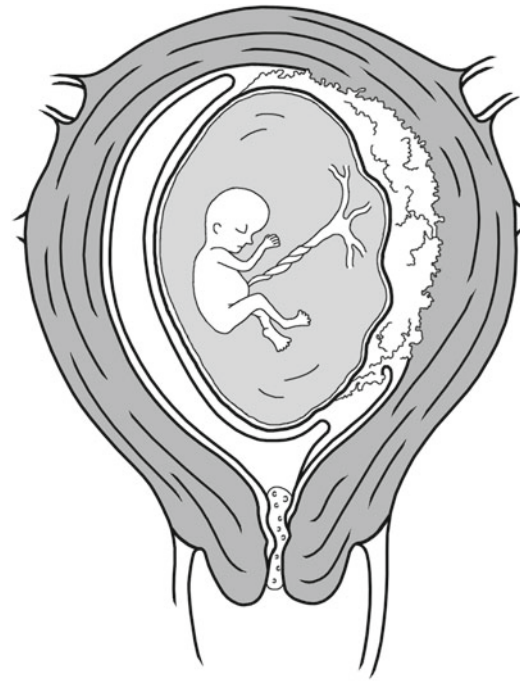


**Fig. 17.3** Gravid uterus

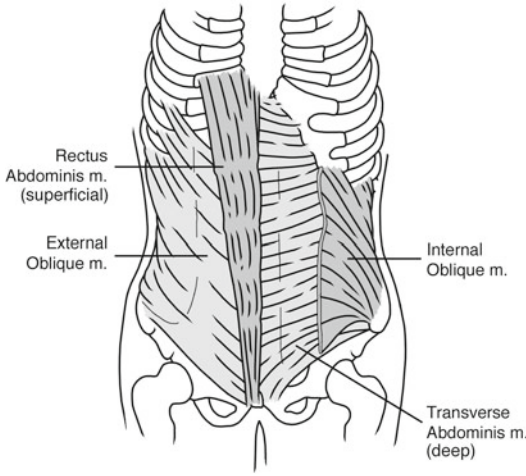



**Fig. 17.4** Truncal musculature—ventral view

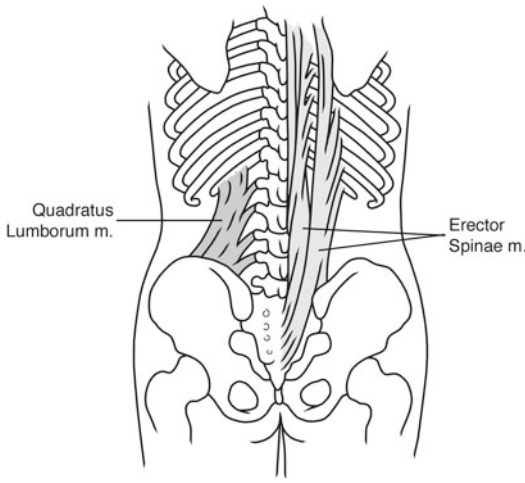



**Fig. 17.5** Truncal musculature—dorsal view

### 17.3.2 The Physiological and Other Associated Systemic Changes Affecting the Pregnant Woman's Ability to Exercise

After conception, the woman's pregnant body not only is affected by structural modifications as previously mentioned but also is influenced by hormonal and biochemical changes. These changes occur in order to provide normal fetal development and parturition (delivery). However, the changes can also cause an array of issues affecting the lower extremity [9, 10].

Shortly after the sperm fertilizes the egg, implantation occurs in the uterus. Once fertilization occurs, the placenta, a unique organ formed to help sustain life for the fetus, which also acts as an endocrine gland, starts to produce numerous hormones. Human chorionic gonadotropin (HCG) is the initial key hormone secreted by the placenta and is used as an indicator of pregnancy when testing the urine or serum. HCG then, in turn, stimulates the formation of relaxin. Relaxin is an estrogen-dependent hormone produced by the corpus luteum (another pregnancy-related structure) that causes softening and stretching of maternal pelvic ligaments and the pubic symphyseal joint. This allows better accommodation of the expanding uterus as well as of delivery. Relaxin has been shown to work by affecting the collagen fibers, increasing water content of soft tissues, and activating fibroblasts in the synthesis of new collagenous tissue [11]. While relaxin is produced in the normal monthly menstrual cycles, levels increase ten times the usual amount during pregnancy [9]. Relaxin serum levels increase during the first trimester and then level off during the second and third trimesters. Other hormones of pregnancy originating from the corpus luteum include but are not limited to progesterone (establishes and sustains the fetus within the uterine cavity), estrogen (responsible for endometrial/uterine lining growth among other functions), and prolactin from both the pituitary and uterus (stimulates the breast milk apparatus among other roles). Additionally, other maternal hormonal alterations affecting a pregnant woman's ability to exercise involve insulin (affects blood glucose levels), cortisol (influences gain in adipose tissue and thinning of the skin leading to easy bruising), thyroid hormone (responsible for increased basal metabolic rate and pulse at rest), and parathyroid hormone (regulates calcium absorption from dietary sources) [8, 10].

The interaction between fetal and maternal tissues causes metabolic changes, which will also influence a pregnant woman's body. Specifically, the normal course of pregnancy induces weight gain on an average of about 10–15 kg (20–40 lbs),

with 50 % from evolving maternal tissues and 50 % from the placental–fetal complex. Two phases encompass the process of fetal–maternal metabolism. The first half of pregnancy is anabolic for the mother and the second half catabolic ("accelerated starvation"). The catabolic state provides an anabolic environment for the fetus, thus enhancing normal fetal growth. However, the catabolic state is extremely taxing in terms of draining energy from the mother [8, 12].

Another significant physiological alteration of pregnancy includes generalized/dependent soft tissue edema from retained extracellular fluid in the periphery of the body. Edema could cause inflammation of musculotendinous structures or entrapment of neural tissues, possibly interfering with certain movements involved with different activities or exercises [2].

### 17.3.3 The Anatomical and Structural Issues Contributing to or Detracting from Performing Different Types of Exercise While Pregnant

The main anatomical issue to consider regarding a woman's pregnant body is the physical change resulting in the forward shift of the center of mass caused by hypertrophy of breast tissue, along with the growth of the fetus inside the mother's uterus. The change in the center of mass moves her body weight more anterior and inferior [1, 3]. To compensate for the increased weight and change in the center of mass, there is an increase in lumbar lordosis, thoracic kyphosis, posterior upper body tilt, and sagittal pelvic tilt [13]. This puts added stress on the SI ligaments and musculature. However, attempts to relocate the center of mass and rebalancing the head over the pelvis in the upright position become less effective over time, due to relaxation and stretching of the local supporting ligaments/connective tissues. This, in turn, contributes to mechanical low back strain (50 % prevalence) along with pain in the pelvic area and SI joints [3, 7, 14]. Widening of the pubic symphysis (up to 1 cm), starting near the end of the first trimester, is associated with tenderness and is aggravated by exercising. These painful symptoms appear to correlate with serum relaxin levels, especially in the first 12 weeks, when serum concentration peaks. Between the fourth and fifth months of gestation, the level of relaxin decreases and then reaches a steady level about half the quantity of its previous peak during the first 3 months. Interestingly, the level of relaxin does not seem to correlate with measured quantity of laxity in joints [2, 15]. As a corollary, the accompanied weight gain also places additional mechanical strain on the lower back, pelvis, and SI areas. Muscular forces increase as much as 100 % for only a 20 % increase in weight, adding to the symptoms of joint discomfort [2]. In fact, forces across the hip and knees during pregnancy may also reach as high as 100 % compared to normal when engaged in impact activities, which potentially could compound more damage to these large weight-bearing joints [14]. Other biomechanical alterations which accompany the pregnant body include increased pressure on the pelvic floor musculature; tightening/shortening of the pectoral, iliopsoas, hamstring, and lumbar extensor muscles; lengthening and weakening of the rectus abdominis and gluteal muscles; and upward flaring of the rib cage. In short, the hormone relaxin contributes to near-global soft tissue elongation within the body, predisposing the pregnant woman to increased risk of overuse injuries with exercising, contributing to tendon strains and ligament sprains [7, 14, 16].

### 17.3.4 Common Musculoskeletal Injuries/Conditions Which Can Be Incurred During and/or Aggravated by Exercise While Being Pregnant, Along with Preventative Measures and Current Standard Medical Guidelines

Although pregnancy induces laxity of tendinous and ligamentous structures secondary to hormonal influence as previously noted, no specific exercise-related injuries have been cited as directly caused

by being pregnant [17]. However, some more common musculoskeletal conditions do exist which can be initiated or exacerbated while exercising in the pregnant state. Typical problems include low back pain; groin pain; pelvic girdle, pubic symphysis, and sacroiliac joint pain; hip/lower limb pain; leg cramps; diastasis recti; meralgia paresthetica; carpal tunnel syndrome; and de Quervain's stenosing tenosynovitis [2, 3, 7, 15].

The incidence of low back pain (LBP) during the course of pregnancy is high, occurring in at least 50–70 % of pregnant women. The rate of LBP increases with increasing age and even more so in those females with a previous history of back pain and prior births. Relaxin plays a major role in contributing to LBP; in fact, the highest serum levels of relaxin during pregnancy have been associated with the greatest amount of pain [18]. Almost one third of afflicted women have to refrain from performing activities of daily living due to their back symptoms. Surprisingly, the amount of weight gain while pregnant does not seem to proportionately correlate with the extent of back problems. As previously noted, as a compensatory mechanism toward the growing fetus, higher shear forces occur on the dorsal/posterior aspect of truncal soft tissue structures which, combined with abnormal pelvic rotation and poor posture in the erect position, predispose certain pregnant women to lumbar discomfort [2, 3, 7]. Similarly, increased upper spinal segment stress also occurs as a result of breast tissue gaining in size and weight, thus consequently causing the shoulders to sag and the neck to ache as well [2, 3].

Variations of LBP experienced by the pregnant woman include pelvic girdle pain (PGP), SI joint dysfunction, and symphysis pubis insufficiency. Their incidence ranges from approximately 4 to 90 %, with 9 to 15 % having severe symptoms. The former entity (PGP) is more generalized and the latter two conditions are more specific, describing posterior weakening and anterior pelvic widening, respectively. Earlier studies have shown that separation of the pubic symphysis from 1 to 2 cm is associated with minimal discomfort, while widening more than two centimeters is associated with a great increase in groin pain. Later studies, however, could not replicate these findings in terms of correlation between the degree of widening and clinically relevant symptoms, although approximately one out of three women does have problems sleeping due to their pelvic pain [3, 15, 16, 19]. Regarding the absolute width of separation, once the symphyseal diastasis approaches 5 cm, however, operative management may have to be considered from a mechanical standpoint since the pelvis has now become an unstable structure, depending on the stage of pregnancy and potential risk to the fetus [13, 15, 16].

Osteitis pubis is another condition that affects the pubic symphyseal joint during pregnancy. This results from resorption and then spontaneous reossification of the adjacent pubic bones, leading to severe, rapid radicular pain from the central pubic and groin area down both thighs which progresses quickly over a few days and is worsened by lower limb movement. Osteitis pubis is usually transient and needs only symptomatic treatment, resolving spontaneously within several days to a few weeks [2]. Most of these conditions do have a common denominator, however, resulting from relaxation of different articulations of the pelvis, which allows greater joint mobility of the pelvic bones, inciting symptoms of pressure and pain from less skeletal support and therefore lack of stability [13, 15, 16].

Lumbosacral/pelvis pathologies occurring during pregnancy may not be solely due to hormonal changes leading to mechanical alterations; however, there may be other preexisting conditions which are exacerbated by changes of pregnancy [16]. For example, lumbar degenerative spondylolisthesis (anterior/posterior vertebral translation) or true spinal discal problems can exist concurrently with LBP as well (Fig. 17.6).

Fortunately, lumbar disc herniation is quite rare, occurring only in one out of 10,000 pregnant women [2, 3]. Likewise, the majority of the above mentioned conditions do tend to resolve on their own after a few weeks postpartum, thus not requiring actual medical management [16]. Lower back pain is usually aggravated by activity and is relieved by sitting or lying down. Symptoms can also be relieved by limiting physical activity, wearing low-heeled shoes, and

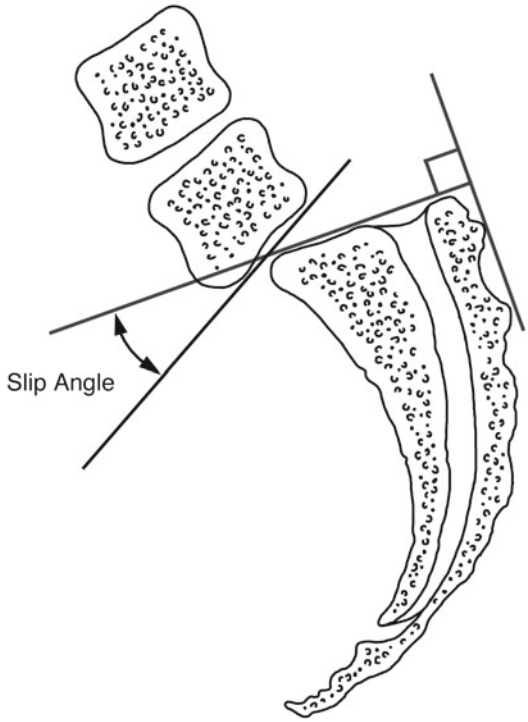



**Fig. 17.6** Spondylolisthesis

applying moist heat compresses. Sometimes, the use of a lumbo–sacroiliac corset/brace can relieve pain by providing external back support.

Diastasis recti is the separation of two bands of the rectus muscle on either side of the linea alba due to stretching of the waistline. This occurs in 75 % of pregnant women as an attempt to increase volume for accommodating fetal growth. This frontal core division of connective tissue causes the abdominal musculature to weaken, thus providing less support dorsally and adding even more to chronic low backache [7, 20]. One study showed that pregnant women who did not exercise had a 90 % incidence of diastasis recti as compared to 12.5 % women who physically train using their transverse abdominis during their workouts [21].

Most musculoskeletal changes affect the lower limbs of pregnant women which can be exacerbated by exercise. This is due to the global effects of relaxin causing ligamentous laxity that can lead to lower extremity dysfunction and pain. The lower extremity joints and limbs must adapt to the change in center of mass and increased body weight by absorbing additional forces. The hip is the most commonly affected area of increased load; it is estimated that 64 % of pregnant women experience hip pain [22, 23]. Hip pain has been linked to transient osteoporosis of the femoral head as well [2, 3]. This idiopathic entity usually occurs during the third trimester, presents with an antalgic gait, and is characterized by pain and limitation in hip range of motion. Pain usually is gradual and exacerbated during weight-bearing activity. The symptoms are similar to pelvic instability and may be incorrectly diagnosed as such. If the osteoporotic hip condition is not recognized, continued weight bearing could cause a femoral neck or intertrochanteric stress fracture. Other skeletal sites, including the sacrum, tibia, rib cage, and spine, may also be afflicted with osteopenia and subsequently increased risk of osteoporotic fractures during pregnancy. The third trimester is particularly an issue for stress fractures because of the large weight gain causing an even greater increase in loading. Treatment needs to include protection of the injured joint to avoid further trauma to the same area. A decrease in activity and in some cases bed rest may need to be prescribed until the painful symptoms are alleviated. Additionally, the use of crutches during weight bearing/ambulation will help to eliminate excessive forces on the hip. If a fracture does indeed occur, surgery will be required to help avoid osteonecrosis, nonunion, and degenerative osteoarthritic changes of the hip [22].

Knee pain occurs in roughly 22 % of pregnant women [22, 24]. As discussed above, a limited range of motion at the hip can be attributed to hip pain. The decreased range of motion at the hip causes a decrease in hip moment and an increase in knee moment. Relaxin causes a decrease in joint stability, which puts more strain on both the hip and knee articulation. Increased strain plus decreased stability allows for greater forces impacted to the acetabular labrum in the hip or cartilage in the knee, predisposing the pregnant woman to a higher chance of injury resulting in mechanical symptoms such as popping and catching. Again, this is dependent on the stage of

pregnancy and degree of loading activity. Finally, recurring patellofemoral pain and ankle sprains are two more orthopaedic conditions commonly found during pregnancy. This should be taken into consideration when planning what sort of exercise program would present minimal risk for the expecting mother to participate in so she won't experience pain and problems [2].

Foot pain is also prevalent in pregnant women, estimated to be at 42 % [22]. Again, this is due to the effects of relaxin on surrounding supportive ligaments. Block et al. found an increase in subtalar and first metatarsophalangeal joint range of motion in the feet of pregnant women [22]. Laxity of the tibialis posterior tendon can cause a 1 cm drop of the talar head, creating a pronated midfoot [23]. The pronated foot and partial longitudinal arch collapse thus creates flattened feet, especially on the stance phase of gait. Studies have found significant changes in foot structure due to pregnancy by measuring locations of anatomical bony landmarks [25]. This change in foot structure due to the effects of relaxin causes an alteration in ambulation. Dynamic gait analysis of shoe wear patterns demonstrates higher forces in the foot while walking during pregnancy [23]. Subsequent adjustment to the change in foot structure and increased loads can cause foot discomfort and pedal pain, among other symptoms [23].

Leg cramps are experienced by about 15–30 % of pregnant women, more often in the latter part of the second and third trimesters, with 75 % happening during nocturnal hours. These extremely strong, fast muscular contractions are severe and very painful, enough to awaken the pregnant woman from deep sleep. Cramps generally occur in the calf muscles and sometimes in the thigh and last from a few seconds to several minutes [26]. The cause of this leg muscle tetany may be due to electrolyte imbalances, primarily insufficient intake of calcium and magnesium. Relaxin has been shown to inhibit calmodulin and calcium influxes into the uterus, allowing for uterine relaxation, and thus prevents premature contractions, causing all muscles in the body to be calcium deficient [27]. The fetal requirement of high calcium levels depletes the quantity of maternal calcium, leading to hypocalcemia. Oral administration of calcium is an effective treatment for muscle cramps. Hammar et al. gave pregnant women 1 g of calcium twice a day for 2 weeks and found a decline in the prevalence and severity of leg cramps [28].

Musculoskeletal conditions occurring in the upper extremity and compression neuropathy from peripheral edema do not tend to be as serious as some of the lower limb conditions because weight bearing is not a factor. However, the quantity of retained fluid can accumulate as much as 6.5–7.5 l a day, causing dependent/pitting edema, especially in the lower limbs [3]. Anatomical issues predisposing the pregnant woman to peripheral nerve injury include certain areas being more prone to excessive pressure and/or existing at superficial locations. Common sites of compressive neural lesions involve the median nerve at the wrist causing carpal tunnel syndrome, the common peroneal nerve at the lateral leg/proximal fibula, and the lateral femoral cutaneous nerve of the anterior hip/thigh. The latter, also known as meralgia paresthetica syndrome, occurs over ten times more often in pregnant women; and the risk is increased even further with associated obesity and diabetes [2, 3].

De Quervain's disease occurs also more commonly during pregnancy on the radial aspect of the wrist. The location of this condition is due to/aggravated by repetitive motions and is influenced by prolactin, relaxin, and progesterone. This overuse type of injury involves inflammation of two tendons of the first dorsal wrist compartment controlling thumb motion: the abductor pollicis longus and the extensor pollicis brevis. Again, these are only temporary conditions, which will resolve spontaneously once the hormonal milieu returns to normal status post delivery [3].

Prevention of trauma is the best management because it is difficult to treat an injury in a pregnant woman. Exercise remains the best preventative medicine modality. Current standard medical guidelines for the prevention of orthopaedic injuries and exercising during the pregnant state stem from the American Congress of Obstetricians and Gynecologists (ACOG). As a preface to recommending a training program, the majority of

studies have found that exercising has a primarily neutral or positive effect on the pregnancy course and its outcome (labor and delivery) [2, 3, 17, 20]. When considering an exercise prescription, a balance must be sought between the possible harmful effects to the mother and fetus and the potential health benefits to both. In terms of duration of physical activity, it is recommended that a woman having an uncomplicated pregnancy engage in moderate exercise sessions for 30–45 min/day up to 7 days a week. This is also supported by the American College of Sports Medicine (ACSM) and Centers for Disease Control and Prevention (CDC) [29–32].

Specific precautions while exercising in the pregnant state include avoidance of visceral shunting of blood flow and therefore prolonged motionless standing (pedal blood pooling and risk of syncope) and excessive supine positioning, especially after the first trimester (to minimize hypotension and reduced heart rate from vena cava compression, thus less blood flow to the fetus). Other issues to consider are exertion at extreme environmental conditions such as excessive heights (risk of altitude sickness), so pregnant women are advised to stay below 6,000–8,000 ft and avoid being submerged under water below 30 ft (chance of decompression sickness and venous air embolism). Additionally, recreational activity involving other players (team sports) tends to have a high potential of collision or hard physical contact, which could harm both the mother and fetus, and therefore should be avoided. These athletic activities include but are not limited to soccer, basketball, and ice hockey. Similarly, the pregnant woman should abstain from athletic endeavors which carry an inherent risk of losing her balance and falling, thus causing abdominal trauma, such as downhill skiing, horseback riding, and gymnastics. Vigorous maneuvers associated with racquet sports (tennis) and impact activities such as jogging/running should be omitted as well, since these could contribute to joint damage from repetitive high loads. Additionally, pregnancy is not an ideal time to begin a vigorous exercise program or to make substantial gains in overall fitness; rather, it is a time to maintain a healthy lifestyle [1–5, 14, 17, 20, 29–31].

Recommended safe and effective physical activity for the pregnant woman includes resistance training with lightweights and multiple repetitions through a dynamic range of motion to enhance muscle tone, thus adding stability and minimizing injury to the already lax ligaments and joints. Lifting techniques and positioning with weights/machines are of paramount importance in terms of safety in order to adjust to morphological changes of the pregnant body. It is also prudent not to engage in repetitive heavy or excessive isometric-type weight lifting to limit the Valsalva maneuver/pressure response in elevating systemic blood pressure excessively while exercising [1, 3, 4, 14, 17, 31]. The exercise regimen should also include some sort of aerobic-type conditioning to maintain cardiorespiratory fitness. Any activity involving movement of large muscle groups in a rhythmic fashion sustained for at least 15 min will be sufficient. The ACSM–CDC guideline for intensity of aerobic exercise is moderate, equating to a brisk walking pace of 3–4 mph (3–5 metabolic equivalents (METs)) and gauged by the revised Borg's rate of perceived exertion (RPE) between 4 and 6, with 1 being the lightest and 10 the hardest. This method is preferred since heart rate is not an accurate predictor of how hard one is working during pregnancy. The following are representative aerobic training modalities which, if followed regularly, will help in accomplishing fitness goals: walking, stationary cycling, hiking, dancing, swimming, rowing, cross-country skiing, skating, rope skipping, and indoor/outdoor group exercise classes (on land or in water). As for flexibility exercises, since the pregnant woman's joints are already lax, she should use slow static stretches and be careful not to overstretch or perform ballistic movements in order to minimize the risk of incurring potential musculoskeletal injury [12, 14, 20, 30, 31].

In short, numerous studies dealing with pregnant women participating in a regular exercise program have demonstrated substantial maternal benefits with minimal fetal risks. Positive effects include improved cardiorespiratory capacity, control of excessive weight gain and fat retention, and better mental/emotional outlook (sense of well-being). Additional beneficial effects of exercising

include lessening of somatic symptoms associated with pregnancy such as insomnia, anxiety, gastrointestinal complaints, leg cramps, pelvis discomfort, and LBP. As long as standard medical guidelines are followed in terms of the "FITT" principle (*f*requency, *i*ntensity, *t*ype/mode, and amount of *t*ime or duration of exercise), working out during pregnancy is safe and effective in terms of maintaining health fitness for both the mother and the fetus [3, 12, 17, 32].

### 17.3.5 Various Modes of Treatment for Common Musculoskeletal Injuries Sustained by Exercising While in the Pregnant State

Trauma during pregnancy is never completely avoidable, even with using preventative measures. After a traumatic episode, treatment of injured pregnant women becomes an issue due to the growing fetus. While there are different modalities to treat the pregnant female's pain and discomfort, protection of the fetus is first and foremost, plus her symptoms may not be relieved completely despite medical management. Modification or cessation of certain activities and exercises may need to take place in order to completely carry to term without undue damage to the mother or fetus [19].

The premier treatment method of pregnancy-related musculoskeletal conditions involves avoidance of certain positions or activities which could exacerbate symptoms. The ultimate goal is to protect the injured area from further harm while enhancing mobility, strength, and endurance. Resting is the first way to treat an injury, though this is not an absolute but a relative requirement. For example, management of LBP, pelvic pain, and SI joint pain requires the pregnant woman to avoid climbing stairs, twisting, bending, and lifting maneuvers. At the same time, she should engage in a physical therapy program concentrating on spinal realignment/stabilization and balancing of the central core muscle group [4, 15, 33–36]. The symptomatic pregnant woman needs to concentrate on maintaining correct posture and utilizing appropriate body mechanics. Specifically, abdominal and hip/gluteal strengthening comprises a major part of the treatment strategy. Exercising the surrounding lumbar and pelvic floor musculature will also help strengthen these muscle groups to support the gravid uterus while adding to the stability of the body. Aquatic exercises are extremely beneficial in offloading joints by allowing her to continue exercising. Exercising also aids in controlling peripheral edema and helps to lower heart rate response as well, adding to the overall positive effects [2, 4, 34]. Furthermore, one study had found that a combination of acupuncture and physiotherapy was more effective than therapy alone in treating pregnancy-related pelvic girdle pain [34]. Another study confirmed that an exercise program to strengthen the abdominal/hamstring muscles plus stretching the iliopsoas and paravertebral muscles was quite successful in decreasing the intensity of LBP and increasing spinal flexibility during the third trimester [37]. A third study demonstrated that physical activity prior to pregnancy reduced the risk of pelvic and LBP while being pregnant [38]. A fourth study observed that a 50 % reduction in disability did indeed occur in pregnant women engaging in a regimen composing of lumbopelvic stabilizing exercises, which continued up to 2 years postpartum [36, 39]. Alternatively, physical therapy programs, along with additional support such as a lumbosacral corset (nonelastic belt worn just proximal to the greater trochanter), can be beneficial in symptomatic management of low back and pelvic girdle pain plus improvement of functional status. However, another review paper found a neutral effect for both treatment modalities as far as treating similar symptoms during pregnancy [4, 15, 19, 35].

As far as other local modalities used in treating musculoskeletal injuries related to pregnancy, most are contraindicated due to potential transfer of heat and/or transcutaneous nerve stimulation to the fetus. These methods include ultrasound, superficial heat, and electrical current. Likewise, a traction apparatus for the spine is controversial

since this mechanical device may place too much pressure on the abdomen from the belt location along with adding too much tension on already lax ligaments [2]. On the other hand, local injections of an anesthetic and steroid mixture, such as for the management of de Quervain's disease, appear to be fairly safe. However, this method also should be utilized judiciously only as an adjunctive treatment measure, along with splinting and occupational rehabilitation exercises. Administration of nonsteroidal anti-inflammatory drugs (NSAIDs) is not recommended during pregnancy due to potential fetal risk [3]. Furthermore, aspirin has been associated with intracranial hemorrhage, while the use of antiprostaglandins may result in the failure in the closure of the ductus arteriosus in the fetus, so these medications should not be utilized while pregnant [40].

## 17.4 Future Directions and Concluding Remarks

In summary, a pregnant woman carries with her an additional internal weight while dealing with morphological and physiological/hormonal alterations of her body. Although pregnancy may be limiting in some aspects, it need not be a condition which severely confines a woman to absolute inactivity. In fact, numerous studies have shown that working out during pregnancy is very beneficial for both the mother and the fetus. Nonetheless, certain musculoskeletal conditions do occur with or become aggravated by exercising while pregnant, but these can be treated symptomatically. Fortunately, many of these incurred problems do tend to resolve spontaneously fairly shortly after delivery, so there is no cause for extreme concern. The pregnant female, however, must take every precaution possible by modifying the manner in which she moves and exercises in order to minimize undue injury to herself and/or the fetus. By doing this, she can still maintain her body in tip-top shape in spite of anatomical and systemic changes to optimize both physical and mental health for her and ultimately benefit the baby to come!

## References


1. Artal R, Sherman C. Exercise during pregnancy: safe and beneficial for most. Phys Sportsmed. 1999;27(8):51–75.
2. Borg-Stein J, Dugan SA, Gruber J. Musculoskeletal aspects of pregnancy. Am J Phys Med Rehabil. 2005;84(3):180–92.
3. Ireland M, Ott S. The effects of pregnancy on the musculoskeletal system. Clin Orthop Relat Res. 2000;372:169–79.
4. Teitz C. Special populations. The Female Athlete, American Academy of Orthopaedic Surgeons AAOS Monograph Series Rosemont, IL: 1996.
5. Treuth M, Butte N, Puyau M. Pregnancy-related changes in physical activity, fitness, and strength. Med Sci Sports Exerc. 2005;37(5):832–7.
6. Agur A, Lee M. Grant's atlas of anatomy. 10th ed. Pennsylvania: Lippincott Williams & Wilkins; 1999.
7. Balogh A. Pilates and pregnancy. RCM Midwives. 2005;8(5):220–2.
8. Berne R, Levy M, Koeppen B, Stanton B, editors. The reproductive glands, Physiology. 5th ed. St Louis, MO: Mosby; 2004. p. 920–78.
9. Zarrow M, Holmstrom EG, Salhanick HA. The concentration of relaxin in the blood serum and other tissues of women during pregnancy. J Clin Endocrinol Metab. 1955;15:22–7.
10. Guyton A, Hall J. Female physiology before pregnancy; and the female hormones. In: Schmitt W, Gruliow R, Faber P, Norwitz A, Shaw P, editors. Textbook of medical physiology. 10th ed. PA: Saunders; 2001. p. 929–43.
11. MacLennan AG. The role of hormone relaxin in human reproduction and pelvic girdle relaxation. Scand J Rhumatol Suppl. 1991;88:7–15.
12. Paisley TS, Joy EA, Price Jr RJ. Exercise during pregnancy: a practical approach. Curr Sports Med Rep. 2003;2(6):325–30.
13. Franklin M, Conner-Kerr T. An analysis of posture and back pain in the first and third trimesters of pregnancy. J Orthop Sports Phys Ther. 1998;28:133–8.
14. Artal R, O'Toole M. Guidelines of the American College of Obstetricians and Gynecologists for exercise during pregnancy and the postpartum period. Br J Sports Med. 2003;37:6–12.
15. Stones W, Vits K. Pelvic girdle pain in pregnancy. BMJ. 2005;331:249–50. doi:10.1136/bmj.331.7511.249.
16. Bastiaanssen JM, de Bie RA, Bastiaenen CH, Essed GG, van den Brandt PA. A historical perspective on pregnancy-related low back and/or pelvic girdle pain. Eur J Obstet Gynecol Reprod Biol. 2005;120:3–14.
17. Clapp J. Exercise through your pregnancy. Champaign, IL: Human Kinetics; 2002.
18. MacLennan AH, Nicolson R, Green RC, Bath M. Serum relaxin and pelvic pain in pregnancy. Lancet. 1986;2:243–5.
19. Stuge B, Hilde G, Vollestad N. Physical therapy for pregnancy-related low back and pelvic pain: a systematic

review. Acta Obstet Gynecol Scand. 2003;82(11): 983–90.
20. Davies G, Wolfe L, Mottola M, MacKinnon C. Joint SOGC/CSEP clinical practice guideline: exercise in pregnancy and the postpartum period. Can J Appl Physiol. 2003;28(3):330–41.
21. Chiarello C, Falzone LA, McCaslin K, Patel MN, Ulery KR. The effects of an exercise program on diastasis recti abdominis in pregnant women. J Womens Health Phys Therap. 2005;29(1):11–6.
22. Ponnapula P, Boberg JS. Lower extremity changes experiences during pregnancy. J Foot Ankle Surg. 2010;49:452–8.
23. Nyska M. Plantar foot pressures in pregnant women. J Med Sci. 1997;33:139–46.
24. Vullo VJ, Richardson JK, Hurvitz EA. Hip knee and foot pain during pregnancy and postpartum period. J Fam Pract. 1996;43:63–8.
25. Alvarez R, Stokes IA, Asprinio DE, Trevino S, Braun T. Dimensional changes of the feet in pregnancy. J Bone Joint Surg Am. 1988;70(A):271–4.
26. Heckman JD, Sassard R. Musculoskeletal considerations in pregnancy. J Bone Joint Surg Am. 1994;76A(11):1720–30.
27. Lou SZ, Chou YL, Chou PH, Lin CJ, Chen UC, Su FC. Sit-to-stand at different periods of pregnancy. Clin Biomech. 2001;16:194–8.
28. Hammar M, Larsson L, Tegler L. Calcium treatment of leg cramps in pregnancy. Effects on clinical symptoms and total serum and ionized serum calcium concentrations. Acta Obstet Gynecol Scand. 1981;60: 345–7.
29. American College of Obstetricians and Gynecologists. Exercise during pregnancy and the postpartum period. Clin Obstet Gynecol. 2003;46(2):496–9.
30. Snyder S, Pendergraph B. Exercise during pregnancy: what do we really know? Am Fam Physician. 2004;69(5):1053, 56.
31. Wolfe L, Davies G. Canadian guidelines for exercise in pregnancy. Clin Obstet Gynecol. 2003;46(2):488–95.
32. Morris S, Johnson N. Exercise during pregnancy: a critical appraisal of the literature. J Reprod Med. 2005;50:181–8.
33. Bastiaenen CH, de Bie RA, Wolters PM, Vlaeyen JW, Bastiaanssen JM, Klabbers AB, Heuts A, van den Brandt PA, Essed GG. Treatment of pregnancy-related low back and/or pelvic girdle pain. Eur J Obstet Gynecol Reprod Biol. 2005;120(1):3–14.
34. Elden H, Ladfors L, Olsen MF, Ostgaard HC, Hagberg H. Effects of acupuncture and stabilizing exercises as adjunct to standard treatment in pregnant women with pelvic girdle pain: randomized single blind controlled trial. BMJ. 2005;330(7494):761. doi:10.1136/bmj.38397.507014.EO.
35. Nilsson-Wikmar L, Holm K, Oijerstedt R, Harms-Ringdahl K. Effect of three different physical therapy treatments on pain and activity in pregnant women with pelvic girdle pain: a randomized clinical trial with 3, 6, and 12 months follow-up postpartum. Spine. 2005;30(8):850–6.
36. Stuge B, Laerum E, Kirkesola G, Vollestad N. The efficacy of a treatment program focusing on specific stabilizing exercises for pelvic girdle pain after pregnancy: a randomized controlled trial. Spine. 2004; 29(4):351–9.
37. Garshasbi A, Faghih Zadeh S. The effect of exercise on the intensity of low back pain in pregnant women. Int J Gynaecol Obstet. 2005;88(3):271–5.
38. Mogren IM. Previous physical activity decreases the risk of low back pain and pelvic pain during pregnancy. Scand J Public Health. 2005;33(4):300–6.
39. Stuge B, Veierod MB, Laerum E, Vollestad N. The efficacy of a treatment program focusing on specific stabilizing exercises for pelvic girdle pain after pregnancy: a two-year follow-up of a randomized clinical trial. Spine. 2004;29(10):E197–203.
40. Moise Jr KJ, Huhta JC, Sharif DS, Ou CN, Kirshin B, Wasserstrum N, Cano L. Indomethacin in the treatment of premature labor. Effects on the fetal ductus arteriosus. N Engl J Med. 1988;319:327–31.

# Prevention and Management of Common Musculoskeletal Injuries in the Aging Female Athlete

18

Mimi Zumwalt and Brittany Dowling

**Abstract**

The onset of menopause causes numerous changes in the female athlete's body. The alteration in hormones, particularly a decrease in estrogen, affects both bone quality AND quantity. This mature life stage herald by the occurrence of menopause is associated with a 3 % increase in bone loss per year. Thus one of the greatest threats to the aging female athlete is decreased bone density with the eventual development of osteoporosis. The reduction in estrogen levels also causes a decline of muscular strength in women. However, with the proper physical training regime, particularly weight bearing plus resistance exercises, bone mass and muscular degeneration can be combated. Aging also exposes the female athlete to certain injuries along with increasing the time to recovery once injured. Prevention of musculoskeletal trauma is the best method to ensure that the aging female athlete will maintain an active lifestyle. Appropriate conditioning programs incorporating strength, flexibility, and mobility, as well as proper nutrition, are crucial in keeping the older female population healthy and active to enhance their life in later years.

**Keywords**

Menopause • Estrogen • 3 % increase in bone loss • Osteoporosis • Weight bearing • Resistance exercises • Strength • Flexibility • Mobility • Nutrition

M. Zumwalt (✉)
Department of Orthopaedic Surgery and Rehabilitation, Texas Tech University Health Sciences Center, 3601 4th Street, Lubbock, TX 79430-9436, USA
e-mail: mimi.zumwalt@ttuhsc.edu

B. Dowling, M.S
Department of Health, Exercise, and Sports Sciences, Texas Tech University, 1545 Shadowtree Court, Colorado Springs 80921, CO, USA
e-mail: dowlingb86@gmail.com

## 18.1 Learning Objectives

After completion of this chapter, you should have an understanding of:

- The hormonal, physiological, and anatomical/musculoskeletal changes in female athletes as they transition from young adulthood into the middle age and beyond

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_18, 

- The differences and similarities between older males and females in terms of their body composition, musculoskeletal components, and athletic performance
- Various musculoskeletal injuries and orthopaedic conditions more common to aging and fairly unique to older female athletes
- Several measures of prevention to keep musculoskeletal injuries in older active females from occurring
- Different methods of treatment for orthopaedic conditions incurred in aging female athletes to include exercise prescription as recommended by various nationally recognized organizations

## 18.2 Introduction

Throughout the lifespan of females, they undergo a multitude of changes to include structural, hormonal, physiological, neurological, and musculoskeletal. In a woman's lifetime, there are two most dramatic stages. One occurs early—pubescence during the teen years, and one happens several decades later—senescence after menopause. It appears that certain aspects of a woman's body come around full circle to the point where she started after birth; she then eventually ends up in a similar state ultimately near the end of life. In particular, the endocrine alterations which affect the quality of bones, making the skeleton so fragile that even the slightest trauma could result in osteoporotic or fragility fractures, and if not addressed promptly and appropriately, could eventually result in dangerous demise. However, it is possible for an older woman to effectively combat some of the negative effects of aging with several positive actions. The primary combatant to aging involves keeping the main muscular components strong in order to protect the skeletal system. The aging female must remain relatively active physically to guard herself from harm. However, this may open her up for incurring potential orthopaedic trauma to her body. Exercise does not necessarily reverse the effects of time, but hopefully will slow it down to the point of allowing an older woman to more effectively cope with the inevitable changes of life.

This chapter will address different issues of biological alterations within a woman's body as she ages, as well as various musculoskeletal/orthopaedic injuries more common in the master female athlete, along with strategies for prevention and management of these acute and chronic traumatic conditions.

## 18.3 Research Findings and Contemporary Understanding of the Issues

### 18.3.1 The Hormonal, Physiological, Anatomical/Musculoskeletal Changes in Female Athletes as They Transition from Young Adulthood into the Middle Age and Beyond

In the past century, the number of elderly adults has increased from 1 to 28 %. In the USA alone, the number of persons 65 years or older was estimated at 39.6 million in 2009. This value translates to 12.9 % of the US population or one in every eight Americans [1]. Advances in health, medicine, and standard of living have greatly contributed to this rise in life expectancy. Most Americans maintain a healthy lifestyle for 85 % of their lifespan; however, the final 15 % of their final years older people are limited significantly with degenerative diseases along with impaired mobility. However, the leading causes of death related to lifestyle can often be preventable. Exercise has been shown to help enhance and even prolong the life cycle. Elderly people are adaptable, thus will respond to endurance and strength training. Endurance training helps to maintain cardiovascular function, enhances exercise capacity, and reduces risk factors for certain diseases. Strength training aids in prevention of loss of muscle mass and strength associated with aging [2].

As mentioned in Chap. 7, after peak bone mass is reached in the mid-twenties, the quantity of bone in the skeleton starts to gradually decline at a rate of 0.3–2 % per year until the fourth decade. After menopause, bone loss is accelerated to over

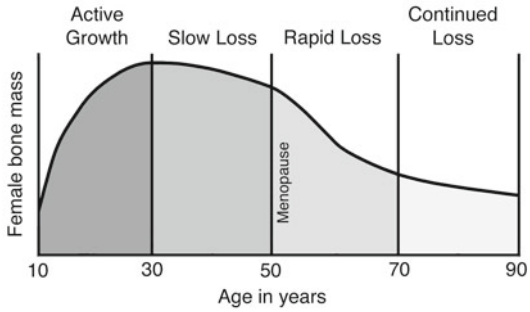



**Fig. 18.1** Rate of bone loss through a women's lifetime

3 % yearly for 5–10 years. This increased rate of bone loss places elderly women at increased risk for osteoporosis [3–8] (Fig. 18.1).

This skeletal manifestation is due to a precipitous drop in the female sex hormone levels, namely, estrogen, which had been previously protective in terms of bone building earlier in life [8].

As a female athlete ages, endurance performance, aerobic capacity, and cardiorespiratory functions all deteriorate, whether due to reduction in relative physical activity or to the natural resultant effects of aging [3, 9, 10]. No matter how well a female athlete takes care of herself, all bodily processes are affected with age. However, the extent of physiological deterioration varies between individuals. Genetic factors have a great influence on the length and quality of life. For example, in females, maximum oxygen uptake tends to decrease approximately 10 % per decade starting in the mid-twenties [11]. However, if training intensity and volume are maintained as compared to their younger counterparts, female master athletes will only lose 1–2 % of their previous aerobic capacity yearly until 50 years of age [3, 4, 10].

Although body weight gradually increases with age from 20 to 70 years old, during 5–10 years prior to menopause, the amount of fat-free mass undergoes a drastic reduction while the percentage of body fat rises (5–10 kg in the mid-thirties to mid-forties). The increase in extent of body fat is greater in more sedentary women [3, 9, 10]. For example, in highly trained female runners around their mid-forties, body fat percentage is about 18 %, as compared to 26 % in their sedentary counterparts. Similarly, women swimmers also remain slimmer, although not as marked as in runners, with body fat staying around 23 %, which is still lower than inactive age-matched females. The average woman's body fat in her youth is approximately 25 %, which rises to over 35 % by age 50. The reasoning behind the increase of body fat is threefold: more dietary intake, less physical activity, and decreased ability to mobilize adipose tissue [10, 12].

In terms of muscular strength, the absolute level required to perform activities of daily living stays the same throughout one's lifetime. On the other hand, the maximal level of muscular strength declines at a steady rate from young adulthood (highest level achieved between age 25 and 35) proceeding onto later years [3, 4, 9, 10, 12].

If physical activity declines or is absent in later years, the loss of muscular strength starts to occur gradually after the third decade, then up to 15 % per decade between the fifth and seventh decades, progressing more quickly in subsequent years, at a rate of up to 30 % per decade [4]. About 25 % of peak force is lost by the time a woman is in her mid-sixties. This age-related loss in strength stems from reduced mass of primarily skeletal muscle (sarcopenia), which in turn affects the basal metabolic rate (10–20 % decline from early adulthood to beyond middle age), and/or restricted physical activity levels [3, 5, 8, 10]. The latter functional disuse condition, hypokinesia, accounts for 50 % of the physiological weakness of muscles and bones [7]. This decline in muscular strength seems to affect the lower extremity to a greater extent than the upper extremity [3]. After peaking in the third to fourth decade, the loss of lean muscle mass is attributed to both a reduction in size and number of muscle fibers [3, 9, 10, 12]. After the fifth decade of life, about 1 % of the total number of muscle fibers are lost per year from atrophic (wasting) changes [10]. Beyond the sixth decade, approximately 15 % of muscle strength is lost every 10 years [5]. In fact, midlife adults not involved in strength training lose about 5–7 lb of muscle every 10 years [9]. In addition, the nervous system's ability to process information and activate the musculoskeletal system is altered, resulting in slower response times with both complex and simple

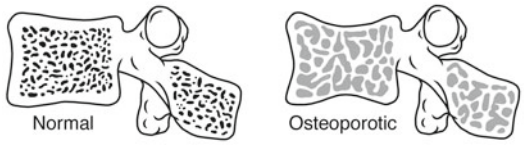



**Fig. 18.2** Normal versus osteoporotic bone

movements [10]. Resistance training can, however, counteract and partially offset ongoing biological muscular weakness by increasing both muscle size and strength [5, 10].

Furthermore, as previously mentioned, older adults who are more sedentary also gain a substantial amount of fat in their subcutaneous tissue, contributing to the natural decline in function associated with aging [10]. As a corollary, the skeletal system in an older female gradually becomes quite frail as well, leading to thinning bones and increased risk of osteoporosis [5–7] (Fig. 18.2).

However, mechanical loading of the skeleton, especially exercising while weight bearing (standing upright) will also help to build bone according to Wolff's law, thus counteracting the continuing bone loss due to inactivity and aging [7, 8, 13]. Even though age-related decline is inevitable, by participating in regular workouts and exercise, older females can fend off some of the inevitable decrements in musculoskeletal strength, power, and endurance [5, 10].

### 18.3.2 The Differences and Similarities Between Older Males and Females in Terms of Their Body Composition, Musculoskeletal Components, and Athletic Performance

The aging process in both men and women is associated with a decline in muscle mass and strength, resulting in a loss of physical function. Sarcopenia (previously described) is a term coined to describe such changes. Sarcopenia results in decreased mobility, increased fatigue, and higher risk for falling [14]. It is estimated that humans lose 20–40 % of skeletal muscle mass and strength from age 20 to 80. A decline in muscle mass is related to weakness, decreased performance, functional impairment, falls, and physical disability [15]. Sarcopenia is also associated with a relative increase in body fat along with a decrease in proprioception, coordination, strength (both muscular and skeletal), power, flexibility (pliability of soft tissues such as joint capsules, articular cartilage, tendons, and ligaments), speed of reaction, and aerobic capacity [4, 10, 16, 17].

Between the second and seventh decades of life, muscular strength decreases about 30 %, along with an approximate 40 % decline in muscle mass. The loss in muscular strength associated with aging is primarily attributed to this great decline of muscle mass. Studies show that with age, there is a decrease in the number of alpha motor neurons, total nerve fibers, and neuromuscular junctions in skeletal muscle [14]. Various studies which have been conducted on both animals and humans find similar results as well. After the age of 60, there is a decrease of up to 50 % of motor neurons in these older individuals as compared to younger populations [18]. Thomlinson and Irving calculated the total number of motor neurons in the spinal cord of the lumbosacral region in 47 subjects ages 13–95. They determined that after age 60, there was a decline of motor neurons, with some subjects having 50 % less than in younger counterparts. Similar results have been found in a study on the biceps brachii where subjects over age 60 have 50 % less motor neurons as compared to those in younger age groups [19, 20].

Evidence shows that with aging there is a loss of motor neurons for both slow and fast twitch muscles, with a greater loss in fast motor units [14]. However, most fibers are reinnervated by other motor neurons, thus affecting a greater loss of muscle fibers within a motor unit. Studies conducted on human and animal subjects have shown that the muscle fibers are reinnervated with different motor neurons [21]. Hashizume and Kanda used a rat model to show a decline in motor neurons as well as a decrease in Type II fibers in the hind limb muscles of older rats [22]. Pettigrew and Gardiner found an increase in Type I muscle

fibers in the plantaris muscle of aged rats [23]. Lochynski, Krutki, and Celichowski also used rats to demonstrate a 22 % decline in Type II motor neurons and a 15 % increase in Type I motor units in the medial gastrocnemius of older rats as compared to younger rats [24]. As aging occurs, there is a greater tendency to lose motor neurons innervating Type II fibers, thus reinnervation tends to be by Type I motor neurons. There is a general shift in the type of muscle from fast twitch to slow twitch as an animal ages [14].

Other studies have found that with age, there is little change in the average cross-sectional area (CSA) of Type I fiber. However, the total muscle CSA innervated by Type I increases with aging, and the total CSA for Type II plus power declines rapidly with age. As the innervation for fast motor units decreases the concentration of muscle work switches over to existing slower motor units [14]. It has also been found that there is an increased density of muscle fibers belonging to a motor neuron with age [25]. A decrease in Type II fibers causes a decline in the muscle's capacity for power (14.). A major contributor to muscle weakness is the decrease in CSA of active muscle tissue. Sugiura and Kanda found as rats increase in age, there is a subsequent decline in CSA and Type II muscles fibers [21]. They also found that with aging, there was a decrease in tension produced in the medial gastrocnemius of the older rats. As a corollary, in humans, the decline in power can be attributed to the loss of Type II fibers, which is reflected in actions such as rising from a chair, climbing stairs, or regaining balance after a gait disturbance [14]. Kostka found velocity at maximal power decreased by roughly 18 % between ages 20–29 and 50–59, with a further 20 % decline in subjects aged 60–69 and 80–89 [26].

Strength gains of more than 100 % can be made in men (60–72 years of age) after lifting weights for about 3 months. Similar studies in both males and females demonstrate that those older than 90 or even 100 years of age participating in strength training could also become physically stronger (~125 %), with a concomitant increase in lean muscle mass (10 %) [16]. One study has shown that an 8-week resistance program in men and women ranging in age between 89 and 92 years old effected an increase in quadriceps strength of 175 % [3, 17]. Likewise, a similar study in women ages 87–96 years old demonstrated an increase of strength three times over baseline values, along with a 10 % gain in muscle size [9]. This rise in muscular strength has been shown to translate into enhancing physical function, decreasing limitations, and increasing mobility, such as speed of gait and stair climbing [13, 16]. Although relative strength gains are comparable for men and women engaged in the same sort of conditioning program, the absolute rise in females is only 50 % that of males [3, 16]. Muscular weakness also occurs earlier in females; thus it is very important to implement and continue a regular resistance training regimen throughout a woman's life [3].

Older adults can make great gains in their capacity to generate force by participating in a regular weight-lifting program. In fact, even as late as the eighth decade of life, intensive muscular training can minimize or even reverse the age-related effects of ongoing weakness [4, 5, 16]. However, improvements made in muscular strength far exceed the enhancement in the quantity of lean muscle mass, meaning that neural activation is responsible for the strength increase [16]. The exception lies in the quality of muscular integrity in men and the ability of their skeletal muscle to retain the capacity for endurance training. Despite the loss of muscle mass over time in males, the remaining muscle still retains 85–90 % of the enzymatic activities required for aerobic type sports [10]. Unfortunately, the amount of strength gains needed to effect a meaningful change in athletic performance is still unknown. What has been shown, however, is the fact that muscular strength appears to improve physical function qualitatively and partially offset age-related bone loss [7, 13, 16]. Therefore, having more muscle mass tends to positively affect bone mineral density in later years [7].

Physical and physiological aspects in the body such as range of motion, coordination, reaction time, and cardiorespiratory fitness can also be improved with exercises specifically aimed at enhancing these parameters. For example, men and women in their sixties and early seventies

can improve their aerobic capacity by over 20 % after engaging in 9 months of endurance training [16]. Similarly, another study found that older subjects 65–90 years of age can also effectively increase flexibility of several large joints after participation in a 12-week program of dancing plus stretching exercises [17].

As one ages, the decrease in muscle mass is accompanied by an increase in adipose tissue [18]. The percentage of fat in women on average changes from about 35 % at 17 years old to approximately 39 % at 60 years of age [2]. However, this change is highly variable and is much less evident in active females. There are a few possible mechanisms that can contribute to this alteration in soft tissue composition. It is thought that due to the loss of muscle mass and strength, there is a subsequent decrease in $VO_{2max}$, which affects the physical activity levels in the elderly. This decline in activity will cause a decrease in daily energy expenditure, which in turn causes an increase in adipose tissue in visceral organs and muscle tissue, as well as total body fat [27]. An increase of fat content in muscles that occurs with aging is associated with a decrease in muscle strength [28]. The age-related increase in muscle weakness translates to a decrease of almost 50 % in isometric knee extension torque strength from age 30 to 80. Even when correcting for decreased muscle mass, there is a decrease in peak torque, indicating a decline in quality of the muscle or reduction in the efficiency of muscle strength with increase in age [18].

As aging occurs, in spite of being able to keep the percentage of body fat low, athletic performance of events comprising endurance plus strength in both males and females inevitably experiences a decline at about 1–2 % per year after the third decade. A study of over 500 subjects (men and women) between the ages of 30 and 70 years old shows that maximal running velocity gradually decreases approximately 8.5 % per decade which is independent of distance. Similarly for cycling performance, speed for both male and female cyclists between 20 and 65 years of age undergoes a gradual drop of about 0.7 % per year. There are some exceptions in sports dependent on athletic skill for successful completion such as golf, equestrian and swimming performance, which continues to improve after years of experience, along with strength and endurance. Thus, the former two types of athletes max out in the third decade, but master swimmers may peak and perform their best as late as 45–50 years of age [3, 10]. In contrast, gymnasts usually peak very early in their teens as far as athletic performance, since flexibility is of paramount importance for this type of sport which tends to decline over time [3].

With advancing age, bone loss causes a decrease in bone density and tensile strength, thus resulting in osteoporosis [2]. Osteoporosis increases the risk of bone fracture. Skeletal bone loss is a serious problem in the aging population, affecting women earlier and more so than men. Osteoporotic changes affect both males and females. However, women are affected at a younger age (females start to lose bone mass at age 30 versus males at age 50), while fewer men (only 20 % compared to 80 % of women) are affected; and even then, men tend to experience a lesser degree of total bone loss than women. After the fourth decade, males lose bone mass at a rate of 0.5–0.75 % per year, while the bone density decline in women is twice that rate—1.5–2 % per year, increasing to at least 3 % per year after menopause. It is not until after the sixth decade that the rate of bone loss in men begins to parallel that of women [3, 5, 6, 8, 12, 17, 29]. Postmenopausal females lose bone density more rapidly in the vertebrae, pelvis, and distal radius [30]. After menopause there is less cortical bone loss in the long bones [30]. Riggs et al. conducted a cross-sectional study of women aged 20–97 years and found large decreases in lumbar spine bone density. The decrease in density was due to loss of vertebral trabecular bone (spongy bone with higher surface area, but less dense) starting in the third decade, whereas cortical bone loss did not begin until middle age [31]. Another sign of normal aging in women is an increase in the cross-sectional area of the femoral neck in the hip and distal radius of the wrist due to formation of trabecular bone in those areas [30].

The age-related changes in bone density are associated with an increased risk of osteoporotic

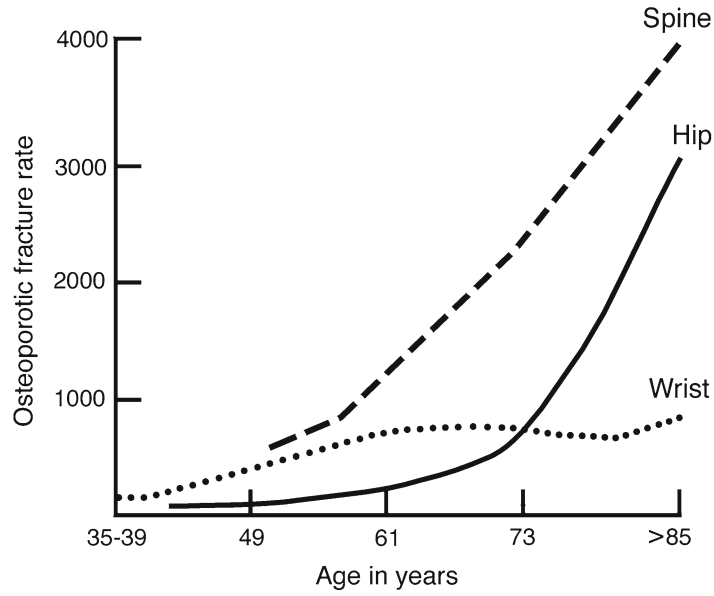



**Fig. 18.3** Osteoporotic fracture rate verses age

fractures in women. According to the World Health Organization (WHO), in the USA approximately 15 % of postmenopausal Caucasian women and 30–35 % of females older than 65 are affected by osteoporosis [6, 32]. Studies have shown that the incidence of Colles' fracture (distal radius) increases in postmenopausal women [30]. Epidemiology also demonstrates that, after the fifth decade of life, about 40–60 % of Caucasian females and 13 % of Caucasian males will suffer one or more clinically significant osteoporotic-related fractures in their lifetime, with 7 % of them experiencing permanent disability [6, 7, 29, 32]. Nine out of ten elderly individuals who sustain a hip fracture are 70 years of age or older [5] (Fig. 18.3). Alarmingly, if an osteoporotic fracture occurs between the ages of 20 and 50, then the risk for another fragility fracture increases to almost 75 % [33].

However, it has been shown that men and women older than 65 who are involved in 10–12 weeks of physical activity such as T'ai Chi (Chinese exercise program consisting of repetitive, rhythmic body movements) or other low-intensity workouts can enhance athletic performance, improve physical balance, and decrease the chance of falling [5, 16] (Fig. 18.4). Another study has demonstrated that this martial arts form, if practiced for at least a few months, can be very effective in reducing the risk of falls by almost 50 % [6, 7].

**Fig. 18.4** T'ai Chi

### 18.3.3 Various Musculoskeletal Injuries and Orthopaedic Conditions More Common to Aging and Fairly Unique in Older Female Athletes

Because the average life expectancy has increased by more than 30 years within the past century, elderly people, especially women, currently are more involved in all sorts of athletic endeavors. Along with this increased participation in physical activity has surfaced different types of musculoskeletal issues, some of which are due to the hormonal changes associated with the female sex, while others occur as a result of the natural aging process [4, 9].

The collagen in ligaments and tendons gradually become less elastic with aging due to loss of water content among other ultrastructural alterations, contributing to increased stiffness. As a result, this type of connective tissue is less able to adapt to mechanical "wear and tear," making it more susceptible to injury along with having less capability for healing. A similar phenomenon happens in cartilaginous tissue, such as the menisci within the knee joint or articular cartilage surrounding bone ends. These connective tissues are unable to distribute force efficiently over time, especially when subjected to repetitive high impact loading. The resultant cumulative microtrauma has an additive effect of joint destruction and cartilaginous degeneration, leading to progressive osteoarthritis and resultant disability. This type of mechanical breakdown is more marked in large weight-bearing joints such as the hip and knee. In fact, middle-aged athletes participating in intense physical loads are more than eight times as likely to develop degenerative arthrosis of their hips. The effects of arthritis are accelerated and exacerbated by previous episodes of injury and/or surgical removal of certain intra-articular structures, such as anterior cruciate ligament tear or partial meniscectomy in the knee, respectively, resulting in posttraumatic arthropathy. Fortunately, unlike the aforementioned soft tissues where one must succumb to natural progressive deterioration, the physiological decline in muscle function due to sarcopenia can be counteracted somewhat by engaging in an intense resistance training program. This is possible since muscular contractility is not influenced to the same extent as loss of muscle mass with aging, enabling maintenance of muscular strength through exercising [14].

As discussed in Chaps. 15–17, the most noted orthopaedic injury occurring in sports participants is the overuse type of microtrauma, such as tendinosis/bursitis/tendonitis affecting the shoulder (rotator cuff disorder/impingement syndrome) and elbow (lateral and medial epicondylitis/tendinosis), and foot (Achilles tendonitis). This type of condition, resulting from chronic, repetitive trauma, accounts for 70 % of veteran athletes beyond the sixth decade versus only ~40 % of younger athletes in their twenties. In addition, 20 % of these overuse injuries can last much longer in older athletes (up to 2 years), affecting their ability to train and/or compete in sports [4].

Another frequent injury which tends to occur in older athletes is muscular strain even with only moderate exercise, an example being performing an eccentric muscle contraction. This is can be related to the fact that the musculotendinous unit is not as flexible as in younger age groups, when much more force is needed to inflict trauma [17]. However, the musculotendinous junction can be affected in a similar manner when compared to other types of connective tissues in terms of relative inflexibility; thus, it is important to also incorporate a stretching routine into the workout regimen in order to decrease risk of injury [4, 17].

Kyphosis, known in layman's terms as "dowager's hump," is a sagittal plane deformity which is characterized by excessive forward spinal flexion, generally in the thoracic region. As opposed to an orthopaedic injury, this is a naturally occurring condition that tends to be especially prominent in women of advancing age. Specifically, postmenopausal women are at greater risk for this type of spinal deformity because of the association between kyphosis and osteoporosis. Supporting evidence was determined by Granito et al., who showed that there is a higher degree of kyphosis in osteoporotic elderly women. They also found a negative correlation between BMD and degree of kyphotic deformity. On the

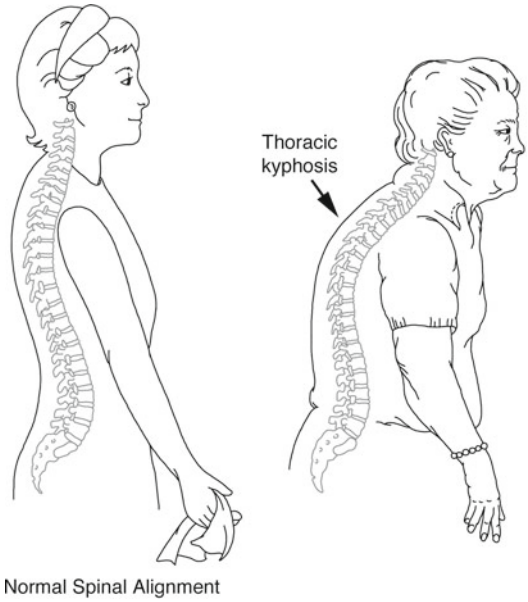



**Fig. 18.5** "Dowager's hump"

other hand, it was noted that women who participate in a regular exercise regime have lower incidences of thoracic kyphosis [34] (Fig. 18.5).

The most common joint disorder in the aging population is osteoarthritis (OA), also known as osteoarthrosis or degenerative joint disease (DJD). A common misconception is that OA is an arthritic disease. On the contrary, because it is not manifested as an inflammatory condition, it is considered more of a degenerative entity. Osteoarthritis begins with the softening of articular cartilage due to a decrease in matrix proteoglycan content. The cartilage thins out, becoming rougher with ulceration, pitting, and fissuring. Continued degeneration is manifested by subchondral bone necrosis and osteophyte formation at the joint margin. The severity of OA is generally classified by the degree of joint space narrowing, osteophyte formation, sclerosis, and bony deformity [35]. The initial presentation of OA varies at different joint sites and generally is first manifested at the metatarsophalangeal joints, followed by the wrist and spine, next in the interphalangeal articulation, carpometacarpal joints, then in tibiofemoral, and lastly seen in the hip joint [6, 7, 29, 35].

The most prevalent type of musculoskeletal trauma occurring in the geriatric female is a fragility fracture due to osteoporosis from excessive bone thinning. The amount of bone loss can be quantified by obtaining a bone density study, with DEXA being the current gold standard. Alarmingly, once osteoporosis sets in, even minimal amounts of force inherent in activities of daily living such as bending over and lifting objects can subject osteopenic bone to potentially injurious loads [6, 7, 29].

Osteoporotic bone is more porous and thus weaker structurally, which can increase the likelihood for a fragility fracture. Compared to younger people, the femur of an older person is only half as strong and has merely a third of the energy absorbing capability before failure [6]. Dynamic models have found that falls create peak hip trochanteric forces ranging from 2.9 to 9.99 kN, which are sufficient to cause a fracture even in healthy bone [36]. Over 90 % of hip fractures occur in older adults beyond the seventh decade [5]. Osteoporosis is a contributing factor to hip fractures; however, other sources need to be considered such as bone quality, muscular strength, soft tissue characteristics, and neuromuscular coordination. Hip fracture generally occurs due to direct impact such as striking the ground after a fall or during car accidents. While rare in younger populations, proximal hip fractures, usually incurred from more violent forces, increase in frequency with age. In fact, 90 % can occur just by falling from only a standing height [35, 36].

More recent longitudinal studies have found a decrease in hip fractures for both men and women from 1990 to 2006, but the factors for this decline are largely unknown [37]. However, women are still three times more likely to suffer from a hip fracture than men. Interestingly, hip fracture rates are lower in people with higher body mass index (BMI weight/height$^2$) [35]. Along the same line, vertebral fractures become more of a concern in the elderly population as well. If an older woman has a height loss of more than 2 in., then this should raise the suspicion for one or more spinal compression fractures resulting from osteoporotic bone [6].

Similarly, wrist fractures herald the early onset of osteoporosis and tend to occur much more often in women between the ages of 50 and 65.

If they occur after this age, then the risk of other types of osteoporotic fracture tends to increase in the future [29]. Fortunately, the incidence of acute orthopaedic trauma in the master athlete is much lower. Only about 1 % of those athletic women hospitalized for sports-related injuries are over 55 years of age [4]. However, the development of OA and site affected varies between each individual based on genetics, joint structure, patterns of mechanical loading, and injury [37].

### 18.3.4 Several Measures of Prevention to Keep Musculoskeletal Injuries in Older Active Females from Occurring

By the time the female reaches adulthood, the physical condition of her body is a by-product of prior years of living. Lifestyle and activity choices affect the body's condition in later years. Participation in athletic activities has positive as well as negative consequences in terms of affecting overall physical body condition. While aging is inevitable, the extent it affects the body is highly regulated by the ability to maintain and preserve muscle mass and bone density throughout one's life [3, 5].

Numerous studies have proven that resistance training exercises have benefits of increasing muscle/bone mass, muscle/bone strength, joint flexibility, balance, and basal metabolic rate [38]. On the other hand, participation in endurance training maximizes cardiorespiratory function [4–6, 16]. Any type of weight-bearing workout, such as dancing, playing volleyball, basketball, walking, jogging, and stair climbing, are beneficial for building muscle and bone as well as cardiorespiratory fitness [5–8]. Other moderate but lower impact exercises, including calisthenics, weight-supported swimming or water aerobics, bicycling, and T'ai Chi, also help in enhancing physical fitness while protecting problematic joints [3, 5, 7, 8] (Fig. 18.4).

However, certain sporting activities should be avoided by older women athletes. These include skiing and rock climbing, as these sports place the body at higher risk of falling, thus causing damage to the already thinning bones and stiffer connective tissues [17]. Similarly, avoidance of contact team sports involving potential collision with stationary objects or opponents is also prudent and well advised [3].

### 18.3.5 Different Methods of Treatment for Orthopaedic Conditions Incurred in Aging Female Athletes, to Include Exercise Prescription as Recommended by Various Nationally Recognized Organizations

Injury is not completely avoidable in the female athlete, no matter what age she may be. Once the master female athlete has suffered a musculoskeletal injury, the first step is prompt initiation of PRICE first aid measures: Protect and prevent the damaged area from further harm, Relative rest for the involved limb while maintaining range of motion to counteract stiffness, apply Ice intermittently to the injured extremity to help with inflammation, use Compression to minimize edema, and Elevate above the heart to aid in swelling. These primary first-line measures are implemented to hopefully alleviate painful symptoms [17, 39]. Administration of medication such as mild analgesics and nonsteroidal anti-inflammatory drugs (NSAIDS) can be used for inflammation and pain. Sports supplements have not been shown conclusively to improve joint condition or enhance athletic performance. In fact, the associated side effects of certain supplements to internal organs far outweighs the touted benefits of any supplement [4]. Oral consumption of glucosamine and chondroitin has been widely debated on its benefits in increasing cartilage components within joints. Numerous studies have shown that a 1,500 mg daily dose of glucosamine sulfate significantly reduces symptoms of osteoarthritis in certain joints of the lower limbs by reducing

pain and aiding in functional improvement [40]. However, other studies have shown that glucosamine provides no clinical effect on pain or function [41].

Medications can be administered by a physician for the prevention and treatment of osteoporosis in older women, which helps to slow the rate of bone breakdown. Controversy centers on whether treatment with calcium and vitamin D for bone building is an effective modality in combating osteoporosis. A study conducted in England on over 5,300 men and women over age 70 found that daily supplementation of calcium, vitamin D, or a combination of both did not significantly affect the fracture rate between groups [42]. On the other hand, Meunier studied a group of French women living in a nursing home setting who were given a daily dose of vitamin D and calcium for 12 months. These researchers found a reduction in the number of hip and non-vertebral fractures by 23 % and, by 18 months, increased bone density in the femoral neck of those taking calcium and vitamin D [43]. While there is still debate on the effectiveness of vitamin D and calcium supplementation in elderly women in terms of bone density, there has been no suggestion that it will decrease bone density. Fish oil supplements have been shown to provide anti-inflammatory effects on soft tissues. A study conducted on 45 women, median age 64 years old, found fish oil supplementation combined with a strength-training regime produced greater improvements in muscle strength and functional capacity compared to strength training alone [44].

Once pain from an injury has subsided, an intensive rehabilitation program should be incorporated, along with an exercise regimen designed to maximize recovery and return to pre-injury status [7, 16, 17]. A structured, balanced workout program should include the following elements: resistive training, cardiovascular endurance activity, flexibility, and balance. Various exercise programs are endorsed by different national organizations, including the American Heart Association (AHA), American College of Sports Medicine (ACSM), and the American Academy of Orthopaedic Surgeons (AAOS) [16]. Specifically speaking, the exercise prescription should involve the following three elements for each workout session: FIT(T)—frequency, intensity, time or duration, and type of physical activity. The ACSM and Center for Disease Control and Prevention (CDC) recommend 20–60 min of moderately intense activity 3–7 days per week [5, 7, 8, 11]. The mode of exercising is dependent on a woman's most current physical fitness level and/or associated musculoskeletal problems. In older women with healthy bones, running in place and other weight-bearing type of mechanical loading can be performed with attention paid toward correct form and technique [8]. However, for those who are frail or females who are already inflicted with arthritic joints, low or minimal impact activities should be chosen, such as exercises done in a pool and bicycling in a sitting or recumbent position [3, 5, 7, 8]. In contrast, to benefit osteoporotic bone, workouts should be done in the standing or erect posture (climbing stairs or walking) to best load the skeleton, especially the spine to optimize bone building [5, 7, 8]. To maximize aerobic fitness, the target heart rate range should be 40–80 % of maximal heart rate of 220 while exercising [5, 8] (see Table 18.1).

**Table 18.1** Target heart rate (THR) ranges

| Age | THR (bpm) |
|---|---|
| 50 years old | 102–136 |
| 60 years old | 96–128 |
| 70 years old | 90–120 |
| 80 years old | 84–112 |

*bpm* = beats per min

In addition to aerobic conditioning, resistance training needs to be incorporated into the workout routine as well to minimize loss of muscle strength and mass, stabilize joints, and maintain bone density [1, 5, 7, 17]. The effect of exercise on bone mass is quite specific in terms of skeletal building; therefore, a training program needs to include both lower and upper extremities [7, 8]. When the older female athlete first starts the

training program, she should begin with mild resistance/light weights (10–12 repetitions for 1–2 sets), then gradually increase the amount of weight lifted as tolerated. She can then work up to 12–15 reps for 2–3 sets, 3–4 sessions per week, for all major muscle groups, concentrating on trunk extensors and the lower extremity muscles to help with body stability and dynamic balance. To ensure appropriate body alignment, especially on the structural type of exercises using multiple muscle groups, plus to direct force through the hip and spine, she can make use of such exercises as free standing squats, dead lifts, and lunges [8]. Additionally, neighboring joints and surrounding musculature close to the injured one(s) should also be included in the rehabilitation phase [17].

Balance and stability training need to also be a part of the exercise regime. Stretching (several short sessions per day are better than one long episode) has been shown to maintain flexibility, balance, and proprioception [5, 8, 17]. Static, passive, and active-assisted stretches for major musculotendinous units should be done on most days of the week. Burke et al. studied 50 women age 65 years or older and divided them into three groups: stretching, strengthening, and control. They found that after 8 weeks, both the stretching and strengthening groups had improved posture as compared to the control group [45]. As for balance and stability training, the following drills have been found to be effective: getting up from a chair without arm usage, alternating one-legged stance, walking backward, and negotiating various obstacles set up on a course. In addition, physio-balls can be used for strengthening core muscles, promoting a neutral spine, and improving postural control as well [8].

A regular exercise program for the elderly woman will promote mobility while decreasing falls, which have been shown to be the sixth leading cause of death [7]. In effect, exercising will maximize residual function and even reduce biological age by as much as two decades [3]. If all conservative management strategies fail to heal the injured extremity, radiographs should be ordered and orthopaedic consultation should be sought since invasive procedures may be warranted at this point [4, 17].

## 18.4 Future Directions and Concluding Remarks

As the active female proceeds from childhood through adolescence into adulthood, she must constantly try to slow down her biological clock as far as the body is concerned in general and the musculoskeletal system in particular. The old adage of "use it or lose it" applies when speaking about maintaining physical fitness in order to continue participating in different athletic activities. However, as time passes, both the muscular and skeletal systems start to gradually decline in terms of strength and integrity. Furthermore, higher risks of potential injuries exist to challenge the master female athlete as well as slower course of recovery after sustaining traumatic episodes. Ideally, the best method of treatment for orthopaedic trauma is of course prevention but certain situations are beyond a woman's control. In that case, measures to protect the injured extremity are of prime importance so she can rehabilitate and physically recover in a timely fashion. The recovery course might last several days to a couple of weeks but may take as long as a few months depending on the extent of injury. In addition, a regular workout program should be instituted, targeting the affected limb but also incorporating resistance training plus aerobic endurance exercises, along with balance and flexibility elements. This way, the aging female can regain strength, function, and mobility to hopefully return to competitive sports or any other athletic endeavor of her choosing.

## References

1. Department of Health and Human Services, Administration of Aging. Aging statistics. A profile of older American; 2011. http://www.aoa.gov/AoARoot/Aging_Statistics/index.aspx.
2. Brooks GA, Fahey TD, Baldwin KM. Exercise physiology: human bioenergetics and its applications. 4th ed. New York, NY: McGraw-Hill; 2005.
3. Shephard, Roy J. Encyclopedia of sports medicine and science: aging and exercise. In: Fahey TD, editor. 1998;1–7. http://www.sportsci.org/encyc/aginginex/agingex.html.

4. Chen AL, Mears SC, Hawkins RJ. Orthopaedic care of the aging athlete. J Am Acad Orthop Surg. 2005;13(6):407–15.
5. Curl WW. Aging and exercise: are they compatible in women? Clin Orthop Relat Res. 2000;372:151–8.
6. Ireland ML. Natton A. Saunden WB. The female athlete osteoporosis. Philadelphia, PA. 2002;249–58.
7. Lindsey C. Fighting frailty in the elderly. Bromechanism 2002;20–29. www.biomech.com.
8. Sorace P. Osteoporosis and exercise. ACSM's Certified News. 2002;12(4):6–7.
9. Kilibwa M. Nutrition and health for women at midlife: recommended approaches for clinicians. J Womens Health Prim Care.2006; 14–26
10. Wilmore JH, Costill DL. Aging and the older athlete. In: Physiology of sport and exercise. Champaign, IL: Human Kinetics; 1994. p. 424–41.
11. Zasadil M, Douglas PS. The master female athlete. Med Sci Sports Exerc. 2005;37(8):1444–7.
12. Solomon EP, Davis WP. Human Anatomy & Physiology. In: Saunden WB, 3rd editor. Philadelphia, PA; 1983.
13. Rejeski JW, Brawley LR. Functional health: innovations in research on physical activity with older adults. Med Sci Sports Exerc. 2006;1:93–9.
14. Lang T, Streeper T, Cawthon P, Baldwin K, Taaffe DR, Harris TB. Sarcopenia: etiology, clinical consequences, intervention, and assessment. Osteoporos Int. 2010;21:543–59. doi:10.1007/s00198-009-1059-y.
15. Dalal M, Ferrucci L, Sun K, Beck J, Fried LP, Semba RD. Elevated serum advanced glycation end products and poor grip strength in older community-dwelling women. Age Ageing. 2009;38:283–9.
16. Brown M. Rose S. The effects of against execise on skeletal muscle clinical considerations. Mech Age Develop. 1992;63:69–72.
17. Leach RE. Orthopaedic surgery essentials sports medicine. In: Onteony A. schepers, Brain D. Buscone, editors. Philadelphia, PA: Lippincott; 2006.
18. Greenlund LJS, Nair KS. Sarcopenia – consequences, mechanisms, and potential therapies. Mech Ageing Dev. 2003;124:287–99.
19. Tomlinson BE, Irving D. The number of limb motor neurons in the human lumbosacral cord throughout life. J Neurol Sci. 1977;34:213–9.
20. Brown WF, Strong MJ, Snow R. Methods for estimating numbers of motor units in biceps-brachialis muscles and losses of motor units with aging. Muscle Nerve. 1988;11:423–32.
21. Sugiura M, Kanda K. Progress of age-related changes in properties of motor units in the gastrocnemius muscle of rats. J Neurophysiol. 2004;92(3):1357–65. doi:10.1152/jn.00947.2003.
22. Hashizume K, Kanda K. Differential effects of aging on motoneurons and peripheral nerves innervating the hindlimb and forelimb muscles of rats. Neurosci Res. 1995;22:189–96.
23. Pettigrew FP, Gardiner PF. Changes in rat plantaris motor unit profiles with advanced age. Mech Ageing Dev. 1987;40:178–89.
24. Lochynski D, Krutki P, Celichowski J. Effect of aging on the regulation of motor unit force in rat medial gastrocnemius muscle. Exp Gerontol. 2008;43:218–28. doi:10.1016/j.exger.2007.11.003.
25. Kanda K, Hashizume K. Changes in properties of the medial gastrocnemius motor units in aging rat. J Neurophysiol. 1989;61:737–46.
26. Kostka T. Quadriceps maximal power and optimal shortening velocity in 225 men ages 23–88 years. Eur J Appl Physiol. 2005;95:140–5.
27. Protcor DN, Balagopal P, Nair KS. Age-related sarcopenia in humans is associated with reduced synthetic rates of specific muscle proteins. J Nutr. 1998;123: 351S–5.
28. Kirkland JL, Tchkonia T, Pirtskhalava T, Han J, Karagiannides I. Adipogenesis and aging: does aging make fat go MAD? Exp Gerontol. 2002;37:757–67.
29. Ioannidis G, Papaioannou A, Adachi JD, Quality of life impacts osteoporotic fracture outcomes. 2002. www.biomech.com.
30. Clarke BL, Khosla S. Female reproductive system and bone. Arch Biochem Biophys. 2010;503(1):118–28.
31. Riggs BL, Melton III LJ, Robb RA, Camp JJ, Atkinson EJ, Peterson JM, et al. Population-based study of age and sex differences in bone volumetric density, size, geometry, and structure at different skeletal sites. J Bone Miner Res. 2004;19:1945–54.
32. Lochmuller EM, Muller R, Eckstein F. Does bone densitometry predict mechanical competence throughout the skeleton? Menopause Management 2003.
33. Wu F, Mason B, Horne A. Postmenopausal fractures linked to premenopausal fracture history. Women's Health Orthopaedic Edition. 2002;5(4):143.
34. Granito RN, Aveiro MC, Renno ACM, Oishi J, Driusso P. Comparison of thoracic kyphosis degree, trunk muscle strength and joint position sense among healthy and osteoporotic elderly women: a cross-sectional preliminary study. Arch Gerontol Geriatr. 2012;54(2):E199–202. doi:10.1016/j.archger.2011.05.012.
35. Whiting WC, Zernicke RF. Biomechanics of musculoskeletal injury. 2nd ed. Champaign, IL: Human Kinetics; 2008.
36. van den Kroonenberg AJ, Hayes WC, McMahon TA. Dynamic models for sideways falls from standing height. J Biomed Eng. 1995;117:309–18.
37. Stevens JA, Rudd RA. Declining hip fracture rates in the United States. Age Ageing. 2010;39(4):500–3.
38. Seguin R, Nelson ME. The benefits of strength training for older adults. Am J Prev Med. 2003;25(3Sii): 141–9.
39. Rich BS. Price: management of injuries: principles and practice of primary care sports medicine: 2001;665–7.
40. Reginster J, Neuprez A, Lecrat MP, Sarlet N, Bruyere O. Role of glucosamine in the treatment for osteoarthritis. Rheumatol Int. 2012;32(10):2959–67. doi:10.1007/s00296-012-2416-2.
41. Wandel S, Juni P, Tendal B, Nuesch E, Villiger PM, Welton NJ, et al. Effects of glucosamine, chondroitin, or placebo in patients with osteoarthritis of hip or knee: network meta-analysis. BMJ. 2010;341:c4675.

42. Grant AM, Avenell A, Campbell MK, McDonald AM, MacLennan GS, McPherson GC, et al. Oral vitamin D3 and calcium for secondary prevention of low-trauma fractures in elderly people (Randomised Evaluation of Calcium Or vitamin D, RECORD): a randomised placebo-controlled trial. Lancet. 2005; 365:1621–8.
43. Meunier P. Prevention of hip fractures by correcting calcium and vitamin D insufficiencies in elderly people. Scand J Rheumatol Suppl. 1996;103:79–80.
44. Rodacki CLN, Rodacki ALF, Pereira G, Naliwaiko K, Coelho I, Pequito D, et al. Fish-oil supplementation enhances the effects of strength training in elderly women. Am J Clin Nutr. 2012;95(2):428–36.
45. Burke TN, Franca FJR, de Menese SRF, Pereira RMR, Marques AP. Postural control in elderly women with osteoporosis: comparison of balance, strengthening and stretching exercises. A randomized controlled trial. Clin Rehabil. 2012. doi:10.1177/0269215512442204.

# Osteoporosis and Current Therapeutic Management

19

Kellie F. Flood-Shaffer

**Abstract**

Osteoporosis which means "porous bones" is one of the most common skeletal disorders faced by women and their health care providers today. It occurs when there is a change in the balance of bone renewal. As estrogen levels diminish with the onset of menopause, there is excessive bone resorption which is not fully compensated for by an increase in bone formation. The most rapid bone loss in a woman's life cycle appears to occur during the first 5 years after menopause. The World Health Organization (WHO) criteria for diagnosing osteoporosis and osteopenia (decreased bone mineral content) are based on a comparison of an individual's bone mineral density (BMD) measured by dual energy X-ray absorptiometry (DEXA) with that of a young adult reference group. BMD is expressed using a T-score that represents the difference in a number of standard deviations relative to the average peak bone mass of same gender, young, healthy adults. The WHO classifies osteoporosis as a T-score lower than −2.5 (World Health Organization (WHO) Scientific group on the assessment of osteoporosis at the primary health care level: report of WHO study group summary meeting report. Brussels, Belgium, 5–7 May, 2004; Kanis and Gluer. Osteoporosis Int. 11:192–202, 2009) and osteopenia as a T-score between −1 and −2.5. The primary goal of treatment is to reduce the risk of fractures. Although numerous effective therapies for the treatment of osteoporosis are readily available in a variety of forms, maintaining a healthy lifestyle with proper, balanced nutrition and sufficient, regular physical activity should be paramount for all women.

K.F. Flood-Shaffer, M.D., F.A.C.O.G. (✉)
Department of Obstetrics and Gynecology,
University of Cincinnati Medical Center,
University of Cincinnati, Cincinnati, OH, USA
e-mail: floodskf@uc.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_19, 

**Keywords**
Osteoporosis • Menopause • Osteopenia • Bone loss

## 19.1 Learning Objectives

After completing this chapter, you should have an understanding of:

1. The definition of osteoporosis;
2. The pathophysiology of osteoporosis
3. Classifications of osteoporosis;
4. Risk factors in the development of osteoporosis;
5. The diagnosis of osteoporosis;
6. Bone mineral density measurement recommendations; and
7. The management of osteoporosis.

## 19.2 Introduction

Osteoporosis which means "porous bones" is one of the most common skeletal disorders faced by women and their health care providers today. It occurs when there is a change in the balance of bone renewal. The process of bone resorption and renewal requires three elements: (a) the regular supply of protein, calcium, and vitamins; (b) the regular use of the bones (exercise); and (c) requisite hormones which harmonize and direct the breakdown and removal of old bone and the creation of new bone.

Osteoporosis is characterized by low bone mass, compromised bone strength, and structural deterioration of the bone tissue which leads to bone fragility and an increased susceptibility to fractures, most commonly of the wrist (distal radius), hip (femoral neck) and spine (especially in the lumbar vertebrae). These fragility fractures lead to a decreased quality of life and a staggering economic burden. There are an estimated 2.8 million physician visits annually, 1.7 million fractures annually, and over 450,000 hospital admissions all at a cost of over $20 billion in the USA alone. The primary goal of treatment is to reduce the risk of fractures. Current estimates are that there are over 57 million Americans affected by low bone mineral density including 9 million with osteoporosis and 48 million with osteopenia.

## 19.3 Research Findings

### 19.3.1 The Definition of Osteoporosis

Osteoporosis is a progressive, skeletal disease characterized by low bone density and deterioration of the microarchitecture of the bone tissue; the consequences of this are bone fragility and increased susceptibility to fracture [1]. The World Health Organization (WHO) criteria for diagnosing osteoporosis and osteopenia (decreased bone mineral content) are based on a comparison of an individual's bone mineral density (BMD) measured by dual energy X-ray absorptiometry (DEXA) with that of a young adult reference group. BMD is expressed using a T-score that represents the difference in a number of standard deviations relative to the average peak bone mass of same gender, young, healthy adults. The WHO classifies osteoporosis as a T-score lower than −2.5 [1, 2] and osteopenia as a T-score between −1 and −2.5. The National Institutes of Health have expanded the definition of osteoporosis to include compromised bone strength, independent of bone density.

### 19.3.2 The Pathophysiology of Osteoporosis

The process of bone remodeling occurs throughout the life cycle. The activity of osteoclasts (bone breakdown or resorption) in normal adults is balanced by the activity of osteoblasts (bone formation).

Estrogen deficiency leads to over-expression of osteoclasts. As estrogen levels diminish with the onset of menopause, there is excessive bone resorption which is not fully compensated for by an increase in bone formation. Thus, osteoporosis results from an imbalance in the two processes of bone remodeling. The result is a net loss of bone tissue with associated changes in the microarchitecture of the bone. The most rapid bone loss in a woman's life cycle appears to occur during the first 5 years after menopause. Osteoclastic activity increases with aging in all humans. Bone loss can be measured in the hip and spine years prior to the last menstrual period (menopause) [3, 4]. BMD is reduced by approximately 50 % between peak bone mass at age 30 and age 90, when bone loss seems to end.

### 19.3.3 Classifications of Osteoporosis

Riggs and Melton have classified osteoporosis as either primary, which has 2 subtypes, or secondary [5]. Primary Osteoporosis is the more common form of the disease and refers to an inadequate bone remodeling process. *Type 1* primary osteoporosis is characterized primarily by trabecular bone loss, although some measurable cortical bone loss may also occur. It is more common in women and is often termed postmenopausal osteoporosis. It results primarily from estrogen deficiency. The skeleton is more sensitive to parathyroid hormone (PTH) and this causes increased calcium resorption from the bone. *Type 2* primary osteoporosis is characterized by both trabecular and cortical bone loss. It is often called osteoporosis of aging. It occurs in adults over the age of 70 and is associated with decreased availability of vitamin D and decreased bone formation. Hallmarks are a decline in calcium absorption, increased PTH levels and bone resorption.

Secondary osteoporosis is caused by genetic, calcium balance, endocrine, gastrointestinal, neoplastic, renal, or rheumatologic disorders or as a result of medication therapies or use of other drugs. Glucocorticoid-induced osteoporosis is the most common secondary form of the disease. Glucocorticoids suppress the osteoblast function, inhibit intestinal calcium absorption, and increase osteoclastic activity. These drugs may also increase renal excretion of calcium. BMD may be increased by as much as 60 % in patients who are on glucocorticoid therapy.

Other important drugs associated with secondary bone loss include but are not limited to: alcohol, tobacco, heparin and other anticoagulants, lithium, anticonvulsants, cytotoxic drugs, gonadotropin-releasing hormone agonists, vitamin A, and Tamoxifen [6]. Chronic medical conditions which may contribute to secondary bone loss include but are not limited to: hemophilia, thalassemia, Cushing's syndrome, hypothyroidism, Type 1 diabetes mellitus, chronic liver disease, malabsorption syndromes (celiac disease), lymphoma, leukemia, disordered eating (anorexia nervosa), rheumatoid arthritis, and primary hyperparathyroidism [6].

### 19.3.4 Risk Factors in the Development of Osteoporosis

Osteoporosis is associated with a number of risk factors, some modifiable and some nonmodifiable. Nonmodifiable factors are genetic factors such as Asian or white race, female gender, family history of fragility fractures, ethnicity, and thin body habitus. These also include advanced age and a personal history of fracture [7, 8]. Modifiable factors include poor nutrition or malnutrition, estrogen deficiency, sedentary lifestyle, smoking, and excessive alcohol intake among others.

Nonmodifiable factors require that the patient receive education on these risk factors and avoid exacerbating their risk of fracture by adopting and/or not modifying any of the modifiable risk factors. Perhaps the most difficult most difficult task that a health care provider may deal with is encouraging and being successful with changing behaviors which predispose patients to osteoporosis. Sedentary lifestyle, smoking, excess use of alcohol, inadequate intake of vitamin D and calcium, and the epidemic of heavy consumption of

carbonated and caffeinated drinks in the USA continues to place younger and younger women at risk of not reaching their genetically determined peak bone mass [9].

## 19.4 Contemporary Understanding of the Issues

### 19.4.1 The Diagnosis of Osteoporosis

The gold standard for the diagnosis of osteopenia and osteoporosis of the hip and spine remains Dual-energy X-ray absorptiometry (DEXA). Dual-energy X-ray absorptiometry machine standard error is small and it has the best normative database available. For trabecular bone measurements, lateral spine DEXA may be superior to anterior-posterior views. There are clinical assessments, radiographic studies, and biochemical measurements which can be used to assist in the diagnosis as well. All women should have their risk factors for osteoporosis assessed at the time of annual physical. A thorough medical history should include review of hormone status, nutritional status (specifically to include vitamin D, calcium, and protein intake), personal history of low trauma fracture, lifestyle factors (to include smoking, alcohol intake, and physical activity level). Family history should also be reviewed with respect to height loss with age, compression or hip fractures. Height should be measured and compared to self-reported maximum height. Repeat measurements of height should be done annually. Radiographic studies of the lateral spine should be included in the evaluation if a compression fracture is suspected or spinal deformity is present. Biochemical tests may be incorporated into the diagnostic evaluation particularly if a secondary cause of osteoporosis is suspected. Labs should include: serum chemistries, complete blood count, thyroid function tests, and a 24-h urine collection for calcium excretion. Further testing may include: intact PTH, urinary free cortisol, erythrocyte sedimentation rate, serum protein electrophoresis, serum 25-hydroxyvitamin D concentration, and other biochemical markers of bone turnover.

A recent study by Pickhardt et al. found that abdominal computed tomography (CT) images obtained for other reasons that include the lumbar spine can be used to identify patients with osteoporosis or normal bone mineral density (BMD) without additional radiation exposure or cost. This study was based upon the data that shows nearly half of all female Medicare beneficiaries have never undergone BMD testing and more than 80 % of all persons with a major osteoporosis related fracture do not have BMD testing or receive pharmacologic agents to reduce fracture risk. In 2011, there were more than 80 million CT scans performed in the USA all of which may carry potentially useful information about BMD. In this study, CT scan was 90 % sensitive and more than 90 % specific for distinguishing osteoporosis from osteopenia and normal BMD. The greatest limitation of the study was that it did not assess potential benefits and costs of using CT primarily to evaluate BMD [10].

### 19.4.2 Recommendations for Bone Mineral Density Measurements

The National Osteoporosis Foundation (NOF) recommends BMD testing be performed on: (a) all women age ≥65 years of age; (b) all postmenopausal women <65 years of age if they have any additional risk factors other than being white, postmenopausal and female; and (c) postmenopausal women who have suffered a fragility fracture (to confirm diagnosis and determine disease severity) [11]. In women aged 65 or greater, if BMD is measured as normal, repeat BMD measurements can be delayed up to 15 years.

As previously mentioned, the T-score is used to determine the presence or absence of bone loss. However, the Z-score may be of more use in younger women and in identifying women who may need to undergo a workup for secondary causes of osteoporosis. The Z-score represents the difference in number of standard deviations between an individual's BMD and the BMD of a population adjusted for age, gender, and race. One should suspect secondary causes

of osteoporosis if the Z-score is >1.5 SD below the mean [12].

The American College of Rheumatology has recommended that patients undergo a baseline lumbar spine or hip BMD before initiating long-term glucocorticoid therapy or other drug therapy associated with bone loss.

Conditions which may falsely elevate BMD measurements especially in the elderly patient include osteophytes, arthritis, and spinal compression fractures. In these cases, a hip or femoral neck BMD is recommended. Another alternative site which has shown to be a good predictor of hip fracture is the calcaneus, which is rich in trabecular bone. A peripheral measurement, such as the calcaneus, is performed using single absorptiometry.

## 19.4.3 The Management of Osteoporosis

### 19.4.3.1 Prevention

Maximizing peak bone mass begins early in life with adequate nutrition and exercise [13–15]. Other lifestyle factors, such as limiting caffeine, alcohol, and carbonated drinks, and avoiding tobacco use play an important role in the prevention of osteoporosis. Caffeine, tobacco, and excess alcohol consumption all play a role in altering calcium absorption and estrogen metabolism and have been directly linked to an increase in hip, wrist and spine fractures.

Regular physical activity with weight bearing exercises should be encouraged. Weight-bearing exercise such as walking, jogging, aerobics, Tai Chi, or weight lifting helps to stimulate osteoblastic activity, thus helping to maintain the balance in bone remodeling after age 30. Physical exercise also increases muscle strength as well as core strength, coordination, agility, and balance which serve to reduce the risk of falls and thereby reduce the risk of fracture.

### 19.4.3.2 Vitamins and Minerals

#### Calcium

The daily recommended dose of calcium for premenopausal women is 1,200 mg/day to maintain bone mass. As calcium absorption decreases with age, the requirement for postmenopausal women increases to 1,500 mg/day [16]. A diet rich in calcium would include any or all of the following calcium-rich foods: dairy products such as skim milk, low-fat or nonfat yogurt, cheese, cottage cheese, and ice cream; dark green vegetables such as broccoli, kale, turnip greens; calcium fortified foods such as orange juice, breads, and cereals; and certain types of fish and shellfish. Sardines and salmon are a good source of calcium especially if the bones are consumed with the fish. Homemade chicken or turkey soup can provide another excellent source of calcium, if the poultry is boiled with the bones in place, thus allowing the calcium to leach out of the bones and into the broth.

Some dairy sources are also rich in fat therefore low calorie sources of calcium must be considered. Diet alone commonly fails to provide the daily recommended dose of calcium for most women in which case supplements are required. The two most common commercially available calcium supplements are in the form calcium carbonate and calcium citrate. Calcium citrate is generally better tolerated than calcium carbonate, which often causes gas and constipation. Daily doses of calcium exceeding 2,000 mg have been associated with increased cardiovascular disease and are not advised [17]. Vitamin D is an important requirement for adequate intestinal calcium absorption.

#### Vitamin D

The daily recommended dose of vitamin D in premenopausal women is 400–600 IU/day and for the postmenopausal woman, the range is 800–1,000 IU/day. The elderly, chronically ill, homebound or institutionalized patient is at greatest risk for vitamin D deficiency and therefore benefits the most from supplementation. Most calcium supplement products on the market, as well as multivitamin formulations are fortified with vitamin D. Women should be made aware that "more does not necessarily mean better" when taking multivitamins. Due to the association of excess vitamin A intake and increased risk of hip fracture, one should not take two multivitamins

in order to get additional vitamin D. In a large trial of elderly women, (mean age, 84), long-term calcium and vitamin D supplementation was associated with a 44 % reduction in hip fracture incidence [18]. Exposure to just 15 min of sunshine daily will provide the vast majority of the daily recommended dose of vitamin D; however, sun-exposed skin creates the additional risk of sunburn and skin cancer.

#### 19.4.3.3 Pharmacologic Therapy

Fracture prevention is the primary goal of pharmacologic therapy for osteoporosis. Secondary goals include: stabilizing and increasing BMD, relief of symptoms of fracture and skeletal deformity (ies), and improvement of the patient's mobility and functional status. Organizations such as the North American Menopause Society (NAMS), the American Association of Clinical Endocrinologists (AACE) and NOF all offer guidelines for intervening with pharmacologic therapy. Each organization bases its therapeutic recommendations on T-scores, risk factors, and prior fracture history [3, 11].

Many factors influence the use of pharmacologic therapy including but not limited to: length of time to realize benefit, impact on quality of life, impact on breast tissue, cardiovascular status, and potential side effects. Cost of therapy is always an issue as many of these therapies are quite expensive.

Prevention *and* treatment therapeutic options include: bisphosphonates, and selective estrogen receptor modulators (SERMs). Prevention-only therapy consists of hormone therapy (HT). Treatment-only therapy includes calcitonin and recombinant human PTH. There are several major clinical trials which have demonstrated the efficacy of each of these therapies in the reduction of fracture risk. Hormone therapy, raloxifene (a SERM), and the bisphosphonates have been shown to reduce risk of vertebral fracture over 3–7 years. The bisphosphonates and HT have been shown to reduce hip fracture risk.

##### Bisphosphonates

The bisphosphonates, of which there are currently several available on the US market, are antiresorptive agents which bind preferentially to hydroxyapatite crystals in the mineralized bone matrix to inhibit osteoclastic activity, thus reducing bone turnover. In addition to arresting bone loss and decreasing fracture risk, the bisphosphonates have been shown to increase BMD in postmenopausal women with osteoporosis by up to 8 % in the spine and up to 4 % in the hip. In micro CT-scans of human bone the bisphosphonates have preserved horizontal trabecular bone, thus additionally maintaining bone strength. Due to the poor bioavailability of bisphosphonates of less than 1 %, there are specific and important dosing instructions and precautions. The oral medications (both tablets and liquid forms) must be taken on an empty stomach with at least 8 oz of water to properly dissolve and absorb the drug from the stomach. The patient must remain upright (sitting or standing) for a minimum of 30 min after administration of the medication. No other food or drink is to be ingested during those 30–60 min. Careful evaluation of candidates for bisphosphonates is required and should include questions concerning any history of difficulty with swallowing, or any history of gastrointestinal disorders which cause esophageal irritation (i.e., gastroesophageal reflux disorder). There are also injectable forms of the bisphosphonates. Not all women are good candidates for bisphosphonates.

##### Alendronate

In the Fracture Intervention Trial (FIT), postmenopausal women with low bone mass were randomized to two groups on the basis of whether or not they had vertebral fractures at baseline. Alendronate showed reduction of vertebral fractures of 47 % at year 3 and reduced incidence of hip fractures up to 56 % [19]. The recommended dose of alendronate for prevention of postmenopausal osteoporosis is 5 mg/day or 35 mg/week; for treatment, the dosage is increased to 10 mg/day or 70 mg/week in either a tablet or liquid solution. Both dosage regimens are equally effective. Alendronate is also indicated for the treatment of glucocorticoid-induced osteoporosis. Importantly, newer data demonstrates that alendronate 10 mg/day can increase or maintain BMD after discontinuation of HT [20].

### Risedronate

Risedronate has the same treatment and prevention indications as alendronate as well as the glucocorticoid-induced osteoporosis prevention and treatment indication. The approved dosage of risedronate is 5 mg/day, 35 mg/week, or 150 mg/month. Risedronate has been shown to decrease both vertebral and nonvertebral fractures in postmenopausal women. At 3 years of use, risedronate was associated with a 70–75 % reduction in first vertebral fracture [21]. This reduction appears to be sustained over at least 5 years [22].

### Ibandronate

Ibandronate was approved by the FDA in May 2003; it inhibits osteoclast-mediated bone resorption. The oral dose is 150 mg once a month and the injectable form is administered intravenously in a dose of 3 mg over 15–30 s every 3 months for the prevention and treatment of osteoporosis in postmenopausal women [23]. A contraindication to Ibandronate is known hypersensitivity to Ibandronate injections or to any of its excipients. Hypersensitivity may cause anaphylaxis; fatal events have been reported.

### Zoledronic Acid

Zoledronic Acid is strictly an injectable bisphosphonates which inhibits osteoclastic bone resorption. It is usually reserved for patients who are good candidates for bisphosphonates but either cannot tolerate or have failed oral therapy. For prevention, the dose is 5 mg every 2 years and for treatment 5 mg is infused intravenously once a year. The patient must be hydrated with intravenous fluid prior to infusion of the drug, and the intravenous line must be flushed with saline solution after infusion as well. The drug is administered over no less than 15 min.

### Selective Estrogen Receptor Modulators (SERMs)

SERMs are nonhormonal agents that bind to the estrogen receptor but induce different estrogenic responses in various tissues. Raloxifene is currently the only SERM which is FDA approved for the prevention and treatment of osteoporosis.

### Raloxifene

Raloxifene has the unique property of activating estrogen receptors in the bone while not activating estrogen receptors in the breast or uterus. The recommended dose of raloxifene is 60 mg/day for both prevention and treatment of osteoporosis. Unlike the bisphosphonates, raloxifene can be taken at any time of day, with or without food. Also, unlike the bisphosphonates, raloxifene has shown an increased risk of hot flashes, leg cramps and thromboembolic events among users [24, 25]. In the Multiple Outcomes of Raloxifene Evaluation (MORE), raloxifene was associated with a 30 % reduction in vertebral fractures in postmenopausal women with at least one vertebral fracture and a 55 % reduction in postmenopausal women with no vertebral fracture over 3 years. BMD improved 2.6 % and 2.7 % at the spine and hip respectively [26]. Raloxifene was shown to have extra skeletal effects which are quite important to the overall health of women. In the MORE study, raloxifene was associated with a 65 % relative risk reduction in all breast cancers with a 90 % reduction in estrogen receptor-positive breast cancers, a 12 % reduction in estrogen receptor-negative breast cancer, and a 76 % relative risk reduction in cardiovascular events in a subgroup of at-risk women in the study group [27].

### RANK Ligand Inhibitor

The RANK ligand is a new member of the tumor necrosis factor superfamily which acts as an osteoblast-expressed ligand that stimulates osteoclastic differentiation. Inhibition of the RANK ligand has proven to b a successful method of treating osteoporosis.

### Denosumab

This medication is a RANK-ligand inhibitor and the only one currently approved in the USA for the treatment of osteoporosis. Denosumab slows the formation, action and survival of osteoclasts. Patients receive a 60 mg injection every 6 months. Common side effects include rash as well as back, bone, joint, and muscle pain.

## 19.5 Current and Future Directions

### 19.5.1 Hormone Therapy

#### 19.5.1.1 Estrogen

Bone tissue is exquisitely sensitive to estrogen. The rationale for estrogen therapy was based on the observation that the bone remodeling rate increased as estrogen levels declined resulting in greater bone resorption than bone formation. Estrogen's beneficial effect on bone is due to several factors including its ability to increase circulating calcitonin levels which leads to decreased bone resorption. Estrogen receptors are also present on osteoblastic cells, which respond to the hormone by increasing collagen production. There are over 50 randomized, placebo-controlled studies which have demonstrated that HT reduces the rate of bone resorption and loss and promotes an increase in BMD [3]. The benefits of HT on BMD are independent of estrogen preparation but are dose dependent. However, estrogen therapy even in doses as low as 0.3 mg of conjugated equine estrogen have been shown to have a beneficial effect on hip and spine BMD [28]. Low-dose birth control pills may be used for perimenopausal women at risk for bone loss, if no contraindications to that therapy exist.

Prior to the publication of the Heart and Estrogen/Progestin Replacement Study (HERS) [29] and the Women's Health Initiative (WHI) [30], most of the data available on the effects of HT in fracture prevention was based on observational and epidemiologic studies [31]. In the HERS trial, the primary evaluation endpoint was postmenopausal women and cardiovascular disease; fracture was a secondary endpoint. Study participants were not selected on the basis of osteoporosis risk. HERS concluded that fracture risk following 4 years of HT (0.625 mg/day of conjugated equine estrogen plus 2.5 mg/day of medroxyprogesterone acetate was not reduced.

The WHI Estrogen plus Progestin trial was conducted in postmenopausal women aged 50–79 (average age 63 years) with an intact uterus. The women were randomized to HT using the same preparation as the HERS trial versus placebo. The HT group showed a 34 % lower relative risk of hip and a 34 % lower relative risk of spine fracture as well as a 24 % lower risk of fracture at all sites.

In a study done in 2002, Greenspan et al. showed that once HT is withdrawn, accelerated bone loss occurs. The reduction of BMD was as much as 4.5 % in the spine and as much as 2.4 % in the hip after just 1 year off of HT [32].

The WHI created a paradigm shift which has nearly entirely changed the way estrogens are prescribed in the USA today, not due to the bone data, but rather due to the other risks which were identified. Women randomized to the HT arm of the study also showed a 29 % increased risk of coronary heart disease and a 41 % higher risk of cerebral vascular accident (stroke). The WHI also found another risk identified in HERS which was an increased risk of venous thromboembolism. Unlike HERS, which found no significant increased risk of breast cancer, the WHI reported a very significant increased risk in invasive breast cancer in women treated with HT. One other positive finding of the WHI in addition to reduced fracture risk was the data which showed a significantly decreased risk of colon cancer development in women taking HT. Following the release of the WHI both the NAMS and the US Preventive Services Task Force have recommended against the uses of estrogen for the primary treatment of osteoporosis, recommending instead, alternative therapies. Both the NAMS and the NOF do still endorse estrogen, when prescribed appropriately, as beneficial to bone health.

#### 19.5.1.2 Androgen

Androgens in women have been studied, with regard to BMD, to a limited degree. Results of two studies which employed testosterone implants and oral methyltestosterone in addition to HT indicated significant beneficial effects on BMD [33, 34]. However, the degree to which these effects were due to aromatization, thus representing estrogen rather than androgen effects, is unclear. The small number of participants in these studies and the lack of long-term, well-controlled trials prevent proper evaluation of the effects of testosterone treatment on BMD.

Currently the use of testosterone or its derivatives for the treatment of bone loss is only recommended in male patients.

#### 19.5.1.3 Progesterone

To date, there is no reliable evidence that topical progesterone creams offer protection against bone loss. In a 1999 study, women were randomized to a progesterone cream 20 mg or placebo cream, to be applied once daily. BMD was assessed every 4 months for 1 year and no significant difference was found between the treatment and control groups with respect to BMD of the hip and spine [35]. There is some evidence that norethindrone acetate may maintain BMD in women receiving gonadotropin releasing hormone agonists for conditions such as endometriosis or uterine leiomyomata. Norethindrone acetate or combination estrogen progestin regimens are used for "add-back" therapy in these women primarily to control vasomotor symptoms such as hot flashes.

#### 19.5.1.4 Calcitonin

Calcitonin is a polypeptide hormone which decreases the rate of bone absorption by inhibiting resorptive activity in osteoclasts. It is available as a subcutaneous injection (100 IU/day) or as a nasal spray (200 IU/day). It is indicated for the treatment of postmenopausal osteoporosis in females more than 5 years post menopause with low bone mass relative to healthy premenopausal females. It stabilizes or increases vertebral and nonvertebral BMD, predominantly at the forearm and lumbar spine. The Prevent Recurrence of Osteoporosis Fractures (PROOF) Study showed that the intranasal dose of calcitonin was associated with a 36 % risk reduction in vertebral fracture and only a 20 % risk reduction in nonvertebral fracture in women with low bone mass and history of vertebral compression fractures. The study had a high dropout rate and failed to show any effect on hip fractures [36].

#### 19.5.1.5 Teriparatide

Teriparatide is a synthetic form of human recombinant parathyroid hormone (PTH). PTH is the primary regulator of calcium and phosphate metabolism in bones. Teriparatide 20 mcg/day injected subcutaneously once a day in the thigh or abdomen is indicated for the treatment of osteoporosis in postmenopausal women at high risk of fracture. It is the first approved treatment for osteoporosis that stimulates both cortical and trabecular bone formation. In a randomized, double-blind trial, involving nearly 1,700 postmenopausal women with prior vertebral fracture, teriparatide 20 mcg/day reduced new vertebral fractures by 65 % and nonvertebral fractures by 35 % [37]. Prior to Teriparatide administration, laboratory tests should be done including BMD by DEXA (spine and hip), total serum calcium, total serum alkaline phosphatase, 25-hydroxyvitamin D, PTH, and creatinine clearance. Use of teriparatide is contraindicated in patients with hypercalcemia, Paget's disease, elevated bone-specific alkaline phosphatase, osteogenic sarcoma, history of skeletal radiation, pregnancy and lactation, unfused epiphyses, bone cancer or metastatic cancer to the bone. Therapy with teriparatide is not recommended for more than 2 years in humans due to the development of osteosarcomas in animal studies.

#### 19.5.1.6 Combination Therapy

Several studies have examined the effect of combining two antiresorptive agents on bone mass and have found that at the spine and total hip, the combination resulted in a greater increase than with one agent alone. A recent randomized trial by Greenspan et al., showed that in women aged 65–90 combination therapy with HT and alendronate was more effective in increasing bone mass than either therapy alone [32]. Currently, combination therapy remains controversial; however, a new study may help to resolve the controversy. In a randomized controlled trial in which patients were assigned in a 1:1:1 ratio to receive 20 μg teriparatide daily, denosumab 60 mg every 6 months, or both the data appear quite promising in favor of combination therapy. BMD was measured at 0, 3, 6, and 12 months in all groups. At 12 months, overall BMD showed a greater improvement in the combination group than in the single therapy groups. Additionally, femoral neck and total hip BMD also increased more in

the combination therapy group. The authors concluded that combination therapy might therefore be useful to treat patients at high risk of fracture [38].

### 19.5.2 Complementary and Alternative Therapy

Phytoestrogens are found in various plants and foods, these substances are similar in action to estrogen. There are conflicting data on phytoestrogens and their effects on BMD Some studies report that phytoestrogens are beneficial for bone resorption and BMD in postmenopausal women, while other studies have reported no effect [39]. Much conflicting information exists with respect to the use of soy (bean) isoflavones in the treatment of patients with osteoporosis. While it seems likely that soy isoflavones have many health benefits, the evidence for using soy as an alternative treatment in osteoporosis remains largely inconclusive. Zhang, in his study, showed that postmenopausal women who ate an average of 11 g of soy protein daily had a lower risk of fracture than those who did not ingest that level of soy [40]. One cup of soy milk contains 7–11 g of soy protein.

One 12 week trial of postmenopausal women revealed that black cohosh extract increased levels of bone-specific alkaline phosphatase, a metabolic marker for bone formation, compared to women on HT or placebo. However, no BMD measurements were done. This study may suggest that black cohosh is beneficial but the data are insufficient to be conclusive. Red clover has undergone similar evaluations and the results may be promising, but are as yet insufficient [41]. Wild yam has not been studied in a controlled trial for the treatment of osteoporosis; however, it has been marketed for that indication in the USA. All herbal remedies and alternative or complementary forms of therapy must be used with caution as little to no data from controlled trials is available. Currently herbals are sold as nutritional supplements and are therefore not regulated by the FDA. Many herbal remedies have interactions with prescription medications, ranging from annoying to life-threatening. No herbal remedy should be initiated without a thorough review and discussion of all other medications that a patient is taking.

## 19.6 Concluding Remarks

Early prevention of bone loss is the key to reducing a woman's lifetime risk of developing osteoporosis and fracture. Women should be encouraged to educate themselves with information on healthy habits and be aware of their personal risk factors both modifiable and non-modifiable. Changing one's modifiable risks by avoiding or discontinuing personal habits which are known to damage bone is vitally important to bone health. Although numerous effective therapies for the treatment of osteoporosis are readily available in a variety of forms, maintaining a healthy lifestyle with proper, balanced nutrition and sufficient, regular physical activity should be paramount for all women. Therapeutic regimens for osteoporosis are safe and beneficial and should be employed as soon as the need is identified.

## References

1. World Health Organization (WHO) scientific group on the assessment of osteoporosis at the primary health care level: report of WHO study group summary meeting report. Brussels, Belgium, 5–7 May; 2004.
2. Kanis JA, Gluer C-C. An update on the diagnosis and assessment of osteoporosis with densitometry. Committee of Scientific Advisors, International Osteoporosis Foundation. Osteoporos Int. 2009;11: 192–202.
3. Management of osteoporosis in postmenopausal women: 2010 position statement of the North American Menopause Society. Menopause. 2010; 17(1): 25–54.
4. Recker RR, Lappe J, Davies K, et al. Characterization of perimenopausal bone loss: a prospective study. J Bone Miner Res. 2000;15:1963–73.
5. Riggs BL, Melton III LJ. Involutional osteoporosis. N Engl J Med. 1992;326:357–62.
6. Fitzpatrick LA. Secondary causes of osteoporosis. Mayo Clin Prac. 2002;77:453–68.
7. Slemenda CW, Christian JC, Williams CJ, et al. Genetic determinants of bone mass in adult women: a

reevaluation of the twin model and the potential importance of gene interaction of heritability estimates. J Bone Miner Res. 1991;6:561–7.
8. Pocock NA, Eisman JA, Hopper JL, et al. Genetic determinants of bone mass in adults: a twin study. J Clin Invest. 1987;80:706–10.
9. Nordin BE, Need AG, Steurer T, et al. Nutrition, osteoporosis, and aging. Ann N Y Acad Sci. 1998;854: 336–51.
10. Pickhardt P, Pooler D, Lauder T, et al. Opportunistic screening for osteoporosis using abdominal computed tomography scans obtained for other indications. Ann Intern Med. 2013;158(8):588–99.
11. Osteoporosis. Belle Mead, NJ: Excerpta Medica, Inc; 2003.
12. Msissinger-Papport BJ, Thacker HL. Prevention for the older woman: a practical guide to prevention and treatment of osteoporosis. Geriatrics. 2002;57:16–27.
13. Lau EM, Woo J. Nutrition and osteoporosis (review). Curr Opin Rheumatol. 1998;10:368–72.
14. Nieves JW, Golden AL, Siris E, et al. Teenage and current calcium intake are related to bone mineral density of the hip and forearm in women aged 30–39 years. Am J Epidemiol. 1995;141:342–51.
15. Gutin B, Kasper MJ. Can vigorous exercise play a role in osteoporosis prevention? A review. Osteoporos Int. 1992;2:55–69.
16. Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Institute of Medicine. Dietary reference intakes: calcium, phosphorus, magnesium, vitamin D, and fluoride. Washington, DC: National Academy Press; 1997.
17. Michaelsson K, Mehus H, Warensjo Lemming E, et al. Long-term calcium intake and rates of all cause and cardiovascular mortality: community based prospective longitudinal cohort study. Br Med J. 2013; 346:f228.
18. Chapuy MC, Arlot ME, Duboeuf F, et al. Vitamin D3 and calcium to prevent hip fractures in elderly women. N Engl J Med. 1992;327:1637–42.
19. Black DM, Cummings SR, Karpf DB, for the Fracture Intervention Trial Research Group, et al. Randomized trial of effect of alendronate on risk of fracture in women with existing vertebral fractures. Lancet. 1996;348:1535–41.
20. Ascott-Evans BH, Guanabens N, Kivinen S, et al. Alendronate prevents loss of bone density associated with discontinuation of hormone replacement therapy: a randomized controlled trial. Arch Intern Med. 2003;163:789–94.
21. Reginster J-Y, Minne HW, Sorenson OH, et al. Randomized trial of the effects of risedronate on vertebral fractures in women with established postmenopausal osteoporosis. Osteoporos Int. 2000;11:83–91.
22. Sorensen OH, Crawford GM, Mulder AH, et al. Long-term efficacy of risedronate: a 5-year placebo-controlled clinical experience. Bone. 2003;32:120–6.
23. Chestnut III CH, Skag A, Christiansen C, et al. Effects of oral ibandronate administered daily or intermittently on fracture risk in postmenopausal osteoporosis. J Bone Miner Res. 2004;19:1241–9.
24. Cranney A, Tugwell P, Zytaruk N, et al. Meta-analysis of therapies for postmenopausal osteoporosis. IV. Meta-analysis of raloxifene for the prevention and treatment of postmenopausal osteoporosis. Endocr Rev. 2002;23:524–8.
25. Ettinger B, Black DM, Mitlak BH. Reduction of vertebral fracture risk in postmenopausal women with osteoporosis treated with raloxifene: results from a 3-year randomized clinical trial. Multiple Outcomes of Raloxifene Evaluation (MORE) investigators. JAMA. 1999;282:637–45.
26. Cummings SR, Eckert S, Krueger KA, et al. The effect of raloxifene on risk of breast cancer in postmenopausal women: results from the MORE randomized trial. Multiple Outcomes of Raloxifene Evaluation. JAMA. 1999;281:2189–97.
27. Barrett-Connor E, Grady D, Sashegyi A, et al. Raloxifene and cardiovascular events in osteoporotic postmenopausal women: a four-year results from the MORE randomized trial. JAMA. 2002;287:847–57.
28. Recker FF, Davies KM, Dowd RM, et al. The effect of low-dose continuous estrogen and progesterone therapy with calcium and vitamin D on bone in elderly women: a randomized, controlled trial. Ann Intern Med. 1999;130:897–904.
29. Simon JA, Hsia J, Cauley JA, et al. Postmenopausal hormone therapy and risk of stroke: the Heart and Estrogen/Progestin Replacement Study (HERS). Circulation. 2001;133:638–42.
30. Writing Group for the Women's Heal Initiative Investigators. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women's Health Initiative randomized controlled trial. JAMA. 2002;288:321–33.
31. Wells G, Tugwell P, Shea B, et al. Meta-analysis of the efficacy of hormone replacement therapy in treatment and preventing osteoporosis in postmenopausal women. Part V. Endocr Rev. 2002;23:529–39.
32. Greenspan S, Resnick N, Parker R. Combination therapy with hormone replacement and alendronate for prevention of bone loss in elderly women: a randomized controlled trial. JAMA. 2003;289:2525–32.
33. Davis SR, McCloud P, Strauss BJ, et al. Testosterone enhances estradiol's effects on postmenopausal bone density and sexuality. Maturitas. 1995;21:227–36.
34. Barrett-Connor E, Young R, Notelovitz M, et al. A two-year, double-blind comparison of estrogen-androgen and conjugated estrogens in surgically menopausal women. Effects on bone mineral density, symptoms and lipid profiles. J Reprod Med. 1999;44: 1012–20.
35. Leonetti HB, Longa S, Anasti J. Transdermal progesterone cream for vasomotor symptoms and postmenopausal bone loss. Obstet Gynecol. 1999; 94:225–8.
36. Chestnut III CH, Silverman S, Andriano K, for the PROOF Study Group, et al. A randomized trial of nasal spray salmon calcitonin in postmenopausal women with established osteoporosis: the Prevent Recurrence of Osteoporotic Fractures Study. Am J Med. 2000;109:267–76.

37. Neer RM, Arnaud CD, Zanchetta JR, et al. Effect of parathyroid hormone (1–34) on fractures and bone mineral density in postmenopausal women with osteoporosis. N Engl J Med. 2001;344:1434–41.
38. Tsai JN, Uihlein A, Lee H, et al. Teriparatide and Denosumab, alone or combined in women with postmenopausal osteoporosis: the DATA study randomized trial. Lancet. 2013;382(9886):50–6. doi:10.1016/S0140-6736(13)60856-9.
39. Supplement: fourth international symposium on the role of soy in preventing and treating chronic disease. The American Society for Nutritional Sciences. 2002;132:588S–619.
40. Zhang X, Shu X-O, Li H, et al. Prospective cohort study of soy food consumption and risk of bone fracture among postmenopausal women. Arch Intern Med. 2005;165:1890–5.
41. PDR for herbal medicines, 4th ed. Thompson PDR; 2007. G5, 95; 765–76, 899–900.

# Part IV

# Exercise Guidelines and Precautions for Active Females

# Traditional and Nontraditional Empirically Based Exercise Programs for Active Females

20

Jacalyn J. Robert-McComb and Anna M. Tacón

**Abstract**

Girls and women are generally less active than boys and men. In order to help promote exercise as a tool for health, major organizations have set exercise guidelines to help people exercise safely, efficiently, and effectively. In order for anyone to continue to exercise, it must be enjoyable. Girls and women need to find either a traditional or nontraditional exercise program that they enjoy and will pursue willingly. An exercise regimen should include cardiovascular endurance, muscular strengthening, and flexibility exercises. Recommendations for cardiovascular fitness include at least 5 days•week$^{-1}$ of moderate activity for at least 30 min each day, OR 3 or more days•week$^{-1}$ of vigorous activity for at least 20 min, OR a combination of the two. Individuals should perform muscular strengthening at least 2 days•week$^{-1}$ and implement flexibility routines a minimum of 2–3 days•week$^{-1}$. Alternative exercises such as yoga, breathing, and mindfulness while exercising are also discussed in this chapter. Physical activity is key to long-term good health regardless of gender and age; however, it is especially important for girls and women to be active throughout their life span.

**Keywords**

Exercise guidelines • Physical activity • Yoga • Aerobic training • Resistance training • Mindfulness

## 20.1 Learning Objectives

After completing this chapter, you should have an understanding of:

1. The physical activity objectives for health from Healthy People 2020;
2. The difference between moderate physical activity, vigorous physical activity, exercise,

J.J. Robert-McComb, Ph.D., FACSM, ACSM P.D. (✉)
A.M. Tacón, Ph.D.
Texas Tech University, Lubbock, TX, USA
e-mail: jacalyn.mccomb@ttu.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_20, 

health-related physical fitness, and sports-related fitness;
3. Physical activity recommendations for health-related fitness from leading health organizations such as the United States Department of Health and Human Services;
4. The American College and Sports Medicine's exercise recommendations for cardiorespiratory fitness, muscular fitness, healthy body composition, and flexibility; and
5. Yoga and meditation exercises and the role mindfulness plays in women's health.

## 20.2 Introduction

A landmark report on the benefits of physical activity was released on July 11, 1996, by the United States Department of Health and Human Services. It was the first Surgeon General's Report (SGR) on Physical Activity and Health [1]. The SGR indicated that although health benefits improve with moderate amounts of physical activity (15 min of running, 30 min of brisk walking, or 45 min of playing volleyball), greater amounts are obtained with greater amounts of physical activity [1]. Healthy People 2000: National Health Promotion and Disease Prevention Objectives followed this landmark report with physical activity objectives for all Americans. As understanding of the benefits of physical activity grew, recommendations followed suit; since, on average, physically active people outlive those who are inactive [2]. In November of 2000, the United States Department of Health and Human Services ([HHS] http://www.hhs.gov) released Healthy People 2010 [3]. In this document it was stated that women are generally less active than men at all ages and by age 75, one in three men and one in two women engage in *no* regular physical activity. Ten years later, Healthy People 2020 [4] was released by HHS. Healthy People 2020 was based on the accomplishments of four previous Healthy People initiatives as mentioned previously. Even after all of this effort by governing agencies to increase the level of physical activity for all Americans, the stated statistics for the number of people who engage in regular physical activity actively from 2000 to 2010 were not any better. Healthy People 2020 [4] states (http://www.healthypeople.gov/2020/):

> More than 80 % of adults do not meet the guidelines for both aerobic and muscle-strengthening activities. Similarly, more than 80 % of adolescents do not do enough aerobic physical activity to meet the guidelines for youth. This has important public health implications.

*Why aren't more people being active*? As an exercise physiologist, I meet women every day who tell me that they are going to begin exercising. They also state unrealistic goals, I am going to exercise vigorously every day for an hour a day. I tell them, do not overdo it or you will not continue exercising. They understand the importance of being active, but they just cannot motivate themselves to carry out the activity.

The initiatives by leading governing agencies to increase the level of physical activity in all Americans reflects the importance of being physically active for long-term good health for all ages and all genders, *especially young girls and women*. Healthy People 2020 [4] defines physical activity as "participation in moderate and vigorous physical activities and muscle-strengthening activities." The American Heart Association and the American College of Sports Medicine (ACSM) recommended that all adults in the USA should accumulate at least 30 min a day of moderate activity minimally 5 days a week or 20 min of vigorous activity at least 3 days a week or a combination of the two. They also recommend at least 2 days a week of muscular strengthening activity [5]. This is a big window for types of activities that can be chosen to meet health objectives.

The National Institutes of Health have funded numerous studies which have assessed the health benefits of a different type of exercise called Mindfulness. Mindfulness incorporates yoga and meditation. The focus of Mindfulness is on awareness. In this chapter we will discuss the guidelines established by leading health organizations for physical activity and exercise. We will also introduce you to a more subtle type of exercise called Mindfulness. What is important is that

young girls and women participate in activities that are enjoyable, maybe it is *Zumba*, maybe it is *Pilates*, maybe it is *Salsa Dancing*; regardless of the activity, the reward comes with dedication to practice. If you do not enjoy the type of activity that you commit yourself to practice, you will not continue in the practice. So it is extremely important to find activities that are fun and rewarding for you and your children.

## 20.3 Research Findings

Why is Physical Activity Important? According to extensive research employing well designed studies, we can now say with certainty this statement as found on the Healthy People 2020 Web site [4]: http://www.healthypeople.gov/2020/topicsobjectives2020/overview.aspx?topicid=33

> Regular physical activity can improve the health and quality of life of Americans of all ages, regardless of the presence of a chronic disease or disability. Among adults and older adults, physical activity can lower the risk of: Early death; Coronary heart disease; Stroke; High blood pressure; Type 2 diabetes; Breast and colon cancer; Falls; and Depression. Perhaps, even more importantly, *among children and adolescents*, physical activity can:
>
> - improve bone health,
> - improve cardiorespiratory and muscular fitness,
> - decrease levels of body fat,
> - reduce symptoms of depression.

Just what are the physical activity objectives for Healthy People 2020? Table 20.1 highlights some of the objectives designed to improve health, fitness, and quality of life through daily physical activity. From this list, you can see the importance of being active in terms of long-term good health. Notice the emphasis on providing avenues for physical activity for all ages, even children in daycare.

Another series of questions probably arise when you look at this table: what is *moderate* exercise, what is *vigorous* exercise, why is the term used *physical activity* rather than *exercise*? We will answer these questions in the next section of the chapter.

### 20.3.1 The Difference Between Moderate Physical Activity, Vigorous Physical Activity, Exercise and Physical Activity, Health-Related Physical Fitness, and Sports-Related Fitness

According to Ainsworth and colleagues moderate-intensity physical activity refers to any activity that burns 3.5–7 kcal/min [6] or 3.5–5 METS [7]. One MET is the oxygen cost of an individual at rest or MET = 1 kcal/kg/h. METS are used because it is an easy way to represent energy. If someone is working at a 5 MET level they are working five times above their resting state. The ACSM defines moderate exercise intensity as 40 % to <60 %of oxygen uptake reserve ($VO_2$ R) or heart rate reserve (HHR) [8]. In the 1996 Surgeon General's Report [1], moderate exercise was defined as some increase in breathing or heart rate or a "perceived exertion" of 11–14 on the Borg Rate of Perceived Exertion (RPE) scale. These levels are equal to the effort a healthy individual might burn while walking at a 3–4.5 mph pace on a level surface, playing golf, gardening and yard work, swimming for recreation, or bicycling. In Appendix 1, steps are outlined to determine a moderate training heart rate using the HRR method. The Borg RPE scale can be found in Appendix 2.

Ainsworth et al. defined vigorous-intensity physical activity as any activity that burns more than 7 cal per min ([6] kcal/min) or equal to 6 METS [7]. The ACSM [8] refers to vigorous exercise as 60 % to <90 % of $VO_2$ R or HHR. In the 1996 Surgeon General's Report [1], vigorous exercise was defined as a large increase in breathing or heart rate (conversation is difficult or "broken") or a "perceived exertion" of 15 or greater on the Borg RPE scale (see Appendix 2). These levels are equal to the effort a healthy individual might burn race walking or walking at a 5 mph pace, engaging in heavy yard work, participating in high-impact aerobic dancing, swimming continuous laps, scuba diving, or bicycling uphill.

Examples of general physical activities that meet the guidelines established by the Centers

**Table 20.1** Selected healthy people 2020 physical activity objectives: a verbatim sample

| |
|---|
| Reduce the proportion of adults who engage in no leisure-time physical activity |
| Increase the proportion of adults who engage in aerobic physical activity of at least moderate intensity for at least 150 min•week$^{-1}$, or 75 min•week$^{-1}$ of vigorous intensity, or an equivalent combination |
| Increase the proportion of adults who engage in aerobic physical activity of at least moderate intensity for more than 300 min•week$^{-1}$, or more than 150 min•week$^{-1}$ of vigorous intensity, or an equivalent combination |
| Increase the proportion of adults who perform muscle-strengthening activities on 2 or more days of the week |
| Increase the proportion of adolescents who meet current Federal physical activity guidelines for aerobic physical activity and for muscle-strengthening activity (See http://www.health.gov/paguidelines/guidelines/chapter3.aspx or Tables 20.5, 20.6, 20.9 and 20.10 for physical activity guidelines for children.) |
| Increase the proportion of children and adolescents who do not exceed recommended limits for screen time |
| Increase the proportion of the Nation's public and private schools that require daily physical education for all students |
| Increase the number of States with licensing regulations for physical activity provided in child care |
| Increase the number of States with licensing regulations for physical activity in child care that require children to engage in vigorous or moderate physical activity |
| Increase the proportion of trips made by walking |
| Increase the proportion of trips made by bicycling |
| Increase the proportion of office visits made by patients with a diagnosis of cardiovascular disease, diabetes, or hyperlipidemia that include counseling or education related to exercise |
| Increase the proportion of physician visits made by all child and adult patients that include counseling about exercise |
| Increase the proportion of employed adults who have access to and participate in employer-based exercise facilities and exercise programs |

Note: For a complete list of objectives see: http://healthpeople.gov/2020/topicsobjectives2020list.aspx?topicid=33

for Disease Control and Prevention(CDC) and the ACSM for moderate activity (3.5–5 METs or 3.5–7 kcal/min) and vigorous activity (more than 7 kcal/min or equal to or greater than 6.0 METs) can be found at the CDC Web site http://www.cdc.gov/nccdphp/dnpa/physical/recommendations/adults.htm *and* at the Compendium of Physical Activities Web site https://sites.google.com/site/compendiumofphysicalactivities/. Table 20.2 highlights the MET cost of general activities from the compendium of physical activities.

What is the difference between *physical activity* and *exercise*? The United States Department of Health and Human Services refers to *physical activity* as, bodily movement that is produced by the contraction of skeletal muscle that substantially increases energy expenditure [1], whereas *exercise* is a type of physical activity that represents structured, planned activities, and repetitive bodily movement, designed to maintainor enhance overall physical fitness [8].

Both physical activity and exercise contribute to increases in physical fitness. *Physical fitness* is a multidimensional concept that has been defined as a set of attributes that people possess or achieve that relates to the ability to perform physical activity [8]. There are skill-related components of physical fitness (also known as sports-related forms of physical fitness) and health-related components of physical fitness. *Skill-related components of physical fitness* include balance, agility, coordination, speed, power and reaction time. These components are associated mostly with sport performance. *Health-related components of physical fitness* include cardiovascular endurance, muscular strength and endurance, flexibility, and body composition. The focus of this chapter is on the health-related components of physical fitness.

### 20.3.2 Physical Activity Recommendations for Health-Related Fitness from Leading Health Organizations

Before beginning any physical activity or exercise program, it is recommended that individuals complete a self-administered questionnaire

**Table 20.2** Metabolic equivalents (MET) values for physical activity levels

| Physical activity intensity | MET |
|---|---|
| *Light intensity activities* | <3 |
| Sleeping | 0.9 |
| Watching television | 1.0 |
| Writing, desk work, typing | 1.8 |
| Walking, 1.7 mph (2.7 km/h), level ground, strolling, very slow | 2.3 |
| Walking, 2.5 mph (4 km/h) | 2.9 |
| *Moderate intensity activities* | 3–6 |
| Bicycling, stationary, 50 W, very light effort | 3.0 |
| Walking 3.0 mph (4.8 km/h) | 3.3 |
| Calisthenics, home exercise, light or moderate effort, general | 3.5 |
| Walking 3.4 mph (5.5 km/h) | 3.6 |
| Bicycling, <10 mph (16 km/h), leisure, to work or for pleasure | 4.0 |
| Bicycling, stationary, 100 W, light effort | 5.5 |
| *Vigorous intensity activities* | >6 |
| Jogging, general | 7.0 |
| Calisthenics (e.g., push-ups, sit-ups, pull-ups, jumping jacks), heavy, vigorous effort | 8.0 |
| Running jogging, in place | 8.0 |
| Rope jumping | 10.0 |

Note: 1 MET = 1 kcal $kg^{-1}$ $h^{-1}$ or 1 MET = 3.5 mL $kg^{-1}$ $min^{-1}$ of $O_2$

From compendium of Physical Activity found at https://sites.google.com/site/compendiumofphysicalactivities/

Project supported by University of Arizona and the National Cancer

to help identify risks that may warrant further medical clearance before embarking on an activity program. Individuals should consult their physician before beginning a new physical activity program if they have chronic diseases, such as cardiovascular disease and diabetes mellitus, or are at high risk for these diseases. Additionally, men over age 45, and women over age 55, should consult a physician before beginning a vigorous activity program [8].

Table 20.3 lists physical activity recommendations for cardiorespiratory (CR) health-related fitness from selected leading health organizations. Public health recommendations have evolved from emphasizing vigorous activity for CR health-related fitness to including the option of moderate levels of activity for numerous health benefits.

**Table 20.3** Physical activity recommendations for health-related fitness from leading health organizations

| |
|---|
| *Organization*: Centers for Disease Control (CDC) and the *2008 Physical Activities Guidelines for Americans* (see http://www.cdc.gov/) |
| *Recommendation*: All adults aged 18–65 years should accumulate a minimum of 30 min of moderate exercise 5 days•week$^{-1}$ or 20 min of vigorous exercise 3 days•week$^{-1}$ (or any combination of the two) |
| It is also acknowledged that for most people, greater health benefits can be obtained by engaging in physical activity of more vigorous intensity or of longer duration |
| Every adult should participate in activities at least twice a week that increase or maintain muscular strength and endurance |
| *Purpose*: Health promotion and prevention of chronic diseases |
| *Organization*: American Heart Association (see http://www.heart.org) |
| *Recommendation*: Older adults and people with disabilities can gain significant health benefits with a moderate amount of physical activity, preferably daily. Physical activity doesn't need to be strenuous to bring health benefits. What's important is to include activity as part of a regular routine |
| Children and adolescents should participate in at least 60 min of moderate to vigorous physical activity every day |
| *Purpose*: Health promotion and prevention of chronic diseases |
| *Organization*: American College of Obstetrics and Gynecology (see http://www.acog.org/) |
| *Recommendation*: For the promotion of a healthy pregnancy and postpartum recovery, women should accumulate 30 min of exercise a day on most days of the week (American College of Obstetrics and Gynecology Committee on Obstetric Practice. Exercise during pregnancy and the postpartum period. Committee Opinion No. 267. Int J Gynaecol Obstet 2002;77:79–81) |
| *Purpose*: Promotion of a healthy pregnancy and postpartum recovery |

### 20.3.3 The American College of Sports Medicine's Exercise Recommendations for Cardiorespiratory Fitness

Aerobic fitness, or CR fitness refers "to the ability to perform large muscle, dynamic, moderate-to-high intensity exercise for prolonged periods" The terms CR fitness, $VO_{2max}$, aerobic capacity, and aerobic fitness are used synonymously. CR fitness is defined as the ability

of the body to engage in physical activity in which oxygen consumption is relied on as the primary energy source [8]. These terms refer to the maximal capacity to produce energy aerobically and are usually expressed in METs or mL $O_2$•$kg^{-1}$•$min^{-1}$. One MET (metabolic equivalent unit) is equal to approximately 3.5 mL $O_2$•$kg^{-1}$•$min^{-1}$. Improvements in the ability of the heart to deliver oxygen to the working muscles and in the muscle's ability to generate energy aerobically results in increased CR fitness [8].

Exercise recommendations must take into account the fitness level of the individual. Individuals with low level of fitness generally demonstrate the greatest improvements in CR fitness, whereas modest increases occur in healthy individuals and in those with high initial fitness levels [10]. Exercise recommendations to improve CR fitness include three components, frequency, intensity, and duration, also known as the FIT principles. The format for an exercise session should include a warm-up period (approximately 5–10 min), a stimulus or conditioning phase (20–60 min), and a cool-down period (5–10 min). Table 20.4 lists the recommendations proposed by the ACSM to improve and maintain CR fitness for Adults. The recommendation range is broad because of the heterogeneity in a response to an exercise stimulus.

For children, the emphasis is on play rather than structured exercises. Tables 20.5 and 20.6 highlight recommendations from leading organizations for children and adolescents t increase aerobic fitness.

### 20.3.4 The American College of Sports Medicine's Exercise Recommendations for Muscular Fitness

Improving muscular functioning through a resistance-training program may provide physiological benefits for girls and women of all ages. Research has clearly indicated that strength can be effectively increased with training in girls before the age of puberty [9] For middle aged, older adult, and postmenopausal women, a reduction in the risk of osteoporosis, low-back pain, hypertension and diabetes have been associated with resistance training programs [10, 11]. Appendix 3 displays resistance training exercises that can be performed by the mature woman.

Unlike cardiovascular activity, intensity for resistance exercise is not easily determined. Miriam E. Nelson [12], author of Strong Women Stay Young, suggests that women beginning a resistance-training program should use a scale similar to the RPE scale (see Appendix 2) to determine the amount of weight that they should incorporate into their resistance-training program. In order for strength gains to continue to accrue there must be a gradual increase in the stress or load placed on the body throughout the resistance-training program. The *overload principle* refers to placing greater demands on the body than what it is accustomed to, and the *principle of progression* refers to the constant application of the overload principle throughout the resistance-training program. Hence, the term *progressive overload* has been coined. The RPE scale has become a popular method to assess *progressive overload*. Nelson suggests that during the first week, individuals should focus on form and that the effort involved in lifting, should be easy or moderate. When learning the exercises, an intensity of 9 (very light) to 11 (light) on the RPE scale would be appropriate. According to Nelson [12], the goal should be for the exercise set (eight repetitions) to become difficult after six or seven repetitions. The ACSM recommends an initial goal of 12–13 (somewhat hard) and a final goal of 15–16 (hard, heavy) on the RPE scale for submaximal training [13, 14]. A target of 19–20 (extremely hard, maximal exertion) on the RPE scale is synonymous with high-intensity strength stimuli for healthy populations [14]. However, for people with high cardiovascular risk or those with chronic disease, the exercise should be terminated if the lifting portion of the exercise becomes difficult corresponding to an RPE of 15–16 [13] or if there are any warning signs such as dizziness, unusual shortness of breath, anginal discomfort, or dysrhythmias [8, 15]. Therefore, a more modest level of exertion should be chosen. Table 20.7 provides resistance guidelines as

**Table 20.4** American College of Sports Medicine's training guidelines for cardiorespiratory fitness

| |
|---|
| *Frequency* |
| 3–5 days•week$^{-1}$ |
| *Intensity* |
| *Frequency* |
| 3–5 days•week$^{-1}$ |
| *Intensity* |
| *Determining intensity using the $HR_{max}$ method* |
| Moderate intensity: 64–76 % of maximum heart rate ($HR_{max}$) for a 20 year old |
| $HR_{max}$ = 220 − age |
| Target heart rate = $HR_{max}$ (64–76 %) |
| i.e., $HR_{max}$ = 220 − 20 |
| $HR_{max}$ = 200 |
| Target heart rate = 200 (64–76 %) |
| Target heart rate may range from 128 to 152 beats min$^{-1}$ |
| Vigorous intensity: 76–96 % of maximum heart rate ($HR_{max}$) for a 20 year old |
| $HR_{max}$ = 220 − age |
| Target heart rate = $HR_{max}$ (76–96 %) |
| i.e., $HR_{max}$ = 220 − 20 |
| $HR_{max}$ = 200 |
| Target heart rate = 200 (76–96 %) |
| Target heart rate may range from 152 to 192 beats min$^{-1}$ |
| *Determining intensity using the HRR method* |
| Moderate intensity: 40–60 % of heart rate reserve (HRR) for a 40 year old with a resting HR of 60 beats min$^{-1}$ |
| HRR = $HR_{max}$ (i.e., 220 − age) − resting heart rate |
| Target heart rate = [(HRR) (exercise intensity)] + resting heart rate |
| i.e., Target heart rate |
| = $HR_{max}$ = 220 − 40 = 180 |
| = HRR = 180 − 60 = 120 |
| = [(HRR) (exercise intensity)] + resting heart rate |
| = [(120) (40–60 %)] + 60 |
| = 108–132 beats min$^{-1}$ is your training heart rate range |
| Vigorous intensity: 60–90 % of heart rate reserve (HRR) for a 40 year old with a resting HR of 60 beats min$^{-1}$ |
| HRR = $HR_{max}$ (i.e., 220 − age) − resting heart rate |
| Target heart rate = [(HRR) (exercise intensity)] + resting heart rate |
| i.e., Target heart rate |
| = $HR_{max}$ = 220 − 40 = 180 |
| = HRR = 180 − 60 = 120 |
| = [(HRR) (exercise intensity)] + resting heart rate |
| = [(120) (60–90 %)] + 60 |
| = 132–168 beats min$^{-1}$ is your training heart rate range |
| *Determining intensity using the $VO_2R$ method* |
| Moderate Intensity 40 % to <60 % of oxygen uptake reserve ($VO_2R$) |
| Vigorous Intensity 60 % to <85 % of oxygen uptake reserve ($VO_2R$) |
| $VO_2R = VO_{2max} - VO_{2rest}$ ($VO_{2max}$ mL•kg$^{-1}$•min$^{-1}$ − 3.5 mL•kg$^{-1}$•min$^{-1}$) |
| Target $VO_2$ mL•kg$^{-1}$•min$^{-1}$ = [($VO_2R$) (exercise intensity)] + $VO_{2rest}$ |
| i.e., Target $VO_2$ mL•kg$^{-1}$•min$^{-1}$ for a person with $VO_{2\ max}$ of 40 mL•kg$^{-1}$•min$^{-1}$ |
| = [(40 mL•kg$^{-1}$.min$^{-1}$ − 3.5 mL•kg$^{-1}$•min$^{-1}$) (60 %)] + 3.5 mL•kg$^{-1}$.min$^{-1}$ (example) |
| = [(36.5 mL•kg$^{-1}$.min$^{-1}$) (60 %)] + 3.5 mL•kg$^{-1}$•min$^{-1}$ (example) |
| = 21.9 mL•kg$^{-1}$•min$^{-1}$ + 3.5 mL•kg$^{-1}$•min$^{-1}$ (example) |
| = 25.4 mL•kg$^{-1}$•min$^{-1}$ (example) |
| *Determining exercise intensity using the rate of perceived exertion* (RPE) *scale* |
| 12–16 (in the range of "somewhat hard" to "hard") on the original 6–20 Borg RPE Scale is the average range associated with physiologic adaptation. However, there is significant interindividual variability in the psychophysiologic relationship to % $HR_{max}$, % HRR or %$VO_2R$ and RPE. Therefore, RPE should only be used as a guideline in setting the exercise intensity |
| *Duration* |
| 20–60 min of continuous aerobic activity. Duration is dependent on the intensity of the activity: *thus*, *lower intensity should be conducted over a longer period of time* |

*Note*: For individuals with $VO_{2max}$ below 40 mL $O_2$•kg$^{-1}$•min$^{-1}$, a minimal intensity of 30 % $VO_2R$ or HRR can elicit improvements in $VO_{2max}$. In contrast, individuals with greater cardiorespiratory (CR) fitness (>40 mL $O_2$ kg$^{-1}$•min$^{-1}$) require a minimal threshold of 45 % $VO_2R$ or HRR. For most individuals, intensities within the range of 60–80 % of $VO_2R$ or HRR or 70–90 % $HR_{max}$ are sufficient to achieve improvements in CR fitness with the appropriate duration and frequency of training.

Sources: Adapted from American College of Sports Medicine. ACSM's guidelines for exercise testing and prescription. 7th ed., Philadelphia: Lippincott Williams & Wilkins, 2006. Adapted from American College of Sports Medicine. ACSM's guidelines for exercise testing and prescription. 9th ed., Philadelphia: Lippincott Williams & Wilkins, 2013

**Table 20.5** National Association for Sport and Physical Education (NASPE) national guidelines for physical activity for children

| |
|---|
| Toddlers should engage in a total of at least 30 min of structured physical activity each day |
| Toddlers should engage in at least 60 min—and up to several hours—per day of unstructured physical activity and should not be sedentary for more than 60 min at a time, except when sleeping |
| Preschoolers should accumulate at least 60 min of structured physical activity each day |
| Preschoolers should engage in at least 60 min—and up to several hours—of unstructured physical activity each day, and should not be sedentary for more than 60 min at a time, except when sleeping |
| Children age 5–12 should accumulate at least 60 min, and up to several hours, of age-appropriate physical activity on all, or most days of the week. This daily accumulation should include moderate and vigorous physical activity with the majority of the time being spent in activity that is intermittent in nature |
| Children age 5–12 should participate each day in a variety of age-appropriate physical activities designed to achieve optimal health, wellness, fitness, and performance benefits |
| Children age 5–12 should participate each day in a variety of age-appropriate physical activities designed to achieve optimal health, wellness, fitness, and performance benefits |

Sources: Adapted from NASPE National Guidelines for Physical Activity for Children http://www.aahperd.org/naspe/standards/nationalGuidelines/PAguidelines.cfm

**Table 20.6** American College of Sports Medicine recommendations for children

| | |
|---|---|
| *Frequency* | Daily |
| *Intensity* | *Moderate intensity* corresponds to noticeable increase in HR with breathing |
| | *Vigorous intensity* corresponds to substantial increase in HR and breathing |
| | Moderate to vigorous intensity aerobic exercise daily with vigorous intensity exercise 3 days•week$^{-1}$ |
| *Time* | ≥60 min per day |
| *Type* | Enjoyable running, walking, swimming bicycling, dancing |
| *Obesity* | Obese children may need to start slowly and build to achieve their goal of daily moderate to vigorous intensity exercise. Rest and recovery periods need to be included between bouts of exercise |

Sources: Adapted from American College of Sports Medicine. ACSM's guidelines for exercise testing and prescription. 9th ed., Philadelphia: Lippincott Williams & Wilkins, 2013

**Table 20.7** American College of Sports Medicine's resistance training guidelines for healthy adults

| |
|---|
| *Frequency* |
| Each major muscle group should be trained 2–3 days•week$^{-1}$ |
| *Intensity* |
| 60–70 % 1-RM (moderate-to-vigorous intensity) for strength improvement in novice and intermediate trainers |
| 80 % or less 1-RM (vigorous-to-very vigorous) for strength improvement in experienced trainers |
| 40–50 % 1-RM (very light-to-light) for older individuals and sedentary individuals whom are just beginning a program |
| <50 % 1-RM to improve muscular endurance |
| *Type* |
| Multijoint exercises affecting more than one muscle group are recommended |
| *Repetitions* |
| 8–12 repetitions is recommended for strength and power improvements |
| 10–15 repetitions is effectives in middle-aged and older individuals beginning exercise to improve strength |
| 15–20 repetitions are recommended to improve muscular endurance |
| *Sets* |
| 2–4 sets are recommended to improve strength and power |
| 2 or more sets are effective for muscular endurance improvements |
| *Pattern* |
| 2–3 min rest intervals between each set are effective |
| Rest at least 48 h between sessions for any single muscle group |
| *Progression* |
| Gradual progression of greater resistance, and/or more repetitions or sets, and/or more frequency is recommended |

Sources: Adapted from American College of Sports Medicine. ACSM's guidelines for exercise testing and prescription. 9th ed., Philadelphia: Lippincott Williams & Wilkins, 2013

outlined by the American College of Sports Medicine for healthy adults [8].

Table 20.8 outlines the ACSM Position Stand on Progression Models in Resistance Training for Healthy Adults [16]. Although it is common to estimate intensity on repetition maximum (RM), this should only be used as a general guideline since RM differs between muscle groups [17]. These guidelines are appropriate for healthy adult women who desire goal-oriented guidelines for

**Table 20.8** Overview of American College of Sports Medicine's position stand on progression models in resistance training for healthy adults

| |
|---|
| Concentric, eccentric and Isometric actions must all be included for all training levels |
| Unilateral and bilateral and multiple-joint exercises should be included |
| Sequence of exercise should be: multiple-joint before single-joint, higher-intensity exercises before lower-intensity, rotation of upper and lower body or opposing exercises |
| Novice individuals should train the entire body 2–3 days•week$^{-1}$ |
| Intermediate individuals should train 3 days•week$^{-1}$ total body or 4 days with a lower/upper body split |
| Advanced lifters should train 4–6 days•week$^{-1}$ |
| *Strength training* |
| 1–3 sets of 8–12 repetitions using an intensity of ~60–70 % 1RM is recommended for novice and intermediate individuals |
| Cycling loads of ~80–100 % 1RM is recommended for advanced individuals |
| Rest period of at least 3 min for core exercises |
| *Muscle hypertrophy* |
| For novice and beginning individuals, an intensity range of 70–85 % 1RM should be used for 1–3 sets of 8–12 repetitions with 1–2 min rest between sets |
| For advanced individuals, working at an intensity of 70–100 % 1RM for 1–12 repetitions in 3–6 sets with a 2–3 min rest time in core exercises |
| *Local muscular endurance* |
| Novice and intermediate training should include a relatively light load with a moderate to high volume (10–15 repetitions with about 1 min rest) |
| Advanced training should use various loading with high repetitions (15–25 repetitions or more with a min or less rest) |
| When circuit training, rest intervals should be the time it takes to get from one exercise to the next |
| *Older adults* |
| Use a slow-to-moderate lifting velocity for 1–3 sets of 8–12 repetitions using 60–80 % 1RM with 1–3 min rest |
| Should train 2–3 days•week$^{-1}$ |

Source: American College of Sports Medicine position stand: progression models in resistance training in healthy adults. Med Sci Sports Exerc 2009;41:687–708

athletic performance enhancement, rather than simply health benefits. In order to more fully understand the guidelines as outlined in Table 20.8, the following terms are defined [18, 19].

*Concentric* (*shortening*): Concentric muscle actions occur when the total tension developed in all the cross-bridges of a muscle is sufficient to overcome any resistance to shortening.

*Eccentric* (*lengthening*): Eccentric muscle actions occur when the tension developed in the cross-bridge is less than the external resistance, and the muscle lengthens despite contact between the myosin cross-bridge heads and the actin filament.

*Hypertrophy*: The muscular enlargement that results from resistance is called hypertrophy and is primarily a result of an increase in the cross-sectional area of the existing fibers.

*Multi-Joint exercise*: multi-joint exercises involve two or more primary joints (i.e., front or back squat, bench press, shoulder press).

*Periodization*: Effective program design involves the use of periodization, which is the varying or cycling of training specificity, intensity, and volume to achieve peak levels of conditioning.

*Power*: Power is precisely defined as the "time rate of doing work" [20] where work is the product of the force exerted on an object and the distance the object moves in the direction in which the force is exerted (power = work/time).

*Repetitions*: Repeating an identical movement for a specific number of times. To improve strength, you must do enough repetitions of each exercise to fatigue your muscles. The number of repetitions needed to cause fatigue depends on the amount of resistance. In general, a heavy weight and a low number of repetitions (1–5) build strength, a lightweight and high number of repetitions (15–20) build endurance, for general fitness purposes, 8–12 repetitions are usually recommended.

*Repetition maximum*: The maximum amount of resistance a person can move a specific number of times is referred to as a repetition maximum (RM). The RM indicates that the muscle has reached a point of fatigue in which the force generating capacity falls below the required force to shorten the muscle against the imposed resistance [7]. One RM is the maximum amount of resistance that can be lifted one time, 5 RM is the maximum amount of weight that can be lifted five times.

*Set*: A set refers to a group of repetitions of an exercise followed by a rest period.

**Table 20.9** American College of Sports Medicine guidelines for resistance training with children

| American College of Sports Medicine guidelines for resistance exercise in children and adolescent populations |
|---|
| Children should be supervised by a qualified instructor when performing exercises |
| High intensity exercises such as 1RM should be avoided and progressive loading should be utilized instead |
| Equipment should be appropriate for the size and skill level of the child |
| The goals of the resistance program should work to increase motor skill and fitness level |
| The child should perform each exercise between 8 and 15 repetitions and weight should only be increased when they can perform this number of repetitions with correct form. If 8 repetitions cannot be performed then the resistance weight needs to be lowered so the child can perform the 8 repetitions with correct form |
| Young children should not perform below 8 repetitions. A training load of 8 or below should be utilized only for older adolescents |
| The focus should be on developing correct form rather than maximizing weight |

Source: Adapted from American College of Sports Medicine. ACSM's guidelines for exercise testing and prescription. 7th ed., Philadelphia: Lippincott Williams & Wilkins, 2006

*Single joint exercise*: Single joint exercise involves only one primary joint (i.e., bicep curl).

Even though guidelines for children and adolescents are similar to those for adults, there are specific guidelines for children and adolescents. Guidelines for children and adolescents can be found in Tables 20.9 and 20.10.

For more specific information on developing effective resistance training programs for specific goals, it is recommended that you access on-line resources at http://www.nsca-lift.org/Home/ (National Strength and Conditioning Association), http://www.acsm.org/ (American College of Sports Medicine), or http://www.exrx.net/index.html (Exercise Prescription on the Net). These resources will also provide recommendations for texts and videos. In addition workout templates and live video clips of the proper form for performing resistance exercise can be found at Exercise Prescription on the Net.

**Table 20.10** Kraemer's age specific exercise guidelines for resistance training

| Resistance training exercise for children | |
|---|---|
| Guidelines by age group | |
| 7 years or younger | Use little or no weight<br>Focus on technique<br>Volume should stay low |
| 8–10 years of age | Can increase number of exercises as well as resistance and volume<br>Important to monitor progression and tolerance of increases |
| 11–13 years of age | Continue slow progression of resistance and volume<br>Begin to introduce advanced exercises using little or no weight<br>Add sport specific exercises |
| 14–15 years of age | Continue resistance progression<br>Advance sport specific components |
| 16 years and older | After demonstrating mastery of proper technique the child should be progressed to entry-level adult programs |

Source: Adapted from Kraemer WJ and Fleck SJ. Strength training for young athletes, 2nd ed. Champaign: Human Kinetics, 2005

### 20.3.5 The American College of Sports Medicine's Exercise Recommendations for Healthy Body Composition

Although national standards have been developed and accepted for body mass index and waist circumference, there are no national standards for body fat percentage. Lohman et al. [20] proposed a set of standards for women using data from the National Health and Nutrition Examination Survey. Table 20.11 lists Lohman's et al. [20] body fat percentage recommendations for women.

Exercise programs to optimize lean body mass should include both cardiovascular and muscular fitness exercise components. In accordance with the United States Department of Health and Human Services, the ACSM recommends a target of 1,000 kcal (or range of 5,400–7,900 steps each day) of physical activity and/or exercise a week for energy expenditure [8]. Reports indicate that at the very least >150 min a week may be necessary for weight loss [21]. Greater amounts of physical activity (>250 min•week$^{-1}$) may be needed to promote long-term weight control [8].

**Table 20.11** Recommendations for body fat percentages for women

| Category | | | Recommended percentage | | |
|---|---|---|---|---|---|
| Essential | | | 8–12 % | | |
| Minimal | | | 10–12 % | | |
| Athletic | | | 12–22 % | | |
| Recommended body fat percentage levels for adults and children | | | | | |
| Age (years) | NR | Low | Mid | High | Obese |
| 6–17 | <12 | 12–15 | 16–30 | 31–36 | >36 |
| 18–34 | <20 | 20 | 28 | 35 | >35 |
| 18–34 PA | | 16 | 23 | 28 | |
| 35–55 | <25 | 25 | 32 | 38 | >38 |
| 35–55 PA | | 20 | 27 | 33 | |
| 55+ | <25 | 25 | 30 | 35 | >35 |
| 55+ PA | | 20 | 27 | 33 | |

*NR* not recommended, *PA* physically active
Source: Lohman TG, Houtkooper LB, Going SB. Body fat measurement goes high-tech: not all are created equal. ACSM Health Fitness J 1997; 7:30–35

Physical activity and/or exercise expenditure in excess of 2,000 kcal•week$^{-1}$ (8,000–12,000 steps per day) have been successful for both short- and long-term weight control [8, 22]. Energy expenditure equivalents for activity can be found on the Fitness Partner's Activity Calorie Calculator (see http://www.primusweb.com/fitnesspartner).

### 20.3.6 The American College of Sports Medicine's Exercise Recommendations for Flexibility

Since flexibility is believed to be transient, it is recommended that flexibility exercises should be performed a minimum of 2–3 days a week [8]. The greatest change in flexibility has been shown to be in the first 15 s of the stretch with no significant improvements after 30 s [23]. Therefore ACSM recommends that each stretch be held for 15–30 s. The optimal number of stretches per muscle group is two to four since no significant improvement in muscle elongation is seen in repeated stretching of five to ten repetitions [24]. Table 20.12 lists general exercise guidelines for achieving and maintaining flexibility [8, 25].

**Table 20.12** General exercise prescription for achieving and maintaining flexibility

| |
|---|
| Precede stretching with light aerobic activity or by external methods (ex: hot bath, moist heat packs) to warm up body |
| Perform a minimum of 2–3 days•week$^{-1}$, 7 days•week$^{-1}$ is most effective |
| Stretch to the end of the range of motion to a point of tightness or slight discomfort |
| Hold each stretch for 10–30 s |
| For older adults, holding each stretch for 30–60 s may show more benefits |
| For proprioceptive neuromuscular facilitation (PNF) stretching, a 3–6 s light-to-moderate contraction followed by a 10–30 s assisted stretch is desirable |
| Perform 60 s of total stretching time for each exercise |

Source: American College of Sport Medicine. ACSM's guidelines for exercise testing and prescription. 9th ed., Lippincott Williams & Wilkins: Philadelphia, 2013

**Table 20.13** High-risk flexibility exercises with alternative stretches

| High-risk stretch | Alternative stretch |
|---|---|
| Standing toe touch | Seated toe touch or modified hurdler's stretch |
| Barre' stretch | Seated toe touch or modified hurdler's stretch |
| Hurdler's stretch | Modified hurdler's stretch (bend the knee so that the tibia moves towards the torso rather than away from the torso as in the traditional hurdler's stretch) |
| Neck circles | Non-twisting directional stretch |
| Knee hyperflexion | Kneeling hip and thigh stretch |
| Yoga plow | Seated toe touch |

Source: American College of Sport Medicine. ACSM's guidelines for exercise testing and prescription. 7th ed., Lippincott Williams & Wilkins: Philadelphia, 2006

Table 20.13 lists some common high-risk stretches and safe alternative exercises. The high-risk exercises could be appropriate for certain groups of athletes, for example, ballet dancers would perform stretches using the barre', whereas the alternative stretch would be more suitable for the average female. There are many Web sources to find appropriate flexibility exercises as well as research articles about the effectiveness of flexibility training [25, 26]. The American Academy

of Orthopaedic Surgeons provides examples of flexibility exercises for the young athlete as well as for older individuals (see http://orthoinfo.aaos.org/main.cfm).

## 20.4 Contemporary Understanding of the Issues

### 20.4.1 Alternate Physical Activity and Programs

As noted above, the majority of healthy children, adolescents, and adult women are not engaged in physical activity consistent with public health recommendations. The issue of physical activity becomes even more crucial for "unhealthy" female populations, for example, women with cardiovascular disease, cancer, or eating disorders. In such cases, physical ability may be limited to due to a patient's condition. In such cases, other types of physical activity, namely, *yoga*, may be recommended due to its proliferation and popularity; specifically, 15 million people practice various forms of yoga in the USA and profess advantages that extend well beyond traditional benefits of exercise [27]. Yoga is an ancient tradition designed to bring balance and health to the physical, mental, emotional, and spiritual domains of an individual [28, 29]. This ancient discipline is often represented metaphorically as a tree consisting of eight "limbs" or aspects: yama (universal ethics), niyama (self-discipline), asana (physical postures), pranayama (breath control), pratyahara (control of the senses), dharana (concentration), dyana (meditation), and samadhi (bliss or transcendent meditative awareness) [28, 29].

In contrast to other branches of yoga, the type known as Hatha focuses on the body and fitness [27]. Indeed, what most people refer to as simply "yoga" is hatha yoga, the most popular type practiced in the West [27–29], which emphasizes the exercise or asana component as exemplified by the ACSM's view of yoga as an "alternate type" of exercise [30]. Yoga, however, is inherently holistic or biopsychosocial in nature [29]. The purpose here is to discuss the following: the beneficial health effects of Western yoga (exercise focus) compared to exercise, as well as the benefits from integrated yoga; mindful awareness, mindfulness or mindfulness meditation; and lastly, the role of mindfulness-based interventions in eating disorders.

### 20.4.2 Health Benefits of Westernized Yoga

A growing body of literature indicates that yoga may be another option for improving and maintaining physical and emotional health. A growing body of literature indicates that yoga may be another option. The most commonly practiced hatha yoga in the West emphasizes the exercise component of physical postures or asanas that stretch and strengthen the body's musculature [27]. Numerous research studies and reviews attest to the structural-physiological benefits of yoga, including findings contrary to the belief that static yoga asanas are only equivalent to light intensity exercise; that is, intensive asanas or sequences have been associated with sufficiently elevated metabolic and heart rate responses to improve cardiorespiratory fitness [31]. In addition to structural–physiological benefits, musculoskeletal–cardiopulmonary and endocrine and autonomic nervous system benefits of yoga are documented [28, 29, 32].

A growing literature supports the notion that certain yoga techniques may improve physical and mental health in regard to stress through down-regulation of the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous system (SNS) [28]. The HPA axis and SNS are triggered responses to a physical and/or psychological demand (stressor) that leads to a cascade of physiological, psychological, and behavioral effects primarily due to the release of cortisol and catecholamines. Over time, constant hypervigilence from repeated firing of the HPA axis and the SNS can lead to system dysregulation and conditions such as obesity, diabetes, autoimmune disorders, depression, substance abuse, and cardiovascular disease [33, 34]. While these studies suggest that yoga has an immediate quieting effect on the SNS/HPA axis responses to stress, the precise mechanism of action has not

been determined. It has been hypothesized, however, that some yoga exercises, or asanas, cause a shift toward parasympathetic nervous system dominance, possibly via direct vagal stimulation [35].

Yoga is associated with rapid stress reduction and anxiolysis, significant decreases in heart rate, systolic and diastolic blood pressure [36–39], as well as increases heart rate variability (HRV) [35]; this is of import because an increase in HRV is associated with healthy functioning and a reduced risk of heart disease [29]. Likewise, Shapiro and colleagues found significant reductions in low-frequency heart rate variability (HRV)—a sign of sympathetic nervous system activation—in depressed patients following an 8-week yoga intervention [40]. Data also show that yoga decreases levels of salivary cortisol [36, 41], blood glucose [42, 43] as well as plasma renin levels, and 24-h urine norepinephrine and epinephrine levels [37]. Furthermore, research suggests that yoga reverses the negative impact of stress on the immune system by increasing levels of immunoglobulin A and natural killer cells [44, 45]. Yoga decreases markers of inflammation such as high sensitivity C-reactive protein and inflammatory cytokines such as interleukin-6 and lymphocyte-1B [46, 47].

In terms of specific disease processes, several literature reviews have been conducted that examine the impact and benefits of yoga on physical and mental conditions including cardiovascular disease [32], metabolic syndrome [48], cancer [49], pain [50], depression [51], and anxiety [52]. Galantino and colleagues [53] published a systematic review of the effects of yoga on children, which is not surprising, given a recent complementary and alternative medicine (CAM) study investigating CAM preferences among children at a tertiary pain clinic where yoga was ranked among the three most popular pain treatments [54]. These reviews have contributed to an ever growing literature supporting the positive health benefits of yoga, especially in the context of chronic illness; that is, from a basic physical activity perspective, yoga may be particularly suited for individuals who have a chronic health disease or condition [29].

Two more research reviews regarding the benefits of yoga have recently been reported [27, 28]. Results from a recent review of studies comparing health benefits of yoga as an alternate exercise regimen to traditional exercise concluded that in both healthy and diseased populations, yoga may be as effective—or better than exercise—at improving a variety of health-related outcomes. In particular, yoga was deemed to be equal or *superior* to exercise in relieving certain symptoms associated with diabetes, multiple sclerosis, menopause, kidney disease and schizophrenia [28]. Recall that the westernized exercise-focused yoga previously discussed stands in contrast to the more holistic or integrated hatha yoga discipline of which exercise is one among several components involved (breath control, meditation, spiritual, and ethical components) [27]. Smith and colleagues wanted to compare the physical and psychological benefits of an exercise-based yoga practice to that of a more holistic or integrated yoga practice. Eight-one undergraduate students were recruited for one of three conditions over a period of 7 weeks with 2 sessions each week: yoga as exercise; integrated yoga including exercise, breath work, meditation, and spiritual/ethical teaching components; and a control group that only met to fill out questionnaires. Participants were assessed on measures of depression and anxiety, hope, stress, flexibility and salivary cortisol. Results showed that both groups' scores decreased significantly on depression and stress as well as increased flexibility and hopefulness compared to the control group. However, only the integrated yoga group demonstrated significant decreases in anxiety symptoms and levels of salivary cortisol; thus, the participants in the holistic yoga intervention experienced benefits above and beyond yoga-as-exercise [27].

Evans and colleagues, in applying the biopsychosocial model to understand the health benefits of yoga, propose that yoga not only shares many of the physical and psychological benefits of exercise—but additionally also provides effects beyond regular exercise in that yoga appears to produce homeostasis across multiple domains of individual functioning and physiological pathways [29].

## 20.5 Future Directions

### 20.5.1 Mindful Awareness and Eating Disorders Premise

Yoga is associated with mindful awareness [29], an openness or receptivity to being aware of, as well as being in…the present moment…and the combination of both yoga and mindfulness is exemplified in the well-known mindfulness-based stress reduction (MBSR) program. Indeed, this psychosocial intervention is a structured therapeutic intervention that combines mindfulness meditation with Hatha yoga [55]. Awareness that a problem, such as an eating disorder, exists is the first key toward change. This leads to the next crucial step in the process of change, that of acceptance, which is an aspect of mindfulness meditation. Mindfulness involves non-judging awareness of each present moment with acceptance of reality as it is—including a *lack of denial* as to our own personal problems and issues. Thirdly, the basic level or component to any process of change is that of behavior, such as replacing a negative behavior with a positive or healthy one. The MBSR exemplifies all three aspects in its psychosocial program by the sheer fact that the heart of mindfulness is self-regulation, that is, holistic self-regulation of emotions, cognitions, and behaviors.

Eating disorders are associated with serious health problems and consequences if individuals do not receive effective treatment. This coincides with the fact that this type of disorder is a multidimensional, complex, and biopsychosocial condition that is viewed as involving major deficits in self-regulation, affect, behavior, and cognitive patterns (distortion) surrounding the focus on and intake of food [56–58]. Thus, mindfulness-based interventions with self-regulation as its core appears clinically appropriate as a treatment for those with disordered eating. Eating disorders can be a major challenge to treatment; for example, in a 5-year study of 2,881 women with bulimia, a high relapse rate of 45 % resulted. Such findings provoke the notion of eating disorders being likened to a chronic disease with periodic remissions [59].

The chaotic absence of self-structural competence (i.e., dysregulation) in eating disorders may connect to a desperate sense of insecurity in the face of threat or challenge; understandably, this leads to extreme [eating-related] behaviors to calm the storm and reduce distress. Of course, the calm does not last for long.

To be fully present with that which distresses one to the core, a genuine trusting of something is needed. Meditation provides the structure and the process for that trust. Mindful, single-pointed attention to the present moment anchors and centers self to a regulating structure and process that merits trust; and, equally as important, it strips away the layers of biased meaning and subjective value judgments to which one is attached that causes distress and suffering in the first place. From this vantage point of stripped and raw experience, things are seen simply as they are, in a neutral and dispassionate process of objective witnessing. Thus, one comes to trust self as a calm and safe container of objective reality—a reality that is experienced one bite of food one moment at a time, which helps to reduce feelings of being overwhelmed.

In sum, a substantial body of literature over the last several decades indicates that the basic deficits in eating disorder populations may be due to ineffective self-regulation in multiple domains [60–62]. Eating disordered groups have problems with affect or emotional regulation, that is, they have difficulty in identifying or being aware of emotional states as well as managing emotions or skillfully modulating feelings so as to be adaptive. As previously mentioned, mindfulness is a self-regulatory process, and mindlessness may be viewed as a component in some eating disorders, for example, binge eating and obesity, and likely plays some role in unsuccessful long-term weight loss programs.

### 20.5.2 MBSR Foundations, Basic Strategies and Structure

Meditation is a unique kind of attention. Meditation can be described as the self-regulation of attention and awareness that immerses the individual into

the fullness of psycho-physiological experience. Basically, meditation is the disciplined practice of paying attention to the present moment with uncritical or non-judging acceptance. This alert, yet calm attentiveness is practiced in two basic forms: exclusive/concentrative meditation or inclusive/mindfulness meditation Exclusive or concentrative meditation, exemplified by transcendental meditation or TM, emphasizes concentration where the individual focuses on an internal or external object (such as a mantra) while minimizing distractions of other stimuli.

Mindfulness, as taught by Kabat-Zinn, is an example of inclusive meditation that involves including rather than excluding stimuli from the field of consciousness [63]. This type of meditation is more reality-based with daily life because we are constantly challenged to pay attention while being bombarded with multiple, competing and distracting stimuli. Mindfulness encourages detached non-judging observation or witnessing of thoughts, perceptions, sensations, and emotions. This provides a means of self-monitoring and self-regulating one's arousal with detached awareness. Where the brain is an animate object that can be measured and touched, the mind is formless and without boundaries [55, 64]. Mindfulness meditation redirects the mind to a level of higher awareness. Individuals are taught to keenly observe their thoughts and emotions and then to let them pass without judging them or becoming immersed in them [63].

A simple analogy is watching the sky on a clear day. Clouds and birds will move across the sky, but they will not stay. Similarly, negative thoughts will enter the mind but mindfulness reorients the individual to the present and broadens self-awareness allowing negative thoughts to pass like the clouds through the sky [65]. Added benefits of the MBSR come from mindfulness meditation being used in conjunction with hatha yoga [55]. An underlying concept of Hatha yoga is that the mind is focused on the asana so it cannot be occupied with distracting thoughts [66]. Additionally, yoga provides the added benefit of giving people with physical illnesses some degree of control over their bodies [66].

The clinically driven MBSR, rooted in Theravada Buddhism and westernized by Kabat-Zinn, was developed originally as the Stress Reduction and Relaxation Program (SRRP) in the Stress Reduction Clinic, at University of Massachusetts Medical Center in 1979 [63]. From modest beginnings at one clinic, this therapeutic intervention has been completed by more than 15,000 patients at the original site alone, not including participants in over 250 MBSR programs around the world at last count in 2004 [67].

The traditional MBSR involves participants meeting once per week for a period of 8 weeks for approximately 2 h. Mindfulness-based interventions include didactic, inductive, and experiential modes of learning about stress responses and mindfulness skill development training. The participants received training in three basic mindfulness practices: the body scan, sitting meditation and hatha yoga. The body scan is a guided and experiential journey through the physical geography of the body from feet to head, while paying attention to whatever feelings and sensations—or lack of sensations—that arise in regions of the body. Mindfulness includes an observational, non-judging attitude with acceptance of ourselves as we are, without harsh criticism; thus, this technique encourages one to get acquainted or reacquainted with his/her body just as it is in the present moment.

Sitting meditation involves mindful attention of the breath, which serves as a focal anchor, with a heightened state of observational yet non-judging awareness of cognitions and the stream of thoughts and distractions that constantly flow through the mind. Hatha yoga, meditation-in-motion, involves stretches and postures (asanas) designed to strengthen and relax the musculoskeletal system and develop mindful movement of the body with essential harmonic regulation of breathing. Participants are given tapes or CDs to facilitate daily homework practice of the techniques learned from the weekly sessions [63].

### 20.5.3 Mindfulness General Research Findings

For over 30 years, abundant research documents the significant benefits of mindfulness interventions across a wide range of populations with various physical and psychological conditions. For example, results of recent mindfulness-based intervention studies continue to find improved levels of various forms of psychological distress such as anxiety, depression, prenatal stress, worry, and rumination [68–72]. Mindfulness-based skills also have been found to be beneficial in conjunction with more serious underlying mental conditions such as anxiety in schizophrenic patients, acute psychotic symptoms, and bipolar and borderline personality disorders [73–76]. Also, empirical evidence from a mindfulness-based relapse prevention program (MBRP), developed by Marlatt's group at the Addictive Behaviors Research Center at the University of Washington, shows promising benefits for addiction populations [77].

Current research also documents the efficacy of mindfulness-based skills training in reducing stress-related symptoms and emotions in those suffering with chronic conditions.

Studies among different cancer groups have documented improved coping, quality of life as well as rapid immune function recovery with lower cortisol levels post-intervention [78, 79].

Additional findings provide evidence as to medical benefits of mindfulness interventions in symptoms associated with the following conditions: attention-deficit hyperactivity disorder (ADHD), chronic fatigue syndrome, chronic heart failure and myocardial ischemia, chronic pain fibromyalgia, HIV, insomnia, rheumatoid arthritis, and Type 2 Diabetes Mellitus [55, 68, 70, 80–84].

### 20.5.4 Mindfulness: Related Programs for Eating Disorders

A growing body of research suggests that mindfulness (i.e., non-judgmental, present-moment awareness) and its related constructs are relevant to understanding the development and maintenance of eating disorders [85]. Anorexia nervosa and bulimia nervosa are both characterized by experiential avoidance and a strong desire to maintain control over eating-related behaviors, urges, thoughts, and feelings [86–88]. Eating disorder behaviors may be reinforced in part because they allow individuals to temporarily avoid other distressing internal experiences by focusing instead on one's weight or eating behavior [89, 90]. Many individuals with eating disorders also have deficits in emotion recognition and emotional awareness [91, 92]. Recognition and awareness of internal experience may be a precondition to cognitive defusion, which is the ability to have distance and perspective from the literal meaning of cognitive activity [87]. More specifically, a small number of case studies and pilot studies suggest that mindfulness and acceptance might be effective foci of treatment for eating disorders [93–97].

Mindfulness is recognized as a vital component of the several third generation behavioral therapies, including Dialectical Behavior Therapy (DBT), Acceptance and Commitment Therapy (ACT), Mindfulness-based Cognitive Therapy (MBCT), and Mindfulness-Based Eating Awareness Training (MB-EAT) [98]. Since these interventions draw heavily from clinical mindfulness-based conceptualization, they will now be briefly reviewed.

According to Wolever and Best [99], Dialectical Behavior Therapy (DBT) has the distinction of being the most extensively studied mindfulness approach within eating disorder populations. DBT originally was implemented to improve dysregulation deficits in those with borderline personality disorder in the early 1990s [100], and since then has been expanded by Telch and colleagues into an 18-session treatment program for those with BED [101, 102]. The underpinning for DBT here involves an affect dysregulation perspective where binges are viewed as serving the maladaptive function of attempts at reducing distressing emotional states with poor strategies. Mindfulness-based

skills of awareness of the present moment with uncritical or nonjudgmental acceptance—being with what is happening rather than reacting and trying to change things—is the bedrock for teaching affect regulation and distress tolerance building strategies. The mindful, uncritical acceptance of fearful thoughts or images as being just that helps clients be aware of their emotions without the reactive behavioral automaticity of a binge eating event.

Both uncontrolled and randomized controlled trials (RCT) have shown significant and positive results. In the uncontrolled trial, 82 % of the sample were binge-free at post-intervention with follow-ups at 3-months (80 %) and 6-months (70 %). The RCT results at post-intervention revealed 89 % abstinence for the treatment group compared to 12.5 % of the control group; at 6-month follow-up, however, modest sustained abstinence was found for the treatment group (56 %) [101, 102].

Another mindfulness-based intervention is that of Acceptance and Commitment Therapy (ACT), based on an avoidance model, which asserts that some types of disorganized behavior are associated with efforts to escape distressing internal-external experiences, feelings, urges, etc. [103, 104]. This therapeutic approach holds promise in that it exposes individuals to the exact stimuli from which they chaotically attempt to avoid, which helps fuel the inflexible and vicious cycle of extreme behavior; however, the confrontation is done from a mindful accepting mindset. The ACT program has been applied notably to anorexia nervosa with publication of a self-help workbook [105]. ACT emphasizes uncritical acceptance of thoughts and feelings while working toward behavior change [103] and incorporates several mindfulness and acceptance strategies such as the *thought parade*. This mindfulness technique involves an individual imagining self's thoughts as being written on cards or plackards carried by people in a parade, for example, "I'm as big as a whale" or "I look like Shamu." The task is to be a witness to one's thoughts in an uncritical and accepting mindset rather than automatically—and mindlessly—engage in an unregulated maladaptive eating reaction to counter distressing thoughts.

Theoretically and therapeutically, ACT therapy for eating disorders is an up and coming approach. Future empirical studies and clinical trials in this line of therapy will be a promising source of clinically scientific literature for this population.

The first mindfulness-based program developed specifically to treat an eating disorder, specifically, that of binge eating, is Kristeller's Mindfulness-Based Eating Awareness Training (MB-EAT), which has evolved into a 12-week program [96, 106–108]. This intervention's approach is based on the view of eating disorders as related to pervasive underlying dysregulation patterns of affect, cognition, behavior, and physiology. MB-EAT combines mindfulness with cognitive therapy and imagery. A scientific approach fosters intake regulation, that is, biological cues of hunger and satiety, and appetite awareness training. The MB-EAT applies strategies from Kabat-Zinn's [63] original program and mindfulness is integrated daily with regard to food craving and eating. Participants experience several guided eating meditations that involve nonjudgmental awareness of thoughts, emotions, and sensations relating to hunger-satiety cues as well as binge triggers. Gradually, the intervention guides participants in the process of mindfulness-based healthy food selection, and culminates with later weeks shifting towards deeper levels of cultivating forgiveness and inner wisdom.

### 20.5.5 Recent Mindfulness and Eating Disorder Research

Mindfulness-based approaches are being used increasingly to treat eating disorders. Indeed, mindfulness is appropriate for eating disorder populations because many patients, as touched upon earlier, have difficulties with regulation—regulating emotional, cognitive, and physical experiences [109]. Lavander, Jardin, and Anderson [110] found that non-eating disordered individuals who exhibited higher levels of dispositional mindfulness were less likely to engage in

disordered eating behaviors. Other findings indicate that mindfulness-based skills offered to young women early in their psychosocial development may aid in preventing eating disorders [111]. Mindfulness-based interventions, in combination with the development of emotion regulation and distress tolerance skills, appear to be effective in managing the urge to binge eat when it arises [112]. Additionally, there is growing interest in developing mindfulness treatments for the specific types of eating disorders, i.e., anorexia nervosa [113], bulimia nervosa [114], and binge eating [107].

According to Butryn and colleagues [85], very little data have been collected to examine whether improvements in mindfulness and it related constructs such as awareness, acceptance, and cognitive defusion, are related to symptom severity and improvement in eating disorder symptoms. Thus, Butryn [85] investigated mindfulness and its relationship to eating disorders symptomatology in 88 women with multiple eating disorder diagnoses in residential treatment. Multiple measures of eating disorder symptomatology, as well as a mindfulness scale, were administered to the participants upon admission and again at discharge. Baseline eating disorder (ED) symptomatology was associated with lower awareness, acceptance, and cognitive defusion, and higher emotional avoidance. Improvements in these variables were related to improvement in ED symptomatology. It was concluded that interventions targeting mindfulness are beneficial to improving eating disorder symptomatology [85]. This generally coincides with findings from a systematic review of eight studies of mindfulness-based interventions in eating disordered participants [98]. Supporting evidence was found for the effectiveness of mindfulness-based interventions for the treatment of eating disorders; however, it was noted that trial quality of the studies was variable with small sample sizes; thus, more and larger trials were recommended. It was concluded that the application of mindfulness-based interventions to eating disorders is a promising approach worthy of further research [98].

The burgeoning literature indicating the efficacy of mindfulness-based interventions for clinical populations has lead to interest in mindfulness skills as a potential adjunct to long-term treatment for eating disorders. Specifically, most mindfulness-based research studies have examined brief group interventions independent of continued or long-term treatment [115].

Consequently, Hepworth [115] conducted an exploratory study to investigate the potential benefits of a "mindful eating group" for individuals with various types of eating disorders in conjunction with long-term treatment at a treatment facility specializing in eating disorders.

Participants were selected based on the following: their level of progress in treatment; a BMI $\geq$ 17; self-reported improvement in mood; and binging/purging purging behaviors of less than once per day—that is, once every couple of days. Potential participants were excluded if they had a current BMI $<$ 17 (since low weight affects cognitive abilities), and if they were diagnosed with major depression as assessed by their psychologist. The concluding sample for this 10-week pilot study ended up being 33 females (mean age of 21.42 years), representing a variety of eating disorders. A trained psychologist and dietitian facilitated the ten mindful eating intervention sessions, that is, one session each week. Disordered eating symptoms were measured by the Eating Attitudes Test-26 (EAT-26) pre-and-post the 10-week program [115].

Results showed significant reductions on all scales of the EAT-26, along with large effect sizes; also, no significant differences were found between eating disorder diagnoses. Results from this pilot suggest potential benefits from an adjunct mindfulness-based intervention for those con-currently undergoing long-term individual treatment [115].

As mentioned previously, the first mindfulness-based program developed in 1999 specifically to treat an eating disorder—binge eating disorder or BED—was Kristeller's Mindfulness-Based Eating Awareness Training (MB-EAT) [106]. This persistent and consistently expanding intervention has evolved into a 12-week program [96, 106–108].

MB-EAT incorporates the clinical value and research of mindfulness, food intake regulation

literature, and emotional dysfunction in binge eating and other disordered eating populations [63, 116, 117]. More specifically, traditional mindfulness meditation as well as guided meditation practices are included to address eating-related self-regulatory processes including emotional versus physical hunger triggers, gastric and sensory-specific satiety or fullness (SSS), food choice, and emotional regulation pertinent to self-concept and stress management [108].

Emergent wisdom and self-acceptance are core components of the program; that is, individuals are encouraged to recognize their own internal strengths and be open to their own understanding and salutations to challenging situations rather than reacting judgmentally to self-perceived variances from internalized norms [107].

Research continues to demonstrate the effectiveness of this program in treating BED [96, 106–108]. For example, this year, Kristeller and colleagues [108] conducted a randomized trial to explore the efficacy of a 12 session MB-EAT, in comparison to a psychoeducational–cognitive–behavioral intervention (PECB) and a wait list control. The two-site study randomized 150 participants. Compared to the wait list control, MB-EAT and PECB showed generally comparable improvement after 1 and 4 months post-intervention on binge days per month, the Binge Eating Scale, and depression. At 4 months post-intervention, 95 % of those individuals with BED in MB-EAT no longer met the BED criteria versus 76 % receiving PECB; furthermore, binges that occurred were likely to be significantly smaller. Also, the amount of mindfulness practice predicted improvement on a range of variables, including weight loss. In conclusion, results indicate that MB-EAT decreased binge eating and related symptoms at a clinically meaningful level, with improvement related to the degree of mindfulness practice [108].

Recently, ongoing research suggests expanding the MB-EAT so that it can be adapted to also address weight loss—without losing its effectiveness for treating associated symptoms of binge eating. Indeed, a recently completed MB-EAT clinical NIH trial broadened its recruitment to include those with a BMI of at least 35. Preliminary data analyses indicate that BED participants showed comparable improvement to those without BED—including a weight loss of 7 lbs. at immediate post [107]. Recently, elements of the MB-EAT program have been successfully adapted for use with restaurant meals, showing weight loss and improved dietary patterns in perimenopausal women [118]. In sum, one aspect of this programs' continuing and evolving effectiveness may well be its adaptability among different disorders. More information regarding the MB-EAT program can be found at its Web site, The Center for Mindful Eating, at http://www.tcme.org/.

## 20.6 Concluding Remarks

Despite the documented benefits of regular physical activity, the majority of children, adolescents, and adult women are not engaged in physical activity consistent with public health recommendations. At one end of the continuum, we have young girls and female athletes who are not taking in enough calories to meet their energy needs associated with exercise, and at the other end of the continuum, we have the vast majority of children, adolescents, and adult women who do not get enough physical activity.

Empirical literature about yoga and mindfulness has been presented. Additionally, the benefits of yoga and mindfulness-based programs have been reviewed as well as research regarding mindfulness incorporated programs specifically in the area of eating disorders. Future research, no doubt, will continue to inform and expand scientific knowledge as to the clinical utility of mindfulness-based interventions in eating disordered populations as well as in other health populations.

There are many resources on the Web to assist with activity and exercise recommendations to achieve optimum health and fitness. The Department of Health and Human Resources Centers for Disease Control and Prevention offers a Web site that provides information to assist in healthy physical activity program

planning and evaluation as well as ideas for healthy physical activity promotion (see http://www.cdc.gov/physicalactivity/index.html).

## 20.7 Appendix 1. Determining Moderate and Vigorous Exercise Intensity Using the Heart Rate Reserve Method (HRR)

STEP 1

Take your resting heart rate. In order for this to be the most accurate, it is suggested that you count the number of beats per minute three mornings in a row before arising and average the three readings.

Reading 1_____bpm Reading 2_____bpm Reading 3_____bpm Average bpm_____

Resting heart rate = _____*(i.e., resting heart rate =72)*

STEP 2

Determine your maximum heart rate (MHR) 220 − age = _____*(i.e., 220 − 35 = 185)*

***Moderate Exercise Intensity***

STEP 3

Target Heart Rate = [(MHR-resting heart rate) (40–59 %)] + resting heart rate

i.e., [(185 − 72) (40–59 %)] + 72 = 117–138 bpm

***Vigorous Exercise Intensity***

STEP 3

Target Heart Rate = [(MHR-resting heart rate) (≥60 %)] + resting heart rate

i.e., [(185 − 72) (≥60 %)] + 72 = 139 bpm or greater

Note: The intensity range to increase and maintain cardiorespiratory fitness (CR) is broad. For individuals with low levels of CR fitness, the lower range will result in improvements in CR fitness. For individuals who are already fit, exercise intensities at the high end of the continuum (60 % to <90 % HRR) may be needed to improve and maintain CR fitness.

## 20.8 Appendix 2. Determining Moderate and Vigorous Exercise Intensity Using the Borg Rating of Perceived Exertion (RPE) Scale

| | |
|---|---|
| _____ | **13 Somewhat hard** |
| **6 No exertion at all** | **14** |
| **7** | **15 Hard (heavy)** |
| **Extremely light (7.5)** | |
| **8** | **16** |
| **9 Very light** | **17 Very hard** |
| **10** | **18** |
| **11 Light** | **19 Extremely hard** |
| **12** | **20 Maximal exertion** |

*Instructions*: While doing physical activity, rate your perception of exertion. This feeling should reflect how heavy and strenuous the exercise feels to you, combining all sensations and feelings of physical stress, effort, and fatigue. Do not concern yourself with any one factor such as leg pain or shortness of breath, but try to focus on your total feeling of exertion. Choose the number that best describes your level of exertion. This will give you a good idea of the intensity level of your activity. Moderate exercise is defined as a "perceived exertion" of **11–14**. Vigorous exercise is defined as a "perceived exertion" of **15 or greater**. The average RPE range associated with physiologic adaptation is **12–16**. However, there is significant interindividual variability.

Note:

**9** corresponds to "very light" exercise, for a healthy person, it is like walking slowly at his or her own pace

**13** on the scale is "somewhat hard" exercise, but it still feels OK to continue.

**17** "very hard" is very strenuous, a healthy really has to push himself or herself and you will probably feel very tired

**19** on the scale is an extremely strenuous exercise level, for most people, this is the most strenuous exercise they have ever experienced

Borg RPE scale © Gunnar Borg, 1970, 1985, 1994, 1998, Division of Nutrition and Physical Activity, National Center for Chronic Disease Prevention and Health Promotion http://www.cdc.gov/nccdphp/dnpa/physical/measuring/perceived_exertion.htm

## 20.9 Appendix 3. Sample Exercise Resistance Program for Postmenopausal Women: 4, 6, 8, and 12-Week Programs

A safe and effective training load for postmenopausal women is from 50 % to 80 % of a 1 RM. This level is enough to allow for the development of strength but light enough to reduce the risk of injury. Progress gradually by starting off with 50 % 1 RM using 2 sets of 6 repetitions for the first 2 weeks. During weeks 3 and 4 increase the lower body intensity by 10 % and the upper body intensity by 5 % as well as increasing the reps to 2 sets of 7 repetitions. When reaching weeks 5 and 6 make similar increases in upper and lower body intensity by increasing intensity by 5 % and 10 % respectively as well as increasing the repetitions in each set to 8. Weeks 7 and 8 have increasing intensity but the repetitions are not increased. Finally, for those who have completed the 8th week, the following weeks have an increased the intensity to 70–80 % of the 1RM for all exercises as well as an additional set of repetitions. The 4-week program is intended for as an introduction to resistance training that can then progress to the 6, 8, and 12 week programs for beginners, intermediates, and advanced exercisers respectively. All programs are performed 3 times per week.

Introduction part I: weeks 1 and 2

| Exercise | 1 RM (lbs) | % 1 RM | Weight (lbs) | Set one | Set two |
|---|---|---|---|---|---|
| Wall squats | 10 | 50 | 5 | 6 | 6 |
| Machine bench press | 20 | 50 | 10 | 6 | 6 |
| Leg press | 100 | 50 | 50 | 6 | 6 |
| Low rows | 40 | 50 | 20 | 6 | 6 |
| Lat pulldown | 40 | 50 | 20 | 6 | 6 |
| Back extension | 60 | 50 | 30 | 6 | 6 |

Introduction part II: weeks 3 and 4

| Exercise | 1 RM (lbs) | % 1 RM | Weight (lbs) | Set one | Set two |
|---|---|---|---|---|---|
| Wall squats | 10 | 60 | 6 | 7 | 7 |
| Machine bench press | 20 | 55 | 12 | 7 | 7 |
| Leg press | 100 | 60 | 60 | 7 | 7 |
| Low rows | 40 | 55 | 22.5 | 7 | 7 |
| Lat pulldown | 40 | 55 | 22.5 | 7 | 7 |
| Back extension | 60 | 55 | 32.5 | 7 | 7 |

Beginner: weeks 5 and 6

| Exercise | 1 RM (lbs) | % 1 RM | Weight (lbs) | Set One | Set Two |
|---|---|---|---|---|---|
| Wall squats | 10 | 70 | 7 | 8 | 8 |
| Machine bench press | 20 | 60 | 12.5 | 8 | 8 |
| Leg press | 100 | 70 | 70 | 8 | 8 |
| Low rows | 40 | 60 | 25 | 8 | 8 |
| Lat pulldown | 40 | 60 | 25 | 8 | 8 |
| Back extension | 60 | 60 | 35 | 8 | 8 |

Intermediate: weeks 7 and 8

| Exercise | 1 RM (lbs) | % 1 RM | Weight (lbs) | Set one | Set two |
|---|---|---|---|---|---|
| Wall squats | 10 | 80 | 8 | 8 | 8 |
| Machine bench press | 20 | 70 | 15 | 8 | 8 |
| Leg press | 100 | 80 | 80 | 8 | 8 |
| Low rows | 40 | 65 | 27.5 | 8 | 8 |
| Lat pulldown | 40 | 65 | 27.5 | 8 | 8 |
| Arm curl | 20 | 70 | 15 | 8 | 8 |
| Arm extension | 40 | 70 | 27.5 | 8 | 8 |
| Back extension | 60 | 65 | 40 | 8 | 8 |

Advanced: weeks 9, 10, 11, and 12

| Exercise | 1 RM (lbs) | % 1 RM | Weight (lbs) | Set one | Set two | Set three |
|---|---|---|---|---|---|---|
| Wall squats | 10 | 80 | 8 | 8 | 8 | 8 |
| Machine bench press | 20 | 70 | 14 | 8 | 8 | 8 |
| Leg press | 100 | 80 | 80 | 8 | 8 | 8 |
| Low rows | 40 | 70 | 28 | 8 | 8 | 8 |
| Lat pulldown | 40 | 70 | 28 | 8 | 8 | 8 |
| Arm curl | 20 | 75 | 15 | 8 | 8 | 8 |
| Arm extension | 40 | 75 | 30 | 8 | 8 | 8 |
| Back extension | 60 | 70 | 42 | 8 | 8 | 8 |

Sample resistance exercises for women

Wall squats

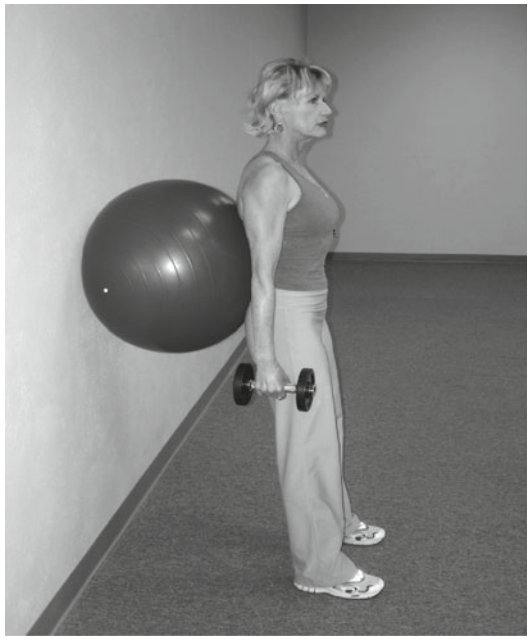
Start position

End position

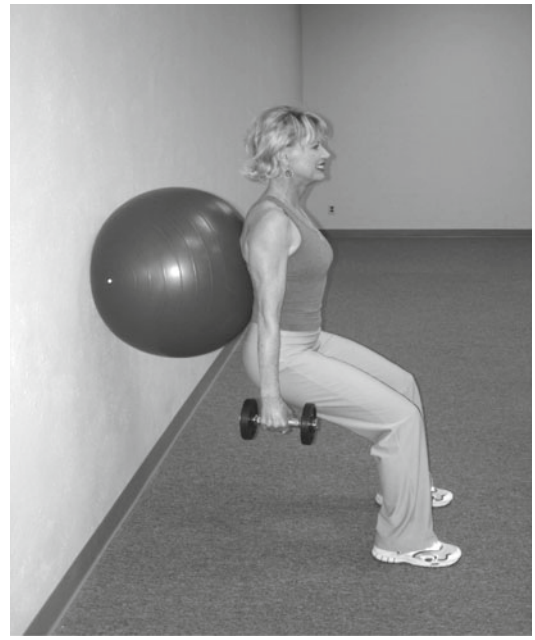
Wall squats with ball

Start position (front view)

End position (front view)



Start position (side view)



End position (side view)

Machine chest press

Start position

End position

Dumbbell arm curl

Start position

End position

Machine arm curl

Start position

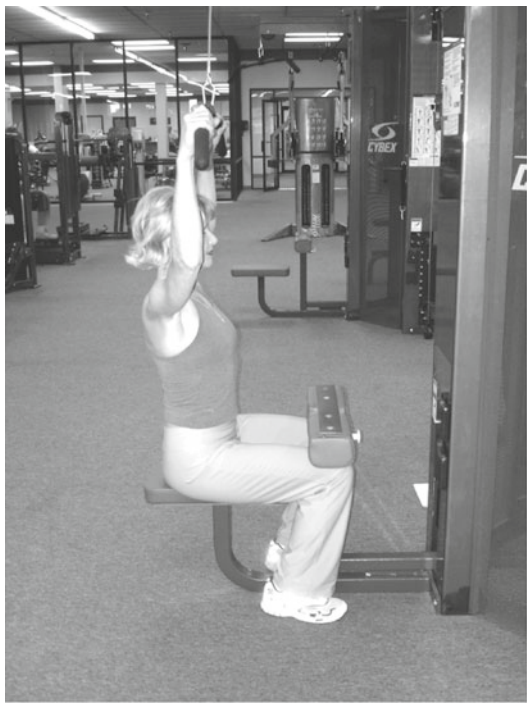
End position

Arm extension

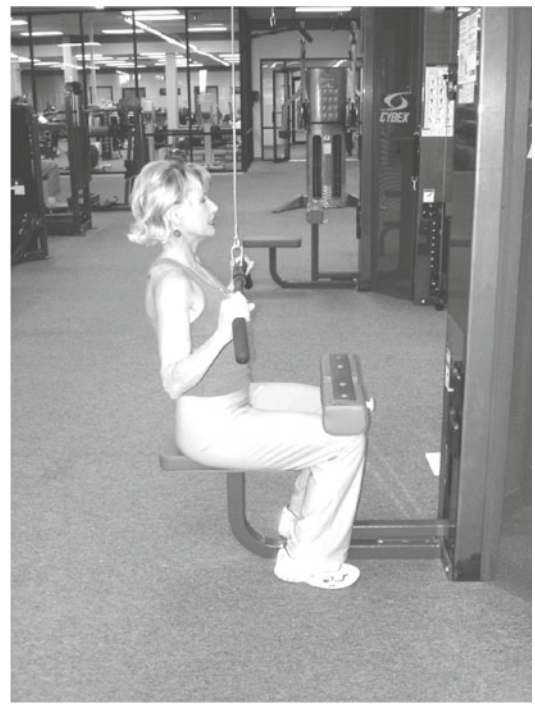

Start position


End position

Lat pulldown



Start position



End position

Back extension


Start position


End position

Leg press

Start position

End position

Low row

Start position

End position

## References


1. U.S. Department of Health and Human Services. Physical activity and health a report of the surgeon general. Atlanta, GA: Centers for Disease Control and Prevention (CDC), National Center for Chronic Disease Prevention and Health Promotion; 1996.
2. Franco O, De Laet C, Peeters A, et al. Effects of physical activity on life expectancy with cardiovascular disease. Arch Intern Med. 2005;165:2355–60.
3. United States Department of Health and Human Services. Healthy people 2010: understanding and improving health. 2nd ed. Washington, DC: U.S. Government Printing Office; 2000.
4. United States Department of Health and Human Services. Healthy people 2020: understanding and improving health. 2nd ed. Washington, DC: U.S. Government Printing Office; 2010.
5. Haskell WL, Lee I, Pate R, et al. Physical activity and public health. Updated recommendation from the American College of Sports Medicine and American Heart Association. Med Sci Sports Exerc. 2007;39(8):1423–34.
6. Ainsworth B, Haskell WL, Whitt MC, et al. Compendium of physical activities: an update of activity codes and MET intensities. Med Sci Sports Exerc. 2000;32(9):S498–516.
7. Ainsworth B, Haskell WL, Herman SD, et al. Compendium of physical activities: a 2nd update of activity codes and MET values. Med Sci Sports Exerc. 2011;43(8):1575–81.
8. American College of Sport Medicine. ACSM's guidelines for exercise testing and prescription. 9th ed. Philadelphia: Lippincott Williams & Wilkins; 2013.
9. Falk B, Tenenbaum G. The effectiveness of resistance training in children. A meta-analysis. Sports Med. 1996;22:176–86.
10. Hurley BF, Hagberg JM, Goldberg AP, et al. Resistance training can reduce coronary risk factors without altering VO2max or percent body fat. Med Sci Sports Exerc. 1988;20:150–4.
11. Kerr D, Morton A, Dick I, et al. Exercise effects on bone mass in postmenopausal women are site-specific and load-dependent. J Bone Miner Res. 1996;11:218–25.
12. Nelson M, Wernick S. Strong women stay young. New York: Bantam Books; 2000.
13. American College of Sport Medicine. ACSM's guidelines for exercise testing and prescription. 7th ed. Philadelphia: Lippincott Williams & Wilkins; 2006.
14. Faigenbaum A, Pollock ML, Ishida Y. Prescription of resistance training for health and disease. Med Sci Sports Exerc. 1996;28:1311–20.
15. Keteyian S, Brawner C. Cardiopulmonary adaptations to exercise. In: Kaminsky LK, editor. ACSM's resource manual for guidelines for exercise testing and prescription. 5th ed. Philadelphia: Lippincott Williams & Wilkins; 2006. p. 313–24.
16. American College of Sports Medicine Position Stand. Progression models in resistance training in healthy adults. Med Sci Sports Exerc. 2009;41:687–708.
17. Hoeger WW, Barette SL, Hale DR. Relationship between repetitions and selected percentages of one repetition maximum. J Appl Sport Sci Res. 1987;1(1):11–3.
18. Dragenich LF, Jarger RT, Kradji AR. Coactivation of hamstring and quadriceps during extension of the knee. J Bone Joint Surg. 1989;71(7):1075–81.
19. Baechle T, Earle R. Essentials of strength training and conditioning. 3rd ed. Champaign, IL: Human Kinetics; 2008.
20. Lohman TG, Houtkooper LB, Going SB. Body fat measurement goes high-tech: not all are created equal. ACSM Health Fitness J. 1997;7:30–5.

21. American College of Sports Medicine Position Stand. Appropriate intervention strategies for weight loss and prevention of weight regain for adults. Med Sci Sports Exerc. 2009;41(2):459–71.
22. Ross R, Janssen I. Physical activity, total and regional obesity: dose-response considerations. Med Sci Sports Exerc. 2001;33:S521–7. discussion S528–29.
23. McHugh MP, Magnusson SP, Gleim GW, et al. Visoelastic stress relaxation in human skeletal muscle. Med Sci Sports Exerc. 1992;24:1375–82.
24. Taylor DC, Dalton Jr JD, Seaber AV, et al. Visoelastic properties of muscle-tendon units. The biomechanical effects of stretching. Am J Sports Med. 1990;18: 300–9.
25. Souza AC, Bentes CM, de Salles BF, et al. Influence of inter-set stretching on strength, flexibility and hormonal adaptations. J Hum Kinet. 2013;36: 127–35.
26. Seco J, Abecia LC, Echevarría E, et al. A long-term physical activity training program increases strength and flexibility, and improves balance in older adults. Rehabil Nurs. 2013;38(1):37–47.
27. Smith JA, Greer T, Sheets T, Watson S. Is there more to yoga than exercise? Altern Ther Health Med. 2011;17(3):22–9.
28. Ross A, Thomas S. The health benefits of yoga and exercise: a review of comparison studies. J Alt Complement Med. 2010;16:3–12.
29. Evans S, Tsao J, Sternlieb B, Zeltzer L. Using the biopsychosocial model to understand the health benefits of yoga. J Complement Integrat Med. 2009;6: 1–24.
30. Schmitz K, Courneya K, Matthews C, et al. American College of Sports Medicine roundtable on exercise guidelines for cancer survivors. Med Sci Sports Exerc. 2010;42(7):1409–26.
31. Hagins M, Moore W, Rundle A. Does practicing hatha yoga satisfy recommendations for intensity of physical activity which improves and maintains health and cardiovascular fitness? BMC Complement Altern Med. 2007;7:1–9.
32. Raub JA. Psychophysiologic effects of Hatha yoga on musculoskeletal and cardio-pulmonary function: a literature review. J Altern Complement Med. 2002;8:797.812.
33. Sterling P. Principles of allostasis: optimal design, predictive regulation, pathophysiology, and rational therapeutics. In: Schulkin J, editor. Allostasis, homeostasis, and the costs of physiological adaptation. Cambridge: Cambridge University Press; 2004. p. 17–64.
34. McEwen BS. Allostasis and allostatic load: implications for neuropsychopharmacology. Neuropsychopharmacology. 2000;22:108–24.
35. Khattab K, Khattab AA, Ortak J, et al. Iyengar yoga increases cardiac parasympathetic nervous modulation among health yoga practitioners. Evid Based Complement Altern Med. 2007;4:511–7.
36. Michalsen A, Grossman P, Acil A, et al. Rapid stress reduction and anxiolysis among distressed women as a consequence of a three month intensive yoga program. Med Sci Monit. 2005;11:555–61.
37. Selvamurthy W, Sridharan K, Ray US, et al. A new physiological approach to control essential hypertension. Indian J Physiol Pharmacol. 1998;42:205–13.
38. Damodaran A, Malathi A, Patil N, et al. Therapeutic potential of yoga practices in modifying cardiovascular risk profile in middle aged men and women. J Assoc Physicians India. 2002;50:633–9.
39. McCaffrey R, Ruknui P, Hatthakit U, Kasetsomboon P. The effects of yoga on hypertensive persons in Thailand. Holist Nurs Pract. 2005;19:173–80.
40. Shapiro D, Cook IA, Davydov DM, et al. Yoga as a complementary treatment of depression: effects of traits and moods on treatment outcomes. Evid Based Complement Altern Med. 2007;4:493–502.
41. West J, Otte C, Geher K, Johnson J, et al. Effects of Hatha yoga and African dance on perceived stress, affect, and salivary cortisol. Ann Behav Med. 2004;28:114–8.
42. Khatri D, Mathur KC, Gahlot S, et al. Effects of yoga and meditation on clinical and biochemical parameters of metabolic syndrome. Diabetes Res Clin Pract. 2007;78:e9–10.
43. Gokal R, Shillito L. Positive impact of yoga and pranayam on obesity, hypertension, blood sugar, and cholesterol: a pilot assessment. J Altern Complement Med. 2007;13:1056–7.
44. Stuck M, Meyer K, Rigotti T, et al. Evaluation of a yoga based stress management training for teachers: effects on immunoglobulin A secretion and subjective relaxation. J Medit Res. 2003;1–8.
45. Rao RM, Telles S, Nagendra HR, et al. Effects of yoga on natural killer cell counts in early breast cancer patients undergoing conventional treatment. Comment to: recreational music-making modulates natural killer cell activity, cytokines, and mood states in corporate employees Masatada Wachi, Masahiro Koyama, Masanori Utsuyama, Barry B. Bittman, Masanobu Kitagawa, Katsuiku Hirokawa. Med Sci Monit 2007;13:CR57–CR70. Med Sci Monit. 2008;14:3–4.
46. Pullen PR, Nagamia SH, Mehta PJ, et al. Effects of yoga on inflammation and exercise capacity in patients with chronic heart failure. J Card Fail. 2008;14:407–13.
47. Schultz PE, Haberman M, Karatha K, et al. Iyengar Yoga can promote well-being in women breast cancer survivors. Spokane, WA: Washington State University; 2007.
48. Innes KE, Bourguignon C, Taylor AG. Risk indices associated with the insulin resistance syndrome, cardiovascular disease, and possible protection with yoga: a systematic review. J Am Board Fame Pract. 2005;18:491–519.
49. Bower J, Woolery A, Sternlieb B, Garet D. Yoga for cancer patients and survivors. Cancer Control. 2005;12:165–71.
50. Evans S, Subramanian S, Sternlieb B. Yoga as treatment for chronic pain conditions: a literature review. Into J Disable Human Dev. 2008;7:25–32.

51. Pilkington K, Kirkwood G, Rampes H, Richardson J. Yoga for depression: the research evidence. J Affect Discord. 2005;89:13–24.
52. Kirkwood G, Rampes H, Tuffrey V, Richardson J, Pilkington K. Yoga for anxiety: a systematic review of the research evidence. Br J Sports Med. 2005;39: 884–91.
53. Galantino ML, Bzdewka TM, Eissler-Russo JL, et al. The impact of modified hatha yoga on chronic low back pain: a pilot study. Altern Ther Health Med. 2004;10:56–9.
54. Tsao J, Meldrum M, Kim SC, Jacob MC, Zeltzer LK. Treatment preferences for CAM in pediatric chronic pain patients. Evid Based Complement Altern Med. 2007;4(3):367–74.
55. Praissman S. Mindfulness-based stress reduction: a literature review and clinician's guide. J Am Accad Nurse Pract. 2008;20:212–6.
56. Dalle Grave R, Di Pauli D, Sartirana M, Calugi S, Shafran R. The interpretation of symptoms of starvation/severe dietary restraint in eating disorder patients. Eat Weight Discord. 2007;12:108–13.
57. Spoor S, Bekker M, Van Heck G, Croon M, Van Strien T. Inner body and outward appearance. J Eat Discord Treat Prevent. 2005;13:479–90.
58. Deaver C, Mitenberger R, Smyth J, Meidinger A, Crosby R. An evaluation of affect in binge eating. Behav Mod. 2003;27:578–99.
59. Keske-Rahkonen A, Hoek H. Incidence and outcomes of bulimia nervosa: a nationwide population-based study. Psycho Med. 2008;8:1–9.
60. Davis R, Jamieson J. Assessing the functional nature of binge eating in the eating disorders. Eat Behav. 2005;6:345–54.
61. Overton A, Selway S, Strongman K, Houston M. Eating disorders—the regulation of positive as well as negative emotion experience. J Clin Psycho Med Settings. 2005;12:39–56.
62. Whiteside U, Chen E. Difficulties regulating emotions—do binge eaters have fewer strategies to modulate and tolerate negative affect? Eat Behav. 2007;8:162–9.
63. Kabat-Zinn J. Full catastrophe living. Using the wisdom of your body and mind to face stress, pain, and illness. New York: Delatorre Press; 1990.
64. Gyatso GK. Transform your life. New York: Tharp Publications; 2001.
65. Ramel W, Goldin P, Carmona PE, McQuaid JR. The effects of mindfulness meditation on cognitive process and affect in patients with past depression. Cognitiv Ther Res. 2004;28:433–55.
66. Hamilton NA, Kitzman H, Guyotte S. Enhancing health and emotion: mindfulness as a missing link between cognitive therapy and positive psychology. J Cognitiv Psychother. 2006;20:123–34.
67. Davidson R, Kabat-Zinn J. Response to "Alterations in brain and immune function produced by mindfulness meditation: three caveats". Psychosom Med. 2004;66:148–52.
68. Greeson J. Mindfulness research update: 2008. Complement Health Pract Rev. 2009;14:10–8.
69. Greeson J, Brantley J. Mindfulness and anxiety disorders: developing a wise relationship with the inner experience of fear. In: Didonna F, editor. New York: Springer; 2008. p. 171–188.
70. Grossman P, Niemann L, Schmidt S, Walach H. Mindfulness-based stress reduction and health benefits. A meta-analysis. J Psychosom Res. 2004;57:35–43.
71. Jain S et al. A randomized controlled trial of mindfulness meditation versus relaxation training: effects on distress, positive states of mind, rumination, and distraction. Ann Behav Med. 2007;33:11–21.
72. Vieten C, Astin J. Effects of a mindfulness-based intervention during pregnancy on prenatal stress and mood: results of a pilot study. Arch Womens Ment Health. 2008;11:67–74.
73. Davis L, Strasburger A, Brown L. Mindfulness: an intervention for anxiety in schizophrenia. J Psychosoc Nurs Ment Health Serv. 2007;45:23–9.
74. Gaudiano B, Herbert J. Acute treatment of inpatients with psychotic symptoms using acceptance and commitment therapy: pilot results. Behav Res Ther. 2006;44:415–37.
75. Harned MS, Banawan SF, Lynch TR. Dialectical behavior therapy: an emotion-focused treatment for borderline personality disorder. J Contemp Psychother. 2006;36:67–75.
76. Williams J. Cognitive therapy (MBCT) in bipolar disorder: preliminary evaluation of immediate effects on between-episode functioning. J Affect Disord. 2008;107:275–9.
77. Bowen S, Chawla N, Marlatt GA. Mindfulness-based relapse prevention for addictive behaviors: a clinician's guide. New York: Guilford Publications, Inc; 2010.
78. Carlson LE, Speca M, Faris P, Patel K. One year pre-post intervention follow-up of psychological, immune, endocrine and blood pressure outcomes of mindfulness-based stress reduction (MBSR) in breast and prostate cancer patients. Brain Behav Immun. 2007;21:1038–49.
79. Witek-Janusek L et al. Effect of mindfulness based stress reduction on immune function, quality of life and coping in women newly diagnosed with early stage breast cancer. Brain Behav Immun. 2008;22:969–81.
80. Elomaa MM, de C Williams AC, Kalso EA. Attention management as a treatment for chronic pain. Eur J Pain. 2009;13:1062–7.
81. Ludwig D, Kabat-Zinn J. Mindfulness in medicine. JAMA. 2008;300:1350–2.
82. Lush E et al. Mindfulness meditation for symptom reduction in fibromyalgia: psychophysiological correlates. J Clin Psychol Med Setting. 2009;16:200–7.
83. Surawy C, Roberts J, Silver A. The effect of mindfulness training on mood and measures of fatigue, activity, and quality of life in patients with Chronic Fatigue Syndrome on a hospital waiting list: a series of exploratory studies. Behav Cognitiv Psychother. 2005;33:103–9.
84. Smith JE et al. Mindfulness-based stress reduction as supportive therapy in cancer care: systematic review. J Adv Nurs. 2005;52:315–27.

85. Butryn ML et al. Mindfulness and its relationship with eating disorders symptomatology in women receiving residential treatment. Eat Behav. 2013;14:13–6.
86. Merwin RM, Wilson KG. Understanding and treating eating disorders: an ACT perspective. In: Blackledge JT, Ciarrochi J, Deane FP, editors. Acceptance and commitment therapy: contemporary theory, research, and practice. Australia: Australian Academic Press; 2009. p. 87–117.
87. Merwin R, Zucker N, Lacy J, Elliot C. Interoceptive awareness in eating disorders: distinguishing lack of clarity from non-acceptance of internal experience. Cognition Emot. 2010;24:892–902.
88. Orsillo SM, Batten SV. ACT as treatment of a disorder of excessive control: Anorexia. Cognitiv Behav Pract. 2002;9:253–9.
89. Hayes SC, Pankey J. Experiential avoidance, cognitive fusion, and an ACT approach to anorexia nervosa. Cognitiv Behav Pract. 2002;9:243–7.
90. Schmidt U, Treasure J. Anorexia nervosa: valued and visible. A cognitive-interpersonal maintenance model and its implications for research and practice. Br J Clin Psychol. 2006;45:343–66.
91. Sim L, Zeman J. Emotion awareness and identification skills in adolescent girls with bulimia nervosa. J Clin Child Adolesc Psychol. 2004;33:760–71.
92. Harrison A, Sullivan S, Tchanturia K, Treasure J. Emotion recognition and regulation in anorexia nervosa. Clin Psychol Psychother. 2009;16:348–56.
93. Anderson D, Simmons AM. A pilot study of a functional contextual treatment for bulimia nervosa. Cognitiv Behav Pract. 2008;15:172–8.
94. Baer R, Fischer S, Huss D. Mindfulness and acceptance in the treatment of disordered eating. J Rational Emot Cognitiv Behav Ther. 2005;23:281–300.
95. Juarascio AS, Forman E, Herbert JD. Acceptance and commitment therapy versus cognitive therapy for the treatment of comorbid eating pathology. Behav Mod. 2010;34:175–90.
96. Kristeller J, Baer R, Quillian-Wolever R. Mindfulness-based approaches to eating disorders. In: Baer RA, editor. Mindfulness-based treatment approaches: clinician's guide to evidence base and applications. San Diego, CA: Elsevier Academic Press; 2006. p. 75–91.
97. Safer DL, Telch CF, Chen EY. Dialectical behavior therapy for binge eating and bulimia. New York: The Guilford Press; 2009.
98. Wanden-Berghe RC, Sanz-Valero J. The application of mindfulness to eating disorders treatment: a systematic review. Eat Disord. 2011;19:34–48.
99. Wolever R, Best J. Mindfulness-based approaches to eating disorders. In: Didonna F, editor. Clinical handbook of mindfulness. New York: Springer; 2009. p. 259–87.
100. Linehan M. Cognitive-behavioral treatment of borderline personality disorder. New York: Guilford Press; 1993.
101. Telch C, Agras W, Linehan M. Group dialectical behavior therapy for binge eating disorder: a preliminary, uncontrolled trial. Behav Ther. 2000;31:569–82.
102. Telch C, Agras W, Linehan M. Dialectical behavior therapy for binge eating disorder. J Consult Clin Psychol. 2001;69:1061–5.
103. Hayes S, Strosahl K, Wilson K. Acceptance and commitment therapy: an experimental approach to behavior change. New York: Guilford Press; 1999.
104. Hayes S, Wilson K, Gifford E, Follette V. Emotional avoidance and behavioral disorders: a functional dimensional approach to diagnosis and treatment. J Consult Clin Psychol. 1996;64:1152–68.
105. Heffner M, Eifert G. The anorexia workbook: how to accept yourself, heal your suffering, and reclaim your life. Oakland, CA: New Harbinger; 2004.
106. Kristeller J, Hallett B. An exploratory study of a meditation-based intervention for binge eating disorder. J Health Psychol. 1999;4:357–63.
107. Kristeller J, Wolever R. Mindfulness-based eating awareness training for treating binge eating disorder: the conceptual foundation. Eat Disord. 2011;19:49–61.
108. Kristeller J, Wolever R, Sheets V. Mindfulness-based eating awareness training (MB-EAT) for binge eating: a randomized clinical trial. Mindfulness. Springer; 2013. DOI: 10.1007/s12671-012-0179-1.
109. Corstorphine E. Cognitive-emotional-behavioural therapy for the eating disorders: working with beliefs about emotions. Euro Eat Disord Rev. 2006;14:448–61.
110. Lavander JM, Jardin B, Anderson DA. Bulimic symptoms in undergraduate men and women: contributions of mindfulness and thought suppression. Eat Behav. 2009;10:228–31.
111. Proulx K. Experiences of women with bulimia nervosa in a mindfulness-based eating disorder treatment group. Eat Disord. 2008;16:52–72.
112. Leahey T, Crowther JH, Irwin SR. A cognitive-behavioral mindfulness group therapy intervention for the treatment of binge eating in bariatric surgery patients. Cognitiv Behav Pract. 2008;15:349–442.
113. Heffner M, Sperry J, Eifert G, Detweiler M. Acceptance and commitment therapy in the treatment of an adolescent female with anorexia nervosa: a case example. Cognitiv Behav Pract. 2002;9:232–6.
114. Safer D, Telch C, Agras WS. Dialectical behavior therapy adapted for bulimia: a case report. Int J Eat Disord. 2001;30:101–6.
115. Hepworth N. A mindful eating group as an adjunct to individual treatment for eating disorders: a pilot study. Eat Disord. 2011;19:6–16.
116. Raynor HA, Epstein LH. Dietary variety, energy regulation, and obesity. Psychol Bull. 2001;127:325.
117. Goldfield G, Adamo KB, Rutherford J, Legg C. Stress and the relative reinforcing value of food in female binge eaters. Physiol Behav. 2008;93(3):579–87.
118. Timmerman G, Brown A. The effect of a mindful restaurant eating intervention on weight management in women. J Nutr Educ Behav. 2012;44:22–8.

# Cardiovascular Exercise Guidelines for Children and Adolescent Women

21

Melissa R. Iñigo and Maria Fernandez-del-Valle

**Abstract**

Exercise participation implemented during childhood and adolescence may promote a continued active lifestyle later in life. This may then prevent the apparition of cardiovascular, metabolic, and other diseases known to be prevalent in young and older adults. Children and adolescents have unique exercise capacity characteristics. Thus, appropriate cardiovascular exercise guidelines were developed in order to receive maximum benefits from exercise training while also emphasizing safety. Furthermore, approaches to training with the youth were developed to promote exercise and sport adherence. This chapter focuses on cardiovascular exercise guidelines for children and adolescent women.

**Keywords**

Cardiovascular training • Children • Adolescence

## 21.1 Learning Objectives

After completing this chapter, you should have an understanding of:

1. Children's and adolescents' unique exercise capacity characteristics
2. How to approach exercise with children and adolescents
3. Cardiovascular exercise guideline for children and adolescents

## 21.2 Introduction

The importance of participating in physical activity is not limited to adults only. Establishing good health habits during the earlier stages of

M.R. Iñigo, M.S. (✉)
Department of Health, Exercise, and Sport Sciences, Texas Tech University, Lubbock, TX, USA

8807 49th Avenue, College Park, MD 20740, USA
e-mail: minigo@umd.edu

M. Fernandez-del-Valle, Ph.D.
Department of Health, Exercise, and Sport Sciences, Texas Tech University, Exercise and Sport Sciences Building, 3204 Main, Lubbock, TX 49423, USA
e-mail: maria.fernandez-del-valle@ttu.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_21, 

life may drive children and adolescents to continue to have an active lifestyle during adulthood [1]. Indeed, researchers see a decline in physical activity participation rates during adolescence, and it continues to decline as individuals get older [2]. This cultural trend can be combated if the promotion of a healthy lifestyle begins during childhood and adolescence because many habits are formed during this critical time [1].

The gains that children and adolescents obtain from exercise are plentiful. Through increased physical activity, specific muscular adaptations and enhancements in motor skill performance occur [3]. Exercise also plays a role in skeletal health, prevention of excess body fat accumulation, and improvements in psychosocial health and well-being. This helps prevent osteoporosis, obesity, and other comorbidities that may develop later in life. Furthermore, regular participation in aerobic physical activity improves mood, self-appraisal, mental discipline, and socialization [4]. Youth physical activity programs, including dynamic sports (swimming, gymnastics, rhythmic gymnastics, soccer, judo, etc.), have been shown to positively influence insulin sensitivity, lower total cholesterol and low-density lipoprotein levels, as well as improve performance measures (maximal oxygen consumption ($VO_2$max), sprint speed, long jump, vertical jump, flexibility, agility, kicking distance, etc.) [3, 5–9].

Children and adolescents have a high exercise capacity relative to their body size. Some cardiorespiratory parameters are different between children, adolescents, and adults. Sex differences in exercise capacity also become apparent in children as they get older due to hormonal changes that adolescent boys and girls experience. These cardiorespiratory parameters are trainable. Children are able to improve their aerobic and glycolytic capacity through physical activity and supervised exercise training. Included in this chapter are cardiovascular exercise guidelines for children and adolescent women.

## 21.3 Research Findings

### 21.3.1 Children's and Adolescents' Unique Exercise Capacity Characteristics

The exercise capacity of children is unique. For instance, a child's maximum oxygen consumption ($VO_2$max) is relatively higher than at any other point in life if no further exercise training ensues (i.e. the individual remains untrained during adulthood) [10]. $VO_2$max, which refers to the maximum amount of oxygen (milliliters) the body can use (per kilogram of body weight), is an important indicator of cardiovascular fitness. A child has a high level of aerobic capacity relative to their body size. Regular exercise at a level that maintains a heart rate (HR) of about 170–180 beats per minute may increase $VO_2$max in children. However, there is no evidence to suggest that the percent increase in $VO_2$max through training is similar between children and adults [10]. Absolute $VO_2$max increases in both genders at the same rate during childhood (until the age of 12). Boys continue to increase at the same rate until the age of 17 or 18, but girls begin to plateau with very small increases after age 12. Between the ages of 17 and 22, women show a decrease in $VO_2$max. This is attributed to a decrease in physical activity and an increase in adipose tissue during their adolescent years [11].

A relationship also exists between age and other cardiopulmonary parameters such as peak work rate, ventilation to carbon dioxide (VE/$VCO_2$) slope, and HR recovery. Younger children have a lower peak work rate than adolescents [10]. Moreover, the peak work rate of girls become increasingly lower compared to boys as they get older [10]. Next, children have a higher VE/$VCO_2$ slope than adults during exercise [10]. VE/$VCO_2$ slope represents ventilatory drive. An abnormally high VE/VCO2 slope may predict heart failure in patients with coronary heart disease. It is associated with decreased pulmonary perfusion, which leads to decreased oxygen

**Table 21.1** Possible causes of increased metabolic costs in children

| |
|---|
| Increased resting basal metabolic rate |
| Increased ventilatory costs |
| Higher stride frequency |
| Lower storage of elastic recoil forces |
| Locomotion style is biomechanically inefficient |
| Higher co-contraction of antagonist muscles |

availability, thus organ viability. In children however, the $VE/VCO_2$ slope is normally higher than in adults because of relatively less efficient breathing during exercise [10]. This slope decreases and normalizes to adult values as children get older. Furthermore, HR recovery is fastest in young children post-exercise [10]. Generally, healthy children can recover quicker from intense exercise bouts compared to adolescent counterparts, which indicates enhanced vagal tone and fitness.

Metabolic cost is also relatively greater in children and adolescents than in adults. At age 5, the excessive metabolic cost is approximately 37 %. However, at age 17, the excessive metabolic cost is only 3 % [11]. See Table 21.1 for factors that contribute to metabolic cost in children.

The glycolytic system of children and adults also differs significantly. When engaged in an activity that utilizes glycolysis (i.e., high-intensity short bout of exercise approximately 2–5 min), children show a lower level of performance compared to adolescents, who in turn show a lower level of performance when compared to adults. These decreases are thought to be the result of a smaller muscle mass, a lower glycolytic capacity, as well as a lack of neuromuscular coordination [11].

### 21.3.2 How to Approach Exercise with Children and Adolescents

Much of the activities that young children engage in should have an emphasis in enjoyment, peer-to-peer interaction, safety, age-appropriateness, and effectiveness. Being active should be enjoyable and something the children look forward to, for at their age, the fun factor promotes exercise adherence [12]. This is especially important for girls because they are more inclined to significantly decrease their level of activity after they reach puberty [2]. Children and adolescents also enjoy the social interaction during training sessions, games, and competitions, which is another important factor that encourages physical activity participation. Training programs for children must also be safe and guided by an expert so that under- or overtraining does not occur, and injuries can be prevented [3]. For instance, weight-bearing activities are allowed provided that it is supervised. However, the child must also be both mentally and physically ready for the stress that strength training entails. Finally, exposing children to multiple physical activities and sports is far more beneficial in encouraging exercise and sport adherence [13]. Specializing in one sport at such an early age (<15 years) may result in overtraining, burnout, and a less positive psychosocial development [13]. Thus, for example, cross training during the active recovery period (1–3 weeks after the in-season) is recommended [3].

### 21.3.3 Cardiovascular Exercise Guidelines for Children and Adolescents

Children and adolescents should exercise at least 60 min per day at a moderate to high intensity for at least 3 days a week [14]. For example, running, swimming, and playing are great activities for the youth. Nevertheless, aerobic activity should not be the only form of exercise in a child's overall active lifestyle (see Chapter 22). Moderate-to-high intense activities are recommended in order to achieve the overall health benefits of physical activity and to improve motor performance skills of children. The standard metabolic equivalent (MET) is often used to precisely define the different exercise intensities. 1 MET refers to the energy (oxygen) used by the body at rest. Moderate intensity is typically between 3 and 4 METS, and high intensity is ≥7 METS. Such moderate and vigorous exercises may consist of continuous activity (60 min) or intermittent bouts (10, 20, or 30 min each). However, this depends

on the goals of the individual as well. A child or an adolescent who is training for a particular organized sport or dance needs a well-designed periodized program that is specific to improving performance for that activity. Otherwise, the effectiveness of the program will not be optimal and can also promote injury. Other important guidelines are that exercise intensity and recovery period should be age-appropriate. The exercises should also be safe and well-supervised by an expert trainer or coach. Finally, the activities should be those that children and adolescents enjoy, and outside play with peers should be encouraged.

## 21.4 Contemporary Understanding of the Issues

When exposing children and adolescents to physical activity, their unique exercise characteristics are important to consider for safety reasons. The key point here is that the exercise program should not be as physically and mentally demanding as that in trained adults. This also increases the likelihood of children and adolescents adhering to exercise throughout their lifespan. The guidelines for cardiovascular exercise training are that children should participate in 60 min per day of at a moderate (3–4 METS) to high (7 METS) intensity (e.g. running, swimming, and playing other sports) [14].

## 21.5 Future Directions

Continuing research in physical activity is paramount in understanding how the body works and in developing a means to maximize health benefits and exercise performance.

One direction is to develop exercise guidelines specific for preadolescent female athletes. Currently, preadolescent female athletes have no guidelines of their own, despite their differences from males. Women experience an earlier onset of adolescent changes; therefore, it might be beneficial to develop different cardiovascular training guidelines to adjust for those changes during the preadolescent period.

In addition, the inclusion of functional exercises (strengthening the deep musculature of the trunk) into cardiovascular exercise programs may enhance cardiovascular exercise performance and prevent injuries (see Chap. 22). Future studies may also look at the benefits of multiple component training programs (i.e., strengthening, functional, and cardiovascular endurance training) in reducing injury, improving performance, and increasing exercise adherence in children and adolescent women.

## 21.6 Concluding Remarks

In this day and age, the importance of developing a healthy and active lifestyle from an early age cannot be stressed enough. The physical, health, and psychosocial benefits are plentiful. Maintaining an active lifestyle throughout one's lifespan may be more likely if one starts participation and adheres to physical activity during childhood. Children and adolescents have unique exercise characteristics. Therefore, the cardiovascular exercise guidelines are different from adults. Strong emphasis is placed on enjoyment, social interaction, safety, age appropriateness, and effectiveness.

## References

1. Kelder SH, Perry CL, Klepp K-I, Lytle LL. Longitudinal tracking of adolescent smoking, physical activity, and food choice behaviors. Am J Public Health. 1994;84(7):1121–6.
2. CDC. Centers for Disease Control and Prevention. Youth risk behavior surveillance-United States, 2011. MMWR Surveill Summ. 2012;61(4):1–162.
3. Faigenbaum AD, Kraemer WJ, Blimkie CJ, et al. Youth resistance training: updated position statement paper from the national strength and conditioning association. J Strength Cond Res. 2009;23 (5 Suppl):S60–79.
4. Annesi JJ. Effects of the youth fit for life protocol on physiological, mood, self-appraisal, and voluntary physical activity changes in African American preadolescents: contrasting after-school care and physical education formats. Int J Clin Health Psychol. 2007;7(3):641–59.
5. Fukuda DH, Stout JR, Kendall KL, Smith AE, Wray ME, Hetrick RP. The effects of tournament preparation

on anthropometric and sport-specific performance measures in youth judo athletes. J Strength Cond Res. 2013;27(2):331–9.
6. Michailidis Y, Fatouros IG, Primpa E, et al. Plyometrics' trainability in pre-adolescent soccer athletes. J Strength Cond Res. 2013;27(1):38–49.
7. Johnson BA, Salzberg CL, Stevenson DA. A systematic review: plyometric training programs for young children. J Strength Cond Res. 2011;25(9):2623–33.
8. Rosenkranz SK, Rosenkranz RR, Hastmann TJ, Harms CA. High-intensity training improves airway responsiveness in inactive nonasthmatic children: evidence from a randomized controlled trial. J Appl Physiol. 2012;112(7):1174–83.
9. Tournis S, Michopoulou E, Fatouros I, et al. Effect of rhythmic gymnastics on volumetric bone mineral density and bone geometry in premenarcheal female athletes and controls. J Clin Endocrinol Metab. 2010;95(6):2755–62.
10. ACSM. ACSM's guidelines for exercise testing and prescription. 8th ed. Philadelphia (PA): Lippincott Williams & Wilkins; 2009.
11. Bar-Or O, Rowland TW. Pediatric exercise medicine: From physiologic principles to health care application. Champaign: Human Kinetics; 2004.
12. Allender S, Cowburn G, Foster C. Understanding participation in sport and physical activity among children and adults: a review of qualitative studies. Health Educ Res. 2006;21(6):826–35.
13. National Association for Sport and Physical Education. Guidelines for participation in youth sport programs: specialization versus multi-sport participation [position statement]. Reston, VA: American Academy of Pediatrics; 2010.
14. Services USDoHaH. Physical activity guidelines for Americans. Washington, DC: US Department of Health and Human Services; 2008.

# 22 Resistance Training Guidelines for Active Females Throughout the Lifespan: Children, Adolescences, Adult Women, and the Aging Woman

Maria Fernandez-del-Valle and Tyrel S. McCravens

**Abstract**

Due to recent changes in physical activity practices, strength training, or resistance training, has become an area of focus in the research of determining the overall health of an individual. As individuals age, disorders relating to both bone and muscle begin to cause a decline in health and functional activity. Bone disorders, such as osteoporosis and osteopenia, have been linked to muscle disorders like sarcopenia (a loss of skeletal muscle mass). While these disorders do affect males, females tend to show higher incidences of these diseases. Current research suggests that resistance training can help to delay the effects of these diseases. Resistance training implemented in the early stages of life, such as childhood and adolescence, has been shown to cause increases in both bone growth, specifically bone mass and bone mineral density, and skeletal muscle mass. Resistance training can help maintain current levels of fitness in adults and improve activities of daily living in the elderly. In order to receive maximum benefits from resistance training, guidelines spanning the entire lifespan needed to be developed. The resistance training guidelines cover everything from basic supervision needs to intensity, duration, and frequency of the program. The guidelines include specific directions to increase hypertrophy, power, strength, and endurance. So, resistance training, which was once thought to cause injury in some, is now seen as a way to increase health and even reduce the chance of injury when done properly.

M. Fernandez-del-Valle, Ph.D. (✉)
Department of Health, Exercise, and Sport Sciences,
Texas Tech University, Exercise and Sport Sciences
Building, 3204 Main, Lubbock, TX 49423, USA
e-mail: maria.fernandez-del-valle@ttu.edu

T.S. McCravens, M.Sc (✉)
Department of Health, Exercise, and Sport Sciences,
Texas Tech University, 2500 Broadway, Lubbock,
TX 79409, USA
e-mail: tyrel.mccravens@ttu.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_22, © Springer Science+Business Media New York 2014

**Keywords**
Resistance training • Children • Adolescences • Hypertrophy Sarcopenia

## 22.1 Learning Objectives

After completing this chapter, you should have an understanding of:

- Strength as a conditional capacity. Strength: the main capacity?
- Effects of resistance training in the muscular-skeletal tissue through lifespan
- How to approach strength training with children and adolescents
- Resistance training guidelines with children, adolescents, adults, and aging women
- Resistance training as a preventive factor from injuries and healthy posture in the future

## 22.2 Introduction

### 22.2.1 Strength as a Conditional Capacity. Strength: The Main Capacity?

Given the recommendations of several organizations, it is clear an analysis about structures related with the quality of life and health in women of all age ranges is needed. The ability to prescribe accurate exercise programs depends on our knowledge of how different elements are involved in proper muscular-skeletal development and functional capacity performance.

In order to reach maximum oxygen uptake, the pulmonary, cardiovascular, and muscular-skeletal systems all have to function together properly. The cardiopulmonary system has been studied during exercise through the lifespan due to its relationship with performance, quality of life, longevity, and prognosis in chronic diseases [1–4], while muscular-skeletal tissue has been neglected for decades. However, due to recent changes in habits, new research and field work have contributed to the development of means and methods of training for high-performance athletes, regular practitioners, and patients with chronic pathologies. Strength training (resistance training, or RT) seems to appear as a basis to improve the quality of life, prevent injuries, and enhance an adequate performance. Therefore, strength seems to be the main component of physical fitness (Fig. 22.1) [5].

For a better understanding, it is necessary to know how components of physical fitness (strength, endurance, speed, flexibility, and coordination) are interrelated and how their improvements are interdependent. Since early 2000s, the theory of strength as an engine capacity has increased in relevance, and some authors have hypothesized that strength is the fundamental physical capacity. Traditionally, strength has been divided into three types: maximal strength, speed strength (muscular speed/power), and strength endurance (muscular endurance); however, there is a strength which is displayed in different forms. As a result, components such as speed and endurance result in maintenance of strength levels at different intensities and influence coordination and flexibility [5–7].

"The ability to generate force is necessary for all types of movements" [8]. The cross-sectional area of the muscle is related to the maximal force production, so motor unit recruitment and activation (size principle), fiber angle pennation, muscle length, joint angle, and contraction velocity are all factors that will alter the expression of muscular strength [8].

Strength is considered essential in giving all individuals, from young to old, the ability to perform activities associated with daily living, recreation, and maximal performance. It can also help to prevent injuries and disabilities that may occur throughout the lifespan. Therefore, an improvement in strength levels enhances the development of basic motor skills that facilitate the realization of more specific tasks [7, 9].

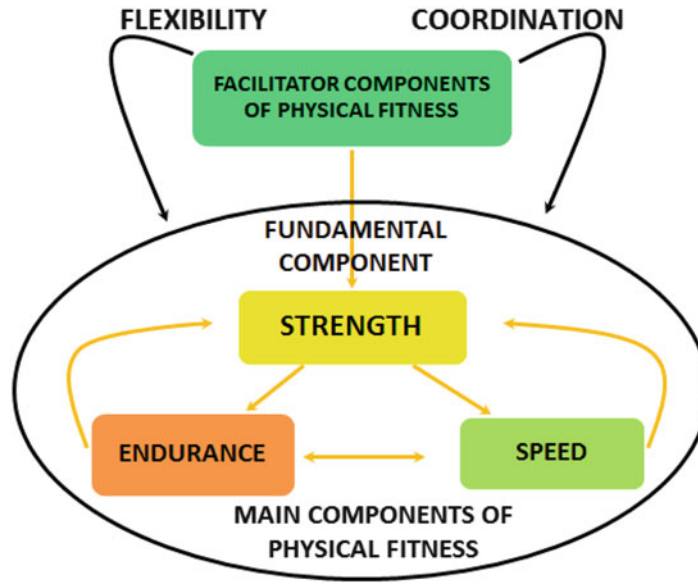



**Fig. 22.1** Components of physical fitness. Modified from Tous Fajardo J. Entrenamiento de la fuerza en los deportes de equipo. Apuntes del Máster Profesional en Alto Rendimiento en Deportes de Equipo. Barcelona (Spain): Byomedic-Mastercede, Fundación FC Barcelona; 2003

### 22.2.2 Skeletal Muscle: An Organ

Skeletal muscle has recently been considered an organ itself [10, 11]. This is because of its ability to synthesize and release a wide range of proteins at rest and during exercise, as well as its ability to metabolize carbohydrates (up to 80 %) and fats. In fact, skeletal muscle can also communicate with other tissue (e.g., adipose and bone) and organs (e.g., liver, pancreas, or brain) which can lead to bone, metabolic, and circulatory disease in the absence of regular activity [12].

Skeletal muscle is the largest reserve of protein in the body that supplies amino acids in order to synthesize proteins in other tissues and muscle in absence of nourishment and provides precursors for gluconeogenesis in the liver [13]. In addition, skeletal muscle is the primary site of glucose placement. Diminished muscle mass results in a diminished glucose metabolism related with higher risk in the genesis of pathologic conditions and chronic diseases. Indeed, healthy skeletal muscle is the major energy consumer and contributor to maintain an elevated metabolic rate (MR) at rest (up to 30 % of VO2 max) and during exercise (up to 90 % of VO2 max) [14, 15] and prevents the genesis of disease. Because of this, an adequate muscle mass and function has to be set up as priority endpoint of recommendations for physical activity (PA) and dietary intake. Moreover, lifestyle behaviors have to be explored to optimize muscle health through the life span.

Exercise produces adaptations necessary to achieve recovery and resume homeostasis through muscle contractions [11]. Maintaining a healthy organ is associated with an intense exercise practice liable to develop muscle mass and its reservoir capacities [16]. A healthy muscle delays disability as we age and improves quality of life [17, 18]. Individuals die as a result of the end of the aging process and deterioration of muscle organ reserve that supports physical functioning [16].

A decreased muscle mass is an index for undernutrition, mortality, and physical disability [19]. In spite of its importance, other indexes such body mass index (BMI) and body weight (BW) are used to assess and predict health status, dependence, longevity, mortality, and risk of diseases [20, 21].

## 22.3 Research Findings

### 22.3.1 Bone and Muscular Growth in Active Female Through Lifespan

As Wolff postulated in 1892, bone in a healthy person will adapt to the loads it is placed under [22]. Muscular contractions represent the most important physiological stimulus that reinforce bone mass and resistance, setting up a possible connection not only between skeletal resistance and age [23] but also with muscular function [24]. This process begins during the fetal stage and increases during second and third decade of life; however, later childhood and adolescent years are the most sensitive periods with greater increases of 1–20 % in those who exercised before pubertal growth to 40–60 % occurring in those who exercised 2 years after menarche during adolescence and only a 5 % in those who exercised after the age of 18 [25–27].

Bone mass (BM) variability depends on inter-individual factors such as age, gender, heredity, pubertal development, and environment (e.g., age, gender). A BM accumulation is more related with pubertal growth, decelerating as adolescence progresses. Bone mineral density (BMD) continues in a linear positive path until twenties; nevertheless, in adulthood, bone grows naturally only when a fracture or an organic disease comes about. BMD losses range from 0.5 to 1 % per year starting in the mid-20s [28] and are more evident in women during the menopause period and subsequent years, where in 5 to 10 years, losses could stand between 5 and 8 % per year. After age 65, BMD losses slow reaching 0.7 % per year. Childhood and adolescence are the greatest times to gain enough bone stock to get greater bone mineral peak, gains that could be increased if exercise starts before adolescence. In addition, prospective studies in Britain and Swiss have reported that BM acquired during childhood determines BM in adulthood [29, 30].

Skeletal muscle mass (SMM) is the largest component of lean body mass in humans (40–50 % of total body weight), with more than 650 muscles allowing every movement and posture for human body [15]. At birth, the SMM is between 23 and 25 % of total body weight (BW) which continues growing by increasing the number of muscle cells (hyperplasia) for a short time after birth. Thereafter, SMM gains occur mainly by an increase in volume of muscle cells (hypertrophy) [31].

Much like BM, SMM changes depend on age, gender, ethnicity, and environmental influences. The SMM acquired in early life changes in early adulthood, where it is reported that fat weight increases while lean body weight (LBW) decreases. Losses result in a 10 % average between the mid-20s and 50s, which is probably caused by nutritional and physical activity changes. In a physically inactive population, after 30s, loss is approximately 3–5 % per decade, and a parallel decline in muscle strength occurs. In addition, after the 50s, greater losses occur varying acutely; thereby, individuals who maintain a good diet and perform resistance exercise programs lose on average an additional 30 % by the age of 80, while individuals unconcerned of their lifestyle (sedentary, poor nutrition, etc.) amass losses as much as 50 % of SMM gained during young adulthood. By the age of 80, half of the SMM has been lost [15, 32].

Sex differences that occur during and after adolescence show SMM accounts for 45 % of women's BW compared to 54 % for males [31]. Those gender differences in total SMM result in a quantitative, not qualitative, muscular variance [33]. Such differences seem to be increased by a greater PA practice in males compared to females at any range of age [34–36]. Those differences result in a poor muscle mass that make females more susceptible to injury, poor physical condition, lower bone mineral content, etc. In addition to the muscular variance, female QOL decreases sharply due to women living longer and having higher rates of disease [37].

Despite of losses in SMM taking place as a part of the aging process, in some cases they can be triggered by a muscle-related diseases (myopathies), inherited (dystrophies, congenital, inflammatory, metabolic, or endocrine myopathies) or acquired (toxic myopathies originated via food and drug administration), resulting in

muscle weaknesses and physical impairment [13]. For that reason, the administration of treatments has to be taken in account in order to prescribe exercise to prevent a reduced function and muscle mass loss [38–40].

Through the years research has focused more on the bone and joint consequences of the aging process than on muscle mass and functioning [41]. However, a decrease in SMM is an evident consequence of the aging process with a serious impairment of musculature functioning [13, 41, 42]. The term *sarcopenia* was redefined recently by the European Working Group on Sarcopenia in Older People (EWGSOP) as an attempt to unify diagnostic criteria. Finally, sarcopenia was defined as the SMM loss associated with an impaired functioning affecting the physical performance [43], and according to this definition, both muscle mass (quantity) and functioning (quality) are affected [43]. Previous to a lack of consensus, epidemiologic research has reported values of sarcopenia from 8 to 40 % in elders between 60 and 70 years [44] and up to 50 % for those who are age 80 or above [42, 45]. Research has shown a higher incidence of sarcopenia depending on the gender, with greater values for females (8.5 %) compared to males (4.1 %) in 65–74 year olds [44]. Due to this methodological lack of concurrence, real values could have been underestimated for adults and elder population for years.

The primary factors affecting the degree of mass and function muscle loss are dietary protein intake and PA habits [46, 47]. In fact, subjects who show active lifestyle and healthy PA habits soften the muscle loss during the aging process [48].

There is a shift of muscle fiber distribution during the aging process that depends on two events: disuse (immobilization) and denervation. The lack of PA has shown a shift of muscle fiber distribution from slow to fast fiber type in aging subjects [49, 50], while denervation occurs mainly in fast motor units contributing to an increase of slower fiber type percentage content [49, 51]. Those adaptations are highly dependent upon PA and health rather than the aging process [52] and further affect the capacity to maintain posture and upright position against gravity (type I or slow fiber type) and the capacity to perform maximal strength and quick responses (type II or fast fiber type) [50].

During aging, the disproportionate unbalance between muscle protein synthesis and breakdown produces a loss in both fast and slow muscle fibers, showing a sharp loss of cross-sectional area of fast twitch type or type II fibers. Therefore, a motor unit loss occurs, and surviving motor units have to outface the loads which adapt through a net conversion of fast fibers to slow fibers. The loss of fast fibers is associated with a loss in muscle power necessary for quick movements (stand up, sit down, climbing stairs) and postural adjustments after stability perturbation. Moreover, this loss is accompanied by an increase of the placement of lipids in adipocytes and muscle fibers [52].

As muscle is lost, the bone turnover is affected because the mechanical stimulus to bone tissue and bone metabolism is decreased. Therefore, the rates of reabsorption and formation imbalance result in a bone mass loss and deterioration of bone microarchitecture [53, 54]. Research has reported a prevalence of 12.5 % sarcopenia in osteopenic premenopausal women. In addition, the 75 % of women suffering from osteopenia and osteoporosis presented associated sarcopenia, from which 25 % where osteopenic and 50 % osteoporotic postmenopausal women. Data suggest that the relationship of SMM, BMD, and risk of osteoporosis is largely associated to PA practice, showing lower incidence in regular PA practitioners [55]. The prevalence of osteopenia in the US population is 49 % in women beginning in their 50s (34 % men); moreover, osteoporosis prevalence is 18 % (5 % men) with higher percentages in female non-Hispanic white women (51 % osteopenia and 19 % osteoporosis) [56, 57]. Osteoporotic women have shown lower SMM compared with non-osteoporotic-matched controls [58] who show a positive relationship between LBM and BMD [59–61]. Therefore, sarcopenic women presented lower LBM and BMD compared to non-sarcopenic postmenopausal women [57].

Bone-related diseases such as osteoporosis and osteopenia are not common in the young,

healthy, and physically active population, such as children, adolescents, or young adults. Those conditions in the early stages of life are influenced by chronic-related diseases such eating disorders [62], cystic fibrosis [63], liver disease [64], renal disease [65], diabetes [66], or cancer [67].

### 22.3.2 Physical Activity Effects in the Muscular-Skeletal Tissue

Resistance training is so important that it is essential to maintain and strengthen other structures involved in physical performance. Not only do bone and muscle tissue receive benefits of physical activity, but other structures benefit as well. Normalized daily activity seems to be enough to maintain the 80–90 % of the mechanical properties of ligaments. Exercise causes the increase of the remaining 10–20 % of strength and stability in non-active female [68, 69]. Regular activity and exercise (including RT) help to maintain and/or increase strength levels and protect tendons from injuries [68].

In addition to muscular-skeletal effects, properly designed and supervised resistance training programs reported psychosocial benefits such as self-image, self-esteem, body image, social functioning, discipline, or collaborative capacity, showing greater improvements when participants start at a very low level if the stimulus is adequate.

#### 22.3.2.1 Children and Adolescents

Physical activity (PA) benefits occur early in life. Recent studies have focused on the SMM benefits of children participating in sports, resistance training, plyometric training, and high-intensity training [70–73]. Resistance and plyometric training both improve muscular strength and power; although, there are no obvious increases in muscle mass. Such muscular changes occur due to neurological adaptations in lieu of the lack of testosterone in children, a hormone known to promote muscle hypertrophy [70]. Muscles attach to growth plates in the bone, and the contraction of muscles in exercise and physical activity provides force application that stimulates proper bone growth [31]. BMD, bone mineral content, and growth hormone activity also increase with resistance and plyometric training and weight-bearing sports such as weightlifting and gymnastics [70, 73, 74]. Research supports that PA during childhood and adolescence results in greater BMD than PA performed during adulthood, showing a 5–8 % higher bone density in those with healthy PA habits during their life [70]. Zanker et al. reported that high-impact exercise before 7 years old seems to be beneficial for BM acquisition. In prepubertal stages, some authors show gains from 2 to 4 % in BM related with different exercise programs; moreover, strength-related exercises and high-impact exercises such as jumps reported longer benefits [75]. Junior Olympic power lifters and gymnasts reported greater BMD values compared with the general population. In addition, preadolescents show higher BMD and strength after high-impact resistance training [18]. Interestingly, high-intensity training augments airway responsiveness and maximizes strength and power development [76].

Current evidence shows that preadolescent girls have similar strength to boys [77]. Sex differences on strength and other physiological adaptations are first observed during the adolescent stage of life. Adolescent boys become taller, more muscular, and have lesser body fat than girls [78]. However, girls go through adolescence earlier than boys [79]. This may suggest that physiological adaptations may be different between sexes during the late preadolescent stage and that exercise guidelines must be distinct for preadolescent girls. Unfortunately, studies on the physiological responses to exercise of young female athletes are scarce due to limitations of noninvasive experiments [78]. Thus, at present, exercise guidelines for preadolescent female and male athletes are the same.

#### 22.3.2.2 Adult and Aging Woman

Research has reported that master athletes (those who train 4–5 days/week) show lower % BF than untrained subjects. In fact, master athletes' data

did not show significant differences in % BF and LBM compared with young athletes.

Interestingly, the master athletes who performed chronic exercise do not demonstrate the same losses of LBM and low-extremity strength compared to sedentary individuals. Wroblewski et al. used magnetic resonance imaging [53] to measure the subcutaneous adipose tissue [80], intramuscular adipose tissue (IMAT), and thigh total muscle area [81] in the low extremity. They included different MRIs comparing three subjects: a 40 year-old triathlete, a 74 year-old sedentary subject, and a 70 year-old triathlete that shows how mid-age adults and elder triathletes present similar TMA, SCAT, and IMAT. Actually, chronic exercisers presented higher functional capacity and QOL throughout the lifespan as age increased [52]. Also, according to Roubenoff [82], a resistance training program adapted to the population needs would lead to faster muscle contractions and greater force generation due to improvements in muscle fiber recruitment and firing rates [82].

It is known that protein synthesis rates decrease with age. However, research reported that progressive resistance training can increase protein synthesis rates in only 2 weeks. Those findings show protein synthesis rate increases up to 182 % following 2 weeks of a supervised RT program in 78–84 year olds [83], following 2 weeks of RT in 63–66 year olds [84], and increases by approximately 50 % after 3 month of supervised progressive RT in 76–92 year olds [85]. Such findings suggest that RT in older men and women increase the rate of muscle protein synthesis [86].

The ability to retain muscle mass and strength in the elderly has been demonstrated in those who practice chronic exercise [87, 88]; therefore, "chronic-exercise is prophylactic against age-related functional decline" [52]. Nevertheless, increases greater than 50 % in lower body strength can be obtained by performing short-term [89] and long-term [87, 90, 91] training interventions in upper decades. Also, those interventions can maintain strength and muscle integrity across the life span [52].

### 22.3.3 How to Approach Strength Training with Children and Adolescents

Fortunately, most children do participate in adequate levels of physical activity. Adolescents, however, tend to show a decrease in physical activity even to the point where many do not even meet the recommended guidelines (American College of Sports Medicine, ACSM) [92]. Resistance training should be approached in a manner that will encourage a positive attitude toward physical activity, which might lessen the decrease in adolescents. Like most activities, it is important that children and adolescents should be having fun while participating. Resistance training is no different. For children (≤13 years old), most RT can be found on the playground. For children who are on the playground playing hopscotch or jumping rope or simply skipping, the National Strength and Conditioning Association (NSCA) states that these types of activities can be similar to plyometrics [70]. Playground equipment, such as jungle gyms, monkey bars, and rock walls, can encourage children to climb and pull themselves up [93]. These types of activities resemble load bearing and body weight RT.

Encouragement should also be an important part of establishing positive attitudes. Positive verbal encouragement can also help to make the activities more enjoyable [94]. The Centers for Disease Control and Prevention, or CDC, has made several recommendations on how to get and keep children active. They include but are not limited to, leading by example, going to parks or community recreational centers, encouraging them, and, again, making the activities fun [95].

### 22.3.4 Resistance Training Guidelines

The safety and efficiency of resistance training has been endorsed and recognized by the medical community and most major health-promoting organizations such the *American Academy of Pediatrics*, the *American College of Sports*

*Medicine*, *British Association of Sport and Exercise Science*, *Canadian Society for Exercise Physiology*, and the *National Strength and Conditioning Association*.

#### 22.3.4.1 Resistance Training Guidelines in Children and Adolescents

In the past, resistance training was avoided for children because of the fear that it would interfere with their growth. This fear was in reference to children's growth plates closing prematurely with the stresses the resistance exercise placed on the plates. The theory originated from research on animal models and from monitoring the effects of the intense exercise regimen maintained by gymnasts, who seemed to have a corresponding decrease in growth [31]. Resistance training was also dismissed under the belief that children could not receive the benefit of increased strength due to their lack of androgenic hormones [31]. These schools of thought have faded though, with the understanding that RT can be done by children when the load applied is appropriate to the age and a qualified individual supervises the activity. In fact, it can be beneficial to children of all ages.

Even though there are several organizations and studies that have suggested their own guidelines for RT programs with children and adolescents, the guidelines are in agreement on several different aspects of the programs.

##### Supervision

Arguably the most important guideline might be that of supervision. The ACSM, NSCA, and AAP (American Academy of Pediatrics) all agree that a certified adult instructor should be with the youth at all times to ensure proper instruction and supervision [70, 92, 96]. Also, studies done by Myer and Wall [97] and Miller and Cheatham [93] suggest in their guidelines that an instructor be present at all times to provide proper supervision and instruction. The AAP even suggests that an instructor to student ratio should be around 1:10 if the children are healthy [96]. Instructors should be able to teach proper lifting techniques (full range of motion for both eccentric and concentric contractions), breathing techniques, body position, and proper spotting techniques. Instructors can also help to maintain a clean and safe environment, as well as be able to adjust the program to meet the needs of those participating.

##### Recommended Age for Starting

The ACSM considers children to be <13 years old and considers adolescents to be between the ages of 13 and 18 [92]. They consider children and adolescents to be able to follow the same guidelines that apply to an adult population.

The NSCA has proposed several guidelines that are similar to those of the ACSM. Age ranges for the NSCA are 11–13 (children) with 12–18 and 14–18 for girls and boys, respectively (preadolescents and adolescents) [70]. Resistance training is not suggested for individuals under the age of 7 years old. This is because those individuals might not be mentally ready to participate in a structured program like RT.

The next set of guidelines is based on the Corbin pyramids. The author has developed physical activity pyramids that give guidelines for both kids and teens [94]. Corbin's pyramids include a structured muscle fitness section designed for children under the age of 7. The types of exercises recommended for children include both body weight type activities and resistance band use [94].

##### Type of Exercise

The main resistance exercises need to be age- and ability-appropriate for the children, and the main focus when working with children is technique regardless of age or stage of development. The ACSM recommends both multi- and single-joint exercises [92]. These exercises should also include multiple muscle groups and major muscle groups, respectively, for multi- and single-joint exercises. This allows the child to perform the more difficult and tiring exercises without fatigue.

The NSCA recommends the use of simple exercises until proper form is maintained. Multi- and single-joint exercises incorporating both large and small muscle groups are encouraged as well. If a whole body workout is being performed

multiple times a week, complex, multi-joint exercises should be done at the start [70]. Also, machine weight, free weights, body weight, and elastic bands are also supported [70, 92].

### Warm-Up and Cooldown

The ACSM recommends that the resistance training session should include a 5–10 min warm-up period to get the body ready for physical activity, a stretching period, and a 5–10 min cooldown period. The stretching period can be after the warm-up or cooldown and should itself be around 10 min [92].

The NSCA also suggests a 5–10 min warm-up phase, and it should include dynamic movements. Current training recommendations include the utilization of dynamic warm-up [70]. Dynamic movements such as hops, skips, jumps, and movement-based exercises are better than performing static stretching to increase body temperature and motor unit excitability. The NSCA also recommends finishing the session with a cooldown phase consisting of calisthenics at a lower intensity [93]. Unlike the ACSM, the NSCA incorporates stretching into the cooldown period [70, 92].

### Conditioning Phase Guidelines

According to the ACSM guidelines, the conditioning phase should be between 20 and 60 min long [92]. Resistance training guidelines recommended by the ACSM consist of 2–4 sets with 8–12 repetitions, or reps, per set, starting at roughly 60–80 % 1 RM. The sets can be composed of different lifts if desired. For example, one set of squats and one set of leg presses would equal two sets for the lower body. Also, keep in mind the inverse relationship between reps and intensity (as reps increase, intensity decreases). The recommended rest time should be between 2 and 3 min per set. When 8–15 reps can be performed with good form and moderate fatigue, progression is needed to ensure gains are continued. The guidelines suggest a frequency of 2–3 days/week and suggest that these days are not consecutive and should allow for 48 h between working on the same muscle groups whether they are part of a whole body workout or specific muscle group workout [92].

The resistance training phase guidelines for the NSCA recommend that the individual start at a low intensity, or a light load, of 1–2 sets consisting of 10–15 reps per set at roughly 50–70 % 1 RM [70]. Guidelines dealing with training for strength and power suggest 1–3 sets of 6–15 reps per set and 1–3 sets of 3–6 reps per set, respectively. Because power lifts use a higher velocity lift than those for strength, the resistance should be lower where it would be higher for strength. Strength exercises can have a higher resistance because the lifting velocity is slower at a more moderate pace. A shorter rest time of 1 min is suggested for most lifts; however, when the intensity is increased, the rest period should increase to 2–3 min. The frequency recommendations given by the NSCA are 2–3 days/week with those days being nonconsecutive in order to provide a 48–72 h break for recovery. A lower frequency can be used if the goal of the program is to maintain gains [70].

Corbin pyramid for teens or adolescents guidelines is set for 1–3 sets with 8–12 reps per set at roughly 40–60 % 1 RM [98].

### Progression

Progression is discussed extensively in the updated position stand of the NSCA [70]; however, for this section, we will discuss it briefly. Progression is used to maximize gains and avoid staleness in the workout and should occur only after technique is mastered. During progression of resistance training, intensity should increase from novice to intermediate to advanced lifters. Because these guidelines are for children, most, if not all, of the individuals should be novices. If starting intensity was at 60 % 1 RM, progression could increase around 10 % for each of the other levels ending at 80 % 1 RM for advanced lifters. Volume is a little different. Again if the program begins at 1–2 sets at 10–15 reps, progression would see an increase in the number of sets but a slight decrease in the reps because we are still increasing intensity. Again as mentioned, rest time will increase with increasing intensity, and frequency can increase from 2–3 times/week up to 3–4 times/week. Power lifts follow a similar progression. While lifting velocity should stay relatively constant, after the first stage, more

weight can be added to see greater power gains. Power progression should also see an increase in the number of sets with a slight decrease in the number of reps; rest times should increase; and frequency, which should start at 2 days/week, could increase to 3 days/week [70].

Corbin proposed that the progression for adolescents may be warranted when the individual can perform the maximum number of reps with moderate fatigue. At this point, reps should be decreased and weight should be increased. The frequency should be 3 days/week [98].

Long-term planning of a training program, called periodization, must also be done in order to avoid overtraining and boredom. Periodization refers to an organized and planned scheduling of varying training, performing exercises, and recovery. This includes planning resistance training sessions 2–3 times a week on nonconsecutive days for beginners and intermediates. Children who are advanced in RT may train 3–4 times per week.

As children age, the resistance exercises they engage in should also progress with them. Dr. William J. Kraemer suggest specific guidelines. Kraemer emphasizes that through each age division, the focus should lie in teaching exercise technique because that component of a resistance training program is of pivotal importance [99].

#### 22.3.4.2 Resistance Training Guidelines with Adults and Aging women

Before getting into resistance training guidelines with adults and aging women, remember that resistance training is another form of exercise that allows the body to gain strength, power, and endurance; change one's physique by increasing muscle size and losing excess adipose tissue; improve balance and proprioception; and enhance motor performance. This involves a wide variety of lifting exercises that require the use of body weight or external resistance such as free weights, weight machines, cords, and bands. Each exercise targets a specific muscle or muscle group. If performed correctly and consistently in a progressively effective manner, RT will inevitably change the muscle's appearance and improve the capability of the body to perform movements.

Active adult women respond to resistance training similar to men with training adaptations leading to increases in hypertrophy, strength, power, and endurance. Muscle hypertrophy refers to the increase in muscle mass. Muscular strength refers to the ability of the muscle to generate force. Power refers to the strength of the muscle times the speed in which the action is performed. Powerful movements are explosive and dynamic such as jumping and throwing a ball. Muscular endurance refers to the ability of the muscle to repeatedly generate the same amount of force over a long period of time.

##### Resistance Training Guidelines for Adults

ACSM resistance training guidelines for adults are based in health-associated benefits of the muscular fitness established during the past decade. The following are general guidelines for the RT exercise prescription from the American College of Sports Medicine [1]. Appropriately designed RT improves the different components of strength (power, function, and endurance) which result in an improved muscular fitness [8].

###### Types of Exercise/Exercise Selection

Exercises can be performed using multiple modalities such as free weights, machines, cords, and unstable equipment. Both free weights and machines are effective for increasing strength; however, weight machines are recommended for novice to intermediate exercisers because they are safer and easier to use where emphasis in free weight exercises is recommended for advanced resistance training [8].

In adults, resistance training exercises include both single- and multi-joint exercises and unilateral and bilateral exercises. Single-joint exercises are useful to engage specific muscles while multi-joint exercises are capable of involving major muscle groups which require greater coordination and muscular balance. Multi-joint exercises have been reported as more effective to promote strength gains because more weight can be lifted [100]. Both unilateral and bilateral exercises produce gains in strength, and further unilateral exercises are useful in sports performance abilities [8].

It is recommended that novice, intermediate, and advanced individuals perform dynamic repetitions with concentric (CON: muscle shortening) and eccentric (ECC: muscle lengthening) muscle actions as well as isometric contractions (ISOM: no changes in muscle length) to stabilize core strength and specific isometric exercises [8].

Additionally, functional exercises are recommended to enhance muscular balance in joints, the core, pelvic, and scapula girdles [101].

### Warm-Up and Cooldown

There is an increasing importance of the dynamic warm-up due to the ability to increase body temperature, motor unit recruitment, kinesthesis activation, and joint mobility [81, 101–103].

### Conditioning Phase Guidelines

As mentioned before by Roubenoff [82], optimum volume allows functional and structural adaptations according to the objectives. Therefore, training out of the optimal stimulus will promote the risk of injuries or will not produce the expected results. The ACSM insures that altering the number of exercises, number of repetitions, and sets, or load, alters the training volume [8]. Due to the interindividual differences in the adaptation response to resistance training, minimum, medium, and maximal volumes are used to program exercise [9]. There is a recommended minimum volume of 1–3 sets per exercise (each muscle group) in novice individuals initially for the first 4 weeks of RT [8, 104, 105]. For intermediate and advanced individuals, research has reported that multiple-set programs with a systematic variation related to the program priorities are recommended [8]. For a medium volume, a recommendation of 4–9 sets (each muscle group) should be used depending on the individual's level (4 sets for a lower intermediate level and 6–9 for a higher intermediate level) and type of exercise selected (4–6 sets for major muscle groups and 8–9 sets for small muscle groups). Maximum volume is aimed at an advanced level, varies between 9 and 12 sets (each muscle group), and is recommended for a high-level, physically conditioned individuals with an RT background [9].

Resistance training frequency recommendations in novice individuals are lower at 2–3 days/week and focus on the entire body. The progression from a novice to an intermediate level depends upon other variables such volume and intensity more than frequency. Therefore, it is recommended that an intermediate individual progresses to 3–4 training days (e.g., from total body workout using 3 days to split body routines using 4 days). Frequency to progress toward an advanced training may vary depending on the objectives, recovery needed, nutrition supplementation, and sport specialization. Football players obtain better results if they train 4–5 days/week, while weight lifters and bodybuilders use high-frequency training programs double splitting routines at 8–12 training sessions per week.

Evidence loading statements and recommendations to maximize muscular strength are loads of 60–70 % of 1 RM for novice to intermediate and 80–100 % of 1 RM for advanced individuals. Notwithstanding, depending on the type of strength that is aimed to improve, the conditioning guidelines are different. The ACSM position stand presents three program designs: hypertrophy, muscular power, or muscular endurance. Therefore, loading, volume, exercise selection, exercise order, and resting periods are adjusted to obtain specific results [8]. These current resistance training guidelines include recommendations for increasing muscle hypertrophy, strength, power, and local muscular endurance. And these recommendations are dependent on physical capacity and training status.

Many different exercises are available for each target muscle or muscle group. In order to efficiently increase muscle hypertrophy, it is recommended that the resistance training program includes a combination of exercises that involve concentric, eccentric, and isometric muscle actions and use single- and multi-joint movements. Typically, a baseline program for novice and intermediate training may include four upper body, four lower body, and two core/abdominal exercises. For advanced training (>3 days/week), it is recommended that a split routine be used (e.g., first and third day: upper body; second and fourth day: lower body). The recommendation for sequencing during an RT session is to first perform exercises that utilize larger prior to small muscle groups, multi-joint prior to single-joint

exercises, or higher intensity prior to lower intensity exercises. Next, interchange between upper and lower body exercises, or exercises that utilize opposing muscle groups. A key element is using the appropriate weight for the desired number of repetitions. To increase muscle hypertrophy among novices and those with some experience (intermediate), performing resistance exercises with moderate weight is recommended (70–85 % of 1 RM) for 1–3 sets of 8–12 repetitions per exercise. Novices in RT should also focus on learning the proper technique for performing the exercises prior to using heavy external resistance. Those advanced in RT (> 1 year of experience) may opt for a more varied program where the loading range is from 70 to 100 % of 1 RM for 3–6 sets of 1–12 repetitions per exercise; however, more repetitions (6–12 RM) are recommended. RM (repetition maximum) refers to the maximum number of repetitions one can perform with a particular weight or external resistance. The magnitude of change in hypertrophy decreases as the muscle becomes accustomed to the load of the external resistance. Therefore, progressing to a heavier external resistance is necessary to avoid training plateaus. The recommendation is to increase the load by 2–10 % when one can perform the exercise with additional 1 or 2 repetitions over the prescribed number of repetitions. Another way to progressively overload the stress placed on the skeletal muscle is through increasing the total repetitions (training volume) performed at the current load. Limiting the rest interval to 1–2 min per set of moderate loading (for novice and intermediate) and 2–3 min per set of heavy loading (for advanced) is recommended. Training frequencies of 2–3 days/week for novices, 2–4 days/week for intermediate, and 4–6 days/week for advanced are recommended. The rest days are for recovery and are essential to prevent the negative effects of overtraining.

The recommendations for increasing muscular strength are similar to that of increasing muscle hypertrophy with the exception of training load, intensity, volume, and rest interval for intermediate and advanced individuals. Novices and intermediate level individuals are recommended to perform resistance training exercises with a load between 60 and 70 % of 1 RM for 1–3 sets of 8–12 repetitions with eventual progression in training load. Intermediate and advanced individuals are recommended to train using systematically varied and progressive multiple-set programs at loads between 80 and 100 % of 1 RM with emphasis on heavy loading and less repetitions (1–6 RM). For heavy loads, the recommended rest interval is longer (3–5 min per set) to allow the body to fully recover prior to performing another set. This has been shown to promote greater strength increases.

To increase muscular power, it is recommended that multi-joint exercises be predominantly used and performed through systematically varied loading strategies. Performing exercises with heavy loads (85–100 % 1 RM) in combination with explosive exercises using light to moderate loading (30–60 % of 1 RM for upper body exercises and 0–60 % of 1 RM for lower body exercises) for 1–3 sets of 3–6 repetitions is recommended. The recommended exercise sequence is similar to the recommendations in increasing muscular hypertrophy and strength. Furthermore, it is recommended that a resistance training program be integrated with the power program. The recommended rest interval is 2–3 min per set for high-intensity exercises and 1–2 min for low-intensity exercises. This resistance training program also follows similar guidelines dealing with progression toward heavier loads. Training 2–3 days/week for novices, 3–4 days/week for intermediate, and 4–5 days/week for advanced is recommended. When training greater than 3 days/week, a split routine is recommended.

Resistance training for increasing muscular endurance involves performing more repetitions of the exercises using lighter loads. The program must also progressively increase in load and volume. It is recommended that single- and multi-joint exercises be performed with a light load for multiple sets of 10–15 repetitions for novice and intermediate training and ≥25 repetitions for advanced training. A short rest interval between sets (1–2 min after performing ≥15 repetitions and <1 min after performing 10–15 repetitions) is recommended (for a summary of RT program design, see Table 22.1).

**Table 22.1** Variables manipulation depending on the aim of the resistance training program design

| Hypertrophy | Power | Endurance |
|---|---|---|
| Loading and volume | | |
| *Novice and intermediate*:<br>Moderate loading (70–85 % of 1 RM)<br>8–12 reps per set<br>1–3 sets per exercise<br>*Advanced*:<br>Loading range 70–100 % of 1 RM<br>1–12 reps per set<br>3–6 sets per exercise | Light to moderate loading: 30–60 % of 1 RM for upper body and 0–60 % of 1 RM lower body<br>1–3 sets per exercise<br>Progression ranges from heavy loading: 85–100 % of 1 RM to increase the force component, or 30–60 % of 1 RM for upper body and 0–60 % of 1 RM for lower body to increase fast force production<br>Multiple (3–6) sets per exercise<br>1–6 reps | *Novice and intermediate*: light loads with large reps (10–15) increase local muscular endurance<br>*Advanced*: various loading strategies for multiple sets per exercise (10–25 reps per exercise)<br>High volume |
| Frequency | | |
| *Novice*: 2–3 days/week<br>*Intermediate*: 4 days/week for total body and split routine<br>*Advanced*: 4–6 days/week for split routines | *Novice*: 2–3 days/week stressing the total body<br>*Intermediate*: 3–4 days/week for total body and split routines<br>*Advanced*: 4–5 days/week for predominantly total body routines | *Novice*: 2–3 days/week stressing the total body<br>*Intermediate*: 3 days/week for total body workouts and 4 days/week for upper/lower body split routine workouts<br>*Advanced*: 4–6 days/week if split routines are used |
| Exercise selection, order, and velocity | | |
| Single- and multi-joint exercises<br>Free weight and machine exercises<br>*Novice and intermediate*: slow to moderate velocities<br>*Advanced*: slow, moderate, and fast depending on the load, repetition number, and goals of each | Fast lifting velocities are needed to optimize power development with submaximal loading | Unilateral and bilateral exercises<br>Single- and multi-joint exercises<br>Slow velocity when 10–15 (moderate number) reps<br>Moderate-fast for increasing repetition number. Faster for large number of reps (15–25 or more) |
| Rest periods | | |
| Novice and intermediate: 1–2 min<br>Advanced (corresponding to each exercise goals or training phase): 2–3 min for high loading for core exercises, or 1–2 min for moderate to moderately high loading of other exercises | At least 2–3 min between sets for core exercises, or 1–2 for assistance exercises | Short rest periods, for example,<br>1–2 min for high-repetition sets (15–20)<br><1 min for moderate-repetition sets (10–15)<br>For circuit, weight training recommends resting periods that take place from going to one station to another |

### Progression

Progression for adults depends on increasing the resistance to promote additional gains. To obtain such gains, the program variables (frequency, intensity, volume, rest intervals, exercises, etc.) have to be suitable to individual characteristics and goals [8].

For progression in those individuals training at a specific load, there is a recommended 2–10 % increase when individual exceeds the current workload on two consecutive sessions [8]. For novice individuals, progression is recommended at 1–3 sets per exercise, and for intermediate to advanced, progression occurs through multiple sets with a systematic variation of volume and intensity, but not all exercises need the same number of sets. That is dependent on the goals and muscle group trained, for example, lifting more upper body than lower body.

To reduce the risk of overtraining and injury, it is not recommended to increase drastically. Besides, not all the exercises need the same number of sets, intensity, or repetitions [8]. Inappropriate manipulation can limit the magnitude of improvements and decrease the level of muscular fitness. To avoid these issues, variables should be manipulated based on the progression principles: progressive overload, specificity, and variation [106]. Progressive overload or gradual increase of stress is recommended by altering one or more of the following variables: intensity (absolute or relative), total repetitions at the current intensity, repetition speed, resting periods, or volume [107]. Specificity, "all training adaptations are specific to stimulus applied" determined by the muscle actions, speed of the movement, range of motion, muscle groups trained, intensity, and loading [8]. Therefore, successful resistance training programs are designed for specific purposes (e.g., hypertrophy, muscular speed/power, or muscular endurance). Variation and periodization are systematic processes applied to manipulate the training variables. The most studied have been intensity and volume [108, 109]. However, periodization can be organized in linear (classical and reverse periodization) and nonlinear models (undulating periodization) affecting the way intensity and volume are manipulated. For instance, classical periodization applies high volume and low intensity at the beginning of the training program while reverse periodization initiates applying high intensity and low volumes, and at the undulating periodization, intensity and volume variations alternate.

## Elder Resistance Training Guidelines

The development of sarcopenia and osteoporosis is multifaceted, and many of the causative factors are uncontrollable. Resistance training has been shown to be a powerful intervention in the prevention and treatment of sarcopenia [33], as well as positively influences the neuromuscular system, hormone concentrations, and protein synthesis rates. Muscle protein synthesis increases after one bout of high-intensity training and peaks approximately 24 after exercise. Moreover, the anabolic effect of the RT is maintained 36–48 h before losses start [8]. For that reason, frequency recommendations should be regulated depending on the population characteristics (initial PA, body composition status, nutrition, disability level, disease, etc.) owing to the value of the SMM as an essential index for independence, longevity, absence of disease, and QOL. The American College of Sports Medicine published in 2009 the "Exercise and Physical Activity Guidelines for Older Adults" [110] which suggests a frequency for RT at 2 days/week.

The ACSM basic recommendations for muscular strength in adults have proven to be effective guidelines for the elderly. However, to progress toward higher levels, it is important to introduce changes gradually. The muscular power ability decreases with aging, and improvements in this type of strength can reduce the risk of falls and enhance the capacity to perform daily tasks.

### Supervision and Safety Points

The role of the instructor supervising and monitoring the training for elders is essential because many individuals must be educated and directed properly. Teaching correct lifting techniques has to be a priority with this population [111].

Eccentric training results in greater muscle soreness; therefore, caution is recommended in

programs that include this type of muscular action [112]. In addition, excessive resistance training loads may acerbate a preexisting condition [111].

Aging adults have to perform exercises "pain-free" with controlled joint movements. This means that individuals with arthritis and joint and bone disorders have to avoid training during times of pain and inflammation. Further, the breathing patterns during resistance training exercises have to be taught to prevent apnea during the practice.

Resistance training can be progressively introduced to individuals with cardiovascular, metabolic, pulmonary, renal, psychiatric-related, and other diseases. Moreover, individuals with uncontrolled conditions (e.g., hypertension, chest pain, metabolic disturbances) need medical assessment prior to training [111].

#### Type of Exercise

For improvements in strength fitness, free weight and machine exercises are recommended. Older adults must perform multiple- and single-joint exercises with a slow to moderate lifting velocity [8, 110].

However, most of the time, free weights and machines are located at gyms or other facilities which some elderly individuals do not have access to on a regular basis. Resistance training can be accomplished, despite not having access to a facility, with the use of therapy bands, fitballs, and other equipment as well as self-body weight.

#### Conditioning Phase

The ACSM recommends starting resistance training with elders using minimal resistance loads for 8 weeks to allow the joints and connective tissues (e.g., ligaments, tendons) to adjust. After a period of not training, it is recommended to restart the training with loads 50 % or less than previous intensity [92]. For muscular strength, 1–3 sets with loads from 60 to 80 % of 1 RM are recommended. The rest periods are 1–3 min if volume is 2–3 days/week. When the objective is to increase muscular power in healthy older adults in addition to muscular strength, it is recommended to perform 1–3 sets per exercise using light to moderate intensity (30–60 % of 1 RM) and 6–10 repetitions with high velocity. Moreover, recommendations to improve local muscular endurance are similar to young adults where moderate to high repetitions (10–15 reps) are used with low to moderate loads (40-–70 % of 1 RM) [8].

Furthermore, to enhance better stability, muscle proprioception, and coordination, functional exercise in a standing position is recommended [111].

#### Progression

The progression guidelines are reported by the ACSM in the "Progression models in resistance training for healthy adults" [8]. Some authors suggest overload first by an increase in repetitions, followed by an increase in resistance [92]. Therefore, the manipulation of the variables will depend on the adaptations of the individual, as well as the health status, strength level, and goals.

### 22.3.5 Resistance Training as a Preventive Factor from Injuries and Healthy Posture in the Future

Resistance training has been found to be an effective form of injury prevention. The NSCA made note that training should be effective in reducing acute and overuse injuries related to sports [70]. In female athletes, training combined with proper form instruction has been shown to decrease ACL injuries. However, one area of concern is that a lot of RT programs are being implemented during a preseason workout. Coaches and instructors have to be careful because adding to workout time could lead to overuse injuries. Therefore, proper rest periods have to be implemented as well [70].

Faigenbaum and Schram looked at five studies dealing with resistance training and incidences of sport injury [113]. The subjects of the articles they reviewed were divided into an exercise group and a control group, ranged in age from 13 to 19 years old or were listed as high school

students, and included both males and females. The subjects were also involved in a variety of sports from handball to football. The training time lasted anywhere from 5 to 6 weeks up to 1 year. The training offered was a multicomponent program that included RT with other forms of training such as cardiovascular, flexibility, and speed and agility drills. Resistance training included weight training, plyometrics, and proprioceptive training. No study made use of all forms of training. One study looked at only training in the form of proprioceptive and plyometric training. All studies reported a decrease in injuries, ranging from knee to acute and overuse injuries, as compared to the control group [113]. Another study by Faigenbaum and Myer agreed that a comprehensive program could help in injury prevention in young athletes [114].

Posture is another area in which resistance training can be beneficial. Corbin et al. list two problems concerning the muscles that can contribute to problems with posture: muscle inflexibility and muscle weakness [98]. When muscles on either side of a joint are weaker or more inflexible than the ones across the joint, poor posture will follow. Stretching, which should also be a part of physical activity, should remedy muscle inflexibility. Exercises that work on strengthening the core muscles are recommended for development of a healthy posture [96]. A proper RT program will strengthen the muscles and allow for a proper posture to be developed.

## 22.4 Contemporary Understanding of the Issues

Resistance training can improve health no matter which stage of life the individual is in. Bone health can be improved when an increased load, which is achieved through RT, is placed on the bone [21–23]. Most of these gains have been seen in late childhood and adolescence [24–26]. It is important to see these gains, because of bone mineral density (BMD). After the 20s, BMD drops as individuals age and a healthy gain in BMD during the earlier years of life can lead to a larger amount in adulthood, which could slow the onset of several bone-related diseases [27–29].

Skeletal muscle mass (SMM) also experiences great benefits when a resistance training program is established. Even though initially gained through hyperplasia, SMM traditionally increases through the process of hypertrophy, which is brought on by RT [30]. Much like bone loss, SMM loss, which starts in the mid-20s, can be hindered by a proper RT program. Also, RT can help build a larger volume of SMM by the time the loss begins [14, 31].

SMM is also affected by gender differences with females showing a lower percentage of SMM as a part of overall body weight [30]. These changes are thought to be brought on by adolescence and the fact that more males than females tend to participate in physical activity [33–35]. This lack of physical activity can carry throughout the lifespan and lead to muscle fiber type and motor unit changes, which can be issues when trying to carry out general tasks [48–51].

Both the losses in bone and skeletal muscle mass can be intertwined. Remember that bone can be strengthened by a muscle applying force to an area of that bone [21–23]. If individuals show a loss of SMM through a lack of physical activity (such as resistance training), bone loss and diseases can occur due to a lack of stimulus to the bone. This would also affect bone formation and breakdown [52, 53]. If the force is no longer applied to the bone, the bone is not being strengthened where it would be if the force was being applied. At this point diseases such as osteopenia and osteoporosis can occur. In fact, research has shown that sarcopenia (loss of SMM) tends to accompany osteopenia and osteoporosis in women, who tend to participate in physical activity less than men of the same age group [54].

Trying to establish good physical activity habits early in life is paramount because there are a host of benefits in participating in physical activity. Resistance training in children and adolescents can produce increases in BMD and increases in strength and power [69, 73].

For relevance toward females, most females show similar strength levels when compared to their male counterparts before adolescence starts [76]. After adolescence, males tend to surpass females in terms of height, strength, and lean body mass [77]. Despite this difference, guidelines for physical activity, including RT, are mostly the same for both genders.

Previously, resistance training of any kind was considered to be detrimental to children and adolescents [70, 113, 114]. The main area of concern involved the epiphyseal cartilage, more commonly known as the growth plates. These tissues were considered to be weak compared to the surrounding structures and therefore more easily injured [70]. Even though injury to children and adolescents is still a concern to many [93], currently, RT has been shown to be safe and effective when proper instruction and supervision is available [70, 96, 97, 113, 114]. Remember that when approaching children and adolescents with an RT program, make sure it is fun [95]. Also, encouragement is also an integral part in getting the individuals to continue participating in the program [115]. Encouragement can come from teachers or parents and should get the kids moving and trying new activities.

The guidelines for resistance training in children and adolescents have come from several sources. Supervision by a qualified instructor is of the upmost importance [70, 92, 93, 96, 97]. Instructors can teach proper technique and insure a clean workout area to maximize safety. Children should participate in some form of the RT that overloads their muscles at a frequency of 60 min per day at 2–3 days/week [94].

For individuals that are both mentally and physically ready to participate in a structured training program (usually around 7–8 years old) [70, 93, 96], different guidelines are suggested. Exercises should include both multi- and single-joint lifts which work both large and small muscle groups. A 5–10 min warm-up and cooldown phase should be included in the training session and include stretching. The intensity should be 1–4 sets consisting of 8–15 reps between 40 and 80 % 1 RM depending on skill level with at least 1–3 min rest between sets [70, 92, 98]. During the resistance training phase, novice individuals should start with a lighter load until proper form and technique can be maintained [70]. Progression should only happen when the lifts can be performed with moderate effort while maintaining proper form and technique [70]. Also, keep in mind that if there is an increase in weight, the number of either sets or reps should drop and rest might have to be increased [98]. The frequency should be 2–3 nonconsecutive days per week [70, 92] and should last around 60 min per training session (for a summary of guidelines, see Table 22.2) [98].

For the adult and aging woman, physical activity can have positive effects. In those who train at least 4–5 days/week, lean body mass and body fat percentage are similar to those who are younger. When comparing the measure of a 40-year-old and 70-year-old, both triathletes, to a sedentary 70-year-old, the triathletes produced similar intramuscular adipose tissue and total muscle area in the thigh, as well as higher functional capacity and quality of life [51]. Also, adult and aging women show increases in protein synthesis after implementing resistance training [81–83]. Elderly individuals have also shown increases in strength after bouts of short- and long-term training [85, 87–89].

Adult women, who participate in resistance training, show similar changes in physique, balance, and performance as their male counterparts when the training is correctly executed and consistent. These changes also include those to hypertrophy, strength, power, and endurance. Regardless of gender, hypertrophy, strength, power, and endurance, each has its own set of guidelines, such as those set by the ACSM, to maximize gains for that specific type. Also, in the case of experience, whether the lifter be a novice, intermediate, or advanced exerciser, different guidelines must be followed to ensure safety and efficiency.

All levels of lifters should perform single- and multi-joint exercises that are unilateral and bilateral, as well as both concentric and eccentric contractions of dynamic repetitions, and isometric exercises [7]. This can also be the period where functional exercises start to work their way into

**Table 22.2** Summary of resistance training guidelines for children and adolescents

| |
|---|
| 1. Make sure a certified instructor is present to ensure proper form or lifting technique is used and a safe environment is maintained |
| 2. For young children, resistance should be from body weight and resistance bands, look to overload the muscles, and should last for 60 min/day at 2–3 days/week |
| 3. For older children who are ready to start a resistance training program (starting at 7–8 years old) and adolescents, machine weights, free weights, body weights, and resistance bands are acceptable |
| 4. Exercises should include multi- and single-joint lifts, working both large and small muscle groups |
| 5. A 5–10 min warm-up and cooldown should be part of the training session and involve stretching |
| 6. Resistance training should include 1–4 sets, 8–15 reps/set |
| 7. Resistance should be set at ~40–80 % 1 RM, with beginners starting with a lighter load |
| 8. Progression should be used when needed. Progression should occur when lifts can be made with proper form and technique at a moderate effort |
| 9. Resistance training should occur 2–3days/week that is nonconsecutive and the session should last 60 min |

*Source*: Adapted from American College of Sports Medicine. ACSM's guidelines for exercise testing and prescription. 8th ed., Baltimore: Lippincott Williams & Wilkins, 2010; Faigenbaum, AD, Kraemer, WJ, Blimkie, CJR, et al. Youth resistance training: updated position statement paper from the national strength and conditioning association. *Journal of Strength and Conditioning Research*. 2009;23(Aug):S60–S79; Corbin, CB, Le Masurier, GC, Lambdin, DD, et al. Fitness for life: elementary school guide for wellness coordinators. Champaign: Human Kinetics, 2010; and Corbin, C, Welk, G, Corbin, W, et al. Concepts of physical fitness: active lifestyles for wellness. 16th ed., New York: McGraw-Hill, 2011

the program [99]. Dynamic warm-up is also needed to prepare the body for the work that is about to be done [99–102].

For the actual resistance training program, it is recommended for novices to build up by performing 4 weeks of 1–3 sets per exercise with a lower volume. After the initial 4 weeks, the number of sets can also increase to 4 and then to 9 during the intermediate level, with major and small muscle groups having 4–6 sets and 8–9 sets, respectively [7]. Once an advanced level is obtained, a set range of 9–12 sets can be implemented [8].

When dealing with frequency of resistance training, it must be noted that rest is key for the muscles to heal and repair between work days. Frequency can be determined by several variables, and depending on these variables, RT can be done with a frequency from a few sessions/week up to 12 sessions/week. Novice and intermediate RT programs should have a frequency of 2–3 times/week and 3–4 times/week, respectively. Advanced individuals can have upwards of 12 lifting sessions/week depending on what is needed to be accomplished [7].

For novice and intermediate lifters, a load of 60–70 % of the 1 RM is recommended, while advanced lifters are recommended to lift 80–100 % of their 1 RM [7].

All of these guidelines can be manipulated to perform a specific function or lead to a specific type of gain for the individual participating in the resistance training program. Lifting different variations of the guidelines can lead to gains in hypertrophy, strength, power, or endurance.

Generally, gains in hypertrophy require both single- and multi-joint types of movements, and the lifter must use concentric, eccentric, and isometric contractions. Novices and intermediates should lift 70–85 % of their 1 RM, at 8–12 reps/set for 1–3 sets, with advanced individuals lifting upwards of 70–100 % of their 1 RM at 1–12 reps/set for 3–6 sets. These workouts can be done at a frequency of 2–3 days/week for novice individuals, 4 days/week for intermediates, and 4–6 days/week for advanced individuals. Novice individuals should lift whole body routines (composed of both upper and lower body as well as core exercises), while advanced individuals should lift split routines (alternating between upper and lower body). Intermediates can lift either. Rest should be between 1 and 2 min from one set to another for novice and intermediate individuals and 1–2 min for moderate loads and 2–3 min for high loading or core exercise for advanced individuals [7].

When putting together a resistance training program geared toward strength, the values are similar to that of hypertrophy. Novice and intermediate individuals should drop to 60–70 % 1 RM at 1–3 sets with 8–12 reps/set. Depending

on the level of an intermediate lifter, they can be considered part of the advanced group and lift 80–100 % of 1 RM with fewer sets and reps/set. Strength training will also benefit using 3–5 min rest periods as opposed to the shorter rest periods used for hypertrophy [7].

Muscle power is based on the rate (speed) of force production. So it only makes sense that muscle power is going to be achieved by performing fast lifts in the resistance training program. Heavier loads are still going to be used to increase the amount of force produced. So heavy loads of 80–100 % 1 RM are going to be integrated with lighter loads of 30–60 % for upper body and 0–60 % for lower body for 3–6 reps/set for 1–3 sets. This RT program will have a frequency of 2–3 days/week, 3–4 days/week, and 4–5 days/week for novice, intermediate, and advanced lifters, respectively. Also, intermediate and advanced lifters can use split routines. Resting periods should be 1–2 min for low intensity and 2–3 min for high intensity [7].

The key to muscle endurance is high volume at low intensity. This helps to prepare the muscles to last for longer periods of time. Novice and intermediate lifters should lift a light load for 10–15 reps while advanced individuals should lift 10–25 reps. Frequency for novice, intermediate, and advanced lifters is 2–3 days/week, 3–4 days/week, and 4–6 days/week, respectively. Also take note that split routines should be used when the frequency is 4 days/week or greater. A shorter rest period of less than 1 min should be used when 10–15 repetitions are completed, and when repetitions are greater than 15, 1–2 min of rest should be used [7].

Progression should occur when the individual is able to perform more than their current workload on two consecutive sessions [7]. If progression does not occur, the individual will hit a plateau where no further gains will be attained. Progression can occur through progressive overload, specificity, and variation [105]. Changing intensity, number or speed of reps, volume, or resting period are all considered to be progressive overloading [106]. Specificity changes could involve changes to muscle actions, ranges of motion, or even switching from one program to another [7]. Variation, also known as periodization, is altering variables such as intensity and volume. Switching between a high volume and low intensity to a low volume high intensity can lead to changes.

Even though many consider the elderly too weak to resistance train, there are several benefits that can be experienced. Two problems that tend to affect the elderly involve bone disorders, such as osteoporosis and osteopenia, and sarcopenia. Resistance training has been shown to help with sarcopenia [32]. This effect can, in turn, help with bone loss. Remember, women who showed lower levels of SMM also presented lower levels of LBM and BMD, with less incident in all for those who participated in physical activity [54–60].

The ACSM has set a standard of guidelines for the elderly, so they too can participate in resistance training. Safety must be a priority with the elderly, and only through proper technique can safety be insured [110]. Also, if any individual has preexisting conditions, a medical assessment should be considered because RT can acerbate those conditions [110].

While single- and multi-joint exercises using free weights, machines, and a multitude of other types of resistance are approved [7, 109], eccentric contractions need to be avoided because they can cause pain [111]. Standing exercises should be considered because of their health benefits [110]. Minimal resistance loads are recommended for the first 8 weeks, and only 50 % of previous workload should be used if the individual took a long break [91]. This helps with minimizing pain caused by lifting which could cause them to abandon any type of resistance training program.

The guidelines themselves are very basic, but they are the same as those for adults. The main objective for the elderly is to be able to get to the point where they can perform activities of daily living. While strength, power, and endurance are a concern for them as well as a younger population, the younger population obviously wants those changes for a different reason.

Muscular strength guidelines are 1–3 sets at 60–80 % 1 RM, with a 1–3 min rest period. These

guidelines are also incorporated into the guidelines for power, which are 1–3 sets at 30–60 % 1 RM and 6–10 reps at a higher velocity. Finally, endurance training has a higher rep number of 10–15 reps but at a lower intensity of 40–70 % 1 RM [7]. For all resistance training, a frequency of 2 sessions/week is recommended [109]. This allows for plenty of time for recovery. Progression will follow the adult guidelines.

When performed properly, RT can help to reduce the occurrence of injury [113, 114] and improve posture [96, 98]. Resistance training has been shown to decrease the number of knee injuries and the number of acute and overuse injuries in young athletes. When added to a preseason program, it can also prepare the athlete for participation, which could decrease injuries as well [70, 113]. Posture can be helped by training the core muscles [96]. Training will help strengthen the muscles that are weaker than the surrounding muscles which will help in improving posture [98].

## 22.5 Future Directions

Continuing research in physical activity is paramount in understanding how the body works and developing a means to maximize results. By understanding one aspect of the muscle, theories could be developed and tested in maximizing energy use, causing gains in hypertrophy, strength, power, and endurance, and maximizing healing times. If research could reveal the specifics of strength development, resistance training programs could be developed that would maximize gains in a minimal amount of time.

One future direction for resistance training could be to look at how one might approach RT toward children. Again with the age of 7–8 years old ideal for a structured RT program, one could look at possibly implementing a program before that age. Designing programs with a semi-structured layout (one that involves both structured exercise and free time) might be an effective way to get younger children used to a structured program that could then be developed more as they get older.

The guidelines are what drives and ensures the safety of a proper resistance training program. While many organizations and individuals agree on several aspects in the guidelines, there are some differences. The ACSM and NSCA both have different recommendations for sets and reps. The NSCA also included guidelines for power type RT. Future studies could provide some clarity on exactly how many sets and reps are needed to best fit the athlete. Also, if guidelines are available for strength and power, could there be other guidelines specific to muscular endurance?

Another direction could lead to changes in physical activity in children and adolescents. Currently, preadolescent female athletes have no guidelines of their own, despite the differences in male and female athletes. Because females experience an earlier onset of adolescent changes, it might be beneficial to develop different resistance training guidelines to adjust for those changes in the late preadolescent period.

For adults, future directions in physical activity could look at implementing functional exercises into a resistance training program. As we age, activities of daily living can become difficult to carry out. Functional exercises could make it easier to perform these tasks. By developing and implementing a proper functional exercise program to go along with RT, one might be able to delay or even possibly eliminate problems dealing with everyday functioning.

For the elderly, there are a couple of directions physical activity could be taken. One major aspect of the elderly is the threat of diseases to physical activity. While the exercises need to be performed "pain-free," many elderly individuals are not able to perform some activities without experiencing some level of pain. The development of guidelines could help minimize the pain experienced. Also, gains tend to be lost quicker in the elderly population. The development of guidelines to help maintain gains, even when not in a program, could serve the elderly population greatly.

One aspect of the studies dealing with overall effects of resistance training on injury prevention was the use of a multiple component training program. Most studies combined RT with some other form of training or instruction. Future studies might look at how much of a part RT alone

would play in decreasing the number of injuries. Another possible direction for future studies could look at using RT as a preventative method when added to a preseason program. Also, when dealing with posture, one could look at muscular strength vs. muscular flexibility. Again this could show if RT had more of an effect on posture or flexibility training.

## 22.6 Concluding Remarks

Recent research in resistance training has shown how beneficial it can be in all stages of life for overall physical health, especially in relation to bone and skeletal muscle disorders. This is especially important for females because of their higher incidences of osteoporosis and osteopenia, as well as sarcopenia. An RT program can help improve skeletal muscle mass, which, in turn, can help improve bone health. These gains can also help in slowing down further losses that are associated with the aging process.

In order to receive maximum gains from resistance training, specific guidelines had to be put into effect that would benefit individuals across the entire lifespan. Guidelines were developed that include children and adolescents, adult women, and the aging woman. While these guidelines generally cover both men and women, women can see the same benefits as their male counterparts. Children and adolescents have guidelines that are focused on developing a healthy attitude toward physical activity and making sure they are properly supervised and mentally and physically ready and have a proper RT program that allows them to achieve the increases in strength they are able to attain. Some of the greatest gains in bone health and skeletal muscle mass occur during this time period. Adults and aging women also have their own RT guidelines. Adults have guidelines that allow them to increase hypertrophy, power, and muscular strength and endurance. Guidelines for the aging women are directed more toward increasing their ability to perform activities of daily living. Resistance training has even been shown to decrease risk of injury when proper guidelines are followed.

Even with everything that is known about resistance training and its benefits, there are still areas that can be improved. Future research has the potential to create new guidelines, increase results gained, and provide new ways to slow the decreases associated with aging. Research may even lead to guidelines that are specific for the female athlete.

## References

1. Garber C, Blissmer B, Deschenes M, et al. ACSM position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. Med Sci Sports Exerc. 2011;43(7):1334.
2. Rodriguez NR, DiMarco NM, Langley S. Position of the American dietetic association, dietitians of Canada, and the American college of sports medicine: nutrition and athletic performance. J Am Diet Assoc. 2009;109(3):509–27.
3. Nelson ME, Rejeski WJ, Blair SN, et al. Physical activity and public health in older adults: recommendation from the American College of Sports Medicine and the American Heart Association. Med Sci Sports Exerc. 2007;39(8):1435.
4. Donnelly J, Blair S, Jakicic J, Manore M, Rankin J, Smith B. American College of Sports Medicine position stand. Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. Med Sci Sports Exerc. 2009;41(2):459.
5. Tous FJ. Entrenamiento de la fuerza en los deportes de equipo. Apuntes del Máster Profesional en Alto Rendimiento en Deportes de Equipo. Barcelona (Spain): Byomedic-Mastercede, Fundación FC Barcelona; 2003.
6. Tous-Fajardo J. Nuevas tendencias en fuerza y musculación. Barcelona: Ergo; 1999.
7. Verkhoshansky YV, Siff MC. Supertraining. 6th ed. Rome, Italy: Verkhoshansky; 2009.
8. ACSM. ACSM position stand. Progression models in resistance training for healthy adults. Med Sci Sports Exerc. 2009;41(3):687–708.
9. Naclerio AF. Entrenamiento Deportivo: fundamentos y aplicaciones en diferentes deportes. 1ªth ed. Madrid: Editorial Médica Panamericana; 2010.
10. Pedersen BK, Febbraio MA. Muscles, exercise and obesity: skeletal muscle as a secretory organ. Nat Rev Endocrinol. 2012;8(8):457–65.

11. Pedersen BK, Febbraio MA. Muscle as an endocrine organ: focus on muscle-derived interleukin-6. Physiol Rev. 2008;88(4):1379–406.
12. Pedersen B. The anti-inflammatory effect of exercise: its role in diabetes and cardiovascular disease control. Essays Biochem. 2006;42:105–17.
13. Wolfe RR. The underappreciated role of muscle in health and disease. Am J Clin Nutr. 2006;84(3):475–82.
14. Zurlo F, Larson K, Bogardus C, Ravussin E. Skeletal muscle metabolism is a major determinant of resting energy expenditure. J Clin Invest. 1990;86(5):1423.
15. Powers SK, Howley ET. Exercise physiology. 8th ed. New York, NY: McGraw-Hill Higher Education; 2011.
16. Pereira A, Izquierdo M, Silva AJ, Costa AM, González-Badillo JJ, Marques MC. Muscle performance and functional capacity retention in older women after high-speed power training cessation. Exp Gerontol. 2012;47(8):620–4.
17. Bagur CC. Ejercicio físico y masa ósea (I). Evolución ontogénica de la masa ósea e influencia de la actividad física sobre el hueso en las diferentes etapas de la vida. Apunts Medicina de l'Esport. 2007;42:40–6.
18. Behm DG, Faigenbaum AD, Falk B, Klentrou P. Canadian Society for Exercise Physiology position paper: resistance training in children and adolescents. Appl Physiol Nutr Metab. 2008;33(3):547–61.
19. Janssen I, Baumgartner RN, Ross R, Rosenberg IH, Roubenoff R. Skeletal muscle cutpoints associated with elevated physical disability risk in older men and women. Am J Epidemiol. 2004;159(4):413–21.
20. NHLBI NHLaBI. Assessing your weight and health risk. http://www.nhlbi.nih.gov/health/public/heart/obesity/lose_wt/risk.htm.
21. Devlin K. Do you believe in fairies, unicorns, or the BMI? 2009. Accessed May, 2012. http://www.maa.org/external_archive/devlin/devlin_05_09.html.
22. Cowin SC, Sadegh AM, Luo G. An evolutionary Wolff's law for trabecular architecture. J Biomech Eng. 1992;114(1):129.
23. Guyton AC, Hall JE. Textbook of medical physiology. 11th ed. Philadelphia, PA: Elsevier Saunders; 2006.
24. Schoenau E, Fricke O. Mechanical influences on bone development in children. Eur J Endocrinol. 2008;159 Suppl 1:S27–31.
25. Bailey DA, McKay HA, Mirwald RL, Crocker PR, Faulkner RA. A six-year longitudinal study of the relationship of physical activity to bone mineral accrual in growing children: the university of Saskatchewan bone mineral accrual study. J Bone Miner Res. 1999;14(10):1672–9.
26. Lang TF. The bone-muscle relationship in men and women. J Osteoporos. 2011;2011:702735.
27. Riggs BL, Melton III LJ, Robb RA, et al. Population based study of age and sex differences in bone volumetric density, size, geometry, and structure at different skeletal sites. J Bone Miner Res. 2004;19(12):1945–54.
28. Haywood K, Getchell N. Life span motor development. 5th ed. Illinois: Thomas-Shore; 2009.
29. Duppe H, Gardsell P, Johnell O, Nilsson BE, Ringsberg K. Bone mineral density, muscle strength and physical activity. A population-based study of 332 subjects aged 15–42 years. Acta Orthop Scand. 1997;68(2):97–103.
30. Cooper C, Cawley M, Bhalla A, et al. Childhood growth, physical activity, and peak bone mass in women. J Bone Miner Res. 1995;10(6):940–7.
31. Malina RM, Bouchard C, Bar-Or O. Growth, maturation, and physical activity. Champaign, IL: Human Kinetics; 2004.
32. Evans WJ, Lexell J. Human aging, muscle mass, and fiber type composition. J Gerontol A. 1995;50(Special Issue):11.
33. Roth S, Ferrell R, Hurley B. Strength training for the prevention and treatment of sarcopenia. J Nutr Health Aging. 2000;4(3):143.
34. Tremblay MSTMS, Colley RCCRC, Saunders TJSTJ, Healy GNHGN, Owen NON. Physiological and health implications of a sedentary lifestyle. Appl Physiol Nutr Metab. 2010;35(6):725–40.
35. Biddle SJ, Pearson N, Ross GM, Braithwaite R. Tracking of sedentary behaviours of young people: a systematic review. Prev Med. 2010;51(5):345–51.
36. Janz KF, Dawson JD, Mahoney LT. Tracking physical fitness and physical activity from childhood to adolescence: the muscatine study. Med Sci Sports Exerc. 2000;32(7):1250–7.
37. Austad SN. Why women live longer than men: sex differences in longevity. Gend Med. 2006;3(2):79–92.
38. Chawla J. Stepwise approach to myopathy in systemic disease. Front Neurol. 2011;2:49.
39. Walsh RJ, Amato AA. Toxic myopathies. Neurol Clin. 2005;23(2):397.
40. Guis S, Mattéi JP, Lioté F. Drug-induced and toxic myopathies. Best Pract Res Clin Rheumatol. 2003;17(6):877–907.
41. Cooper C, Dere W, Evans W, et al. Frailty and sarcopenia: definitions and outcome parameters. Osteoporos Int. 2012;23(7):1839–48.
42. Morley JE. Sarcopenia: diagnosis and treatment. J Nutr Health Aging. 2008;12(7):452–6.
43. Cruz-Jentoft AJ, Baeyens JP, Bauer JM, et al. Sarcopenia: European consensus on definition and diagnosis Report of the European Working Group on sarcopenia in older people. Age Ageing. 2010;39(4):412–23.
44. Abellan van Kan G. Epidemiology and consequences of sarcopenia. J Nutr Health Aging. 2009;13(8):708–12.
45. von Haehling S, Morley JE, Anker SD. From muscle wasting to sarcopenia and myopenia: update 2012. J Cachex Sarcopenia Muscle. 2012;3(4):213–7.

46. Morais J, Chevalier S, Gougeon R. Protein turnover and requirements in the healthy and frail elderly. J Nutr Health Aging. 2006;10(4):272.
47. Paddon-Jones D, Short KR, Campbell WW, Volpi E, Wolfe RR. Role of dietary protein in the sarcopenia of aging. Am J Clin Nutr. 2008;87(5):1562S–6S.
48. Visser M, Simonsick EM, Colbert LH, et al. Type and intensity of activity and risk of mobility limitation: the mediating role of muscle parameters. J Am Geriatr Soc. 2005;53(5):762–70.
49. D'Antona G, Pellegrino MA, Adami R, et al. The effect of ageing and immobilization on structure and function of human skeletal muscle fibres. J Physiol. 2003;552(2):499–511.
50. Pette D. Activity-dependent adaptive responses of skeletal muscle fibers. In: Mooren FC, Volker K, editors. Molecular and cellular exercise physiology. Champagne, IL: Human Kinetics; 2005. p. 263–74.
51. Hortobagyi T, Dempsey L, Fraser D, et al. Changes in muscle strength, muscle fibre size and myofibrillar gene expression after immobilization and retraining in humans. J Physiol. 2004;524(1):293–304.
52. Wroblewski AP, Amati F, Smiley MA, Goodpaster B, Wright V. Chronic exercise preserves lean muscle mass in masters athletes. Phys Sportsmed. 2011;39(3):172–8.
53. Hamrick M, McNeil P, Patterson S. Role of muscle-derived growth factors in bone formation. J Musculoskelet Neuronal Interact. 2010;10(1): 64–70.
54. Kanis J, Burlet N, Cooper C, et al. European guidance for the diagnosis and management of osteoporosis in postmenopausal women. Osteoporos Int. 2008;19(4):399–428.
55. Walsh MC, Hunter GR, Livingstone MB. Sarcopenia in premenopausal and postmenopausal women with osteopenia, osteoporosis and normal bone mineral density. Osteoporos Int. 2006;17(1):61–7.
56. Looker AC, Melton LJ, Harris TB, Borrud LG, Shepherd JA. Prevalence and trends in low femur bone density among older US adults: NHANES 2005–2006 compared with NHANES III. J Bone Miner Res. 2009;25(1):64–71.
57. Looker AC, Johnston CC, Wahner HW, et al. Prevalence of low femoral bone density in older US women from NHANES III. J Bone Miner Res. 2009;10(5):796–802.
58. Gillette-Guyonnet S, Nourhashemi F, Lauque S, Grandjean H, Vellas B. Body composition and osteoporosis in elderly women. Gerontology. 2000;46(4):189–93.
59. Douchi T, Yamamoto S, Oki T, Maruta K, Kuwahata R, Nagata Y. Relationship between body fat distribution and bone mineral density in premenopausal Japanese women. Obstet Gynecol. 2000;95(5):722–5.
60. Blain H, Vuillemin A, Teissier A, Hanesse B, Guillemin F, Jeandel C. Influence of muscle strength and body weight and composition on regional bone mineral density in healthy women aged 60 years and over. Gerontology. 2001;47(4):207–12.
61. Bakker I, Twisk JWR, Van Mechelen W, Kemper HCG. Fat-free body mass is the most important body composition determinant of 10-yr longitudinal development of lumbar bone in adult men and women. J Clin Endocrinol Metab. 2003;88(6):2607–13.
62. Grinspoon S, Thomas E, Pitts S, et al. Prevalence and predictive factors for regional osteopenia in women with anorexia nervosa. Ann Intern Med. 2000;133(10):790–4.
63. Paccou J, Zeboulon N, Combescure C, Gossec L, Cortet B. The prevalence of osteoporosis, osteopenia, and fractures among adults with cystic fibrosis: a systematic literature review with meta-analysis. Calcif Tissue Int. 2010;86(1):1–7.
64. Bonkovsky HL, Hawkins M, Steinberg K, et al. Prevalence and prediction of osteopenia in chronic liver disease. Hepatology. 2005;12(2):273–80.
65. Marcén R, Caballero C, Uriol O, et al. Prevalence of osteoporosis, osteopenia, and vertebral fractures in long-term renal transplant recipients. Paper presented at transplantation proceedings 2007; 2007.
66. Kemink S, Hermus A, Swinkels L, Lutterman J, Smals A. Osteopenia in insulin-dependent diabetes mellitus; prevalence and aspects of pathophysiology. J Endocrinol Invest. 2000;23(5):295.
67. Camacho PM, Dayal AS, Diaz JL, et al. Prevalence of secondary causes of bone loss among breast cancer patients with osteopenia and osteoporosis. J Clin Oncol. 2008;26(33):5380–5.
68. Komi PV. Strength and power in sport, vol. 3. Oxford: Wiley-Blackwell; 2003.
69. Frank C. Ligament injuries: pathophysiology and healing. J Am Acad Orthop Surg. 1996;4(2):74–83.
70. Faigenbaum AD, Kraemer WJ, Blimkie CJ, et al. Youth resistance training: updated position statement paper from the national strength and conditioning association. J Strength Cond Res. 2009;23(5 Suppl):S60–79.
71. Fukuda DH, Stout JR, Kendall KL, Smith AE, Wray ME, Hetrick RP. The effects of tournament preparation on anthropometric and sport-specific performance measures in youth judo athletes. J Strength Condition Res. 2013;27(2):331–9.
72. Michailidis Y, Fatouros IG, Primpa E, et al. Plyometrics' trainability in pre-adolescent soccer athletes. J Strength Condition Res. 2013;27(1):38–49.
73. Tournis S, Michopoulou E, Fatouros I, et al. Effect of rhythmic gymnastics on volumetric bone mineral density and bone geometry in premenarcheal female athletes and controls. J Clin Endocrinol Metab. 2010;95(6):2755–62.
74. Ward K, Roberts S, Adams J, Mughal M. Bone geometry and density in the skeleton of pre-pubertal gymnasts and school children. Bone. 2005;36(6):1012–8.
75. Zanker C, Gannon L, Cooke C, Gee K, Oldroyd B, Truscott J. Differences in bone density, body composition, physical activity, and diet between child gym-

nasts and untrained children 7–8 years of age. J Bone Miner Res. 2003;18(6):1043–50.
76. Ratel S. High-intensity and resistance training and elite young athletes. Med Sport Sci. 2011;56:84–96.
77. Faigenbaum AD, Milliken LA, Westcott WL. Maximal strength testing in healthy children. J Strength Cond Res. 2003;17(1):162–6.
78. McManus A, Armstrong N. Physiology of elite young female athletes. Med Sport Sci. 2011;56:23–46.
79. Payne VG, Isaacs LD. Human motor development: a lifespan approach. 8th. New York, NY: McGraw-Hill Humanities/Social Sciences/Languages; 2011.
80. Pescatello LS, Franklin BA, Fagard R, Farquhar WB, Kelley GA, Ray CA. American College of Sports Medicine position stand. Exercise and hypertension. Med Sci Sports Exerc. 2004;36(3):533–53.
81. Faigenbaum AD, McFarland JE, Schwerdtman JA, Ratamess NA, Kang J, Hoffman JR. Dynamic warm-up protocols, with and without a weighted vest, and fitness performance in high school female athletes. J Athl Train. 2006;41(4):357–63.
82. Roubenoff R, Castaneda C. Sarcopenia—understanding the dynamics of aging muscle. JAMA. 2001;286(10):1230–1.
83. Hasten DL, Pak-Loduca J, Obert KA, Yarasheski KE. Resistance exercise acutely increases MHC and mixed muscle protein synthesis rates in 78–84 and 23–32 yr olds. Am J Physiol Endocrinol Metab. 2000;278(4):E620–6.
84. Yarasheski KE, Zachwieja JJ, Bier DM. Acute effects of resistance exercise on muscle protein synthesis rate in young and elderly men and women. Am J Physiol Endocrinol Metab. 1993;265(2):E210–4.
85. Yarasheski KE, Pak-Loduca J, Hasten DL, Obert KA, Brown MB, Sinacore DR. Resistance exercise training increases mixed muscle protein synthesis rate in frail women and men≥ 76 yr old. Am J Physiol Endocrinol Metab. 1999;277(1):E118–25.
86. Roth SM, Ivey FM, Martel GF, et al. Muscle size responses to strength training in young and older men and women. J Am Geriatr Soc. 2002;49(11):1428–33.
87. Faulkner JA, Davis CS, Mendias CL, Brooks SV. The aging of elite male athletes: age-related changes in performance and skeletal muscle structure and function. Clin J Sport Med. 2008;18(6):501–7.
88. Newman AB, Kupelian V, Visser M, et al. Strength, but not muscle mass, is associated with mortality in the health, aging and body composition study cohort. J Gerontol A. 2006;61(1):72–7.
89. Trappe S, Gallagher P, Harber M, Carrithers J, Fluckey J, Trappe T. Single muscle fibre contractile properties in young and old men and women. J Physiol. 2004;552(1):47–58.
90. McCrory JL, Salacinski AJ, Hunt SE, Greenspan SL. Thigh muscle strength in senior athletes and healthy controls. J Strength Condition Res. 2009;23(9):2430.
91. Louis J, Hausswirth C, Bieuzen F, Brisswalter J. Muscle strength and metabolism in master athletes. Int J Sports Med. 2009;30(10):754–9.
92. ACSM. ACSM's guidelines for exercise testing and prescription. 8th ed. Baltimore, MD: Lippincott; 2010.
93. Miller MG, Cheatham CC, Patel ND. Resistance training for adolescents. Pediatr Clin North Am. 2010;57(3):671.
94. Corbin CB, LeMasurier G, Lambdin D, Greiner M. Fitness for life: elementary school guide for wellness coordinators. Champaign, IL: Human Kinetics; 2010. http://books.google.com.mx/books?hl=es&lr=&id=A5rWGLHabcQC&oi=fnd&pg=PR1&dq=Corbin+CB,+LeMasurier+G,+Lambdin+D,+Greiner+M.&ots=1Cs_OHs1vG&sig=YZS-lAmrX4ARXDMb9FSMMzoiwJk&redir_esc=y#v=onepage&q&f=false.
95. CDC. Centers for Disease Control and Prevention. Making physical activity a part of a child's life; 2011. http://www.cdc.gov/physicalactivity/everyone/getactive/children.html.
96. McCambridge T, Stricker P. Strength training by children and adolescents. Pediatrics. 2008;121(4):835–40.
97. Myer GD, Wall EJ. Resistance training in the young athlete. Operat Tech Sport Med. 2006;14(3):218–30.
98. Corbin CB, Lindsey R, Welk G. Concepts of physical fitness: active lifestyles for wellness. 16th ed. Boston: McGraw-Hill; 2011.
99. Kraemer WJ, Fleck SJ. Strength training for young athletes. Champaign, IL: Human Kinetics; 2005.
100. Stone MH, Plisk SS, Stone ME, Schilling BK, O'Bryant HS, Pierce KC. Athletic performance development: volume load-1 set vs. multiple sets, training velocity and training variation. Strength Condition J. 1998;20(6):22–31.
101. Verstegen M, Williams P. Core performance. Emmaus, PA: Rodale; 2004.
102. Faigenbaum AD, McFarland JE, Johnson L, et al. Preliminary evaluation of an after-school resistance training program for improving physical fitness in middle school-age boys. Percept Mot Skills. 2007;104(2):407–15.
103. Faigenbaum A, Milliken L, Moulton L, Westcott W. Early muscular fitness adaptations in children in response to two different resistance training regimens. Pediatr Exerc Sci. 2005;17(3):237.
104. Kraemer WJ, Newton RU. Training for muscular power. Phys Med Rehabil Clin N Am. 2000;11(2):341–68. vii.
105. Peterson MD, Rhea MR, Alvar BA. Maximizing strength development in athletes: a meta-analysis to determine the dose–response relationship. J Strength Cond Res. 2004;18(2):377–82.
106. Kraemer WJ, Ratamess NA. Fundamentals of resistance training: progression and exercise prescription. Med Sci Sport Exerc. 2004;36(4):674–88.
107. Fleck SJ, Kraemer WJ. Designing resistance training programs. Champaign, IL: Human Kinetics; 2004.

108. Stone MH, Potteiger JA, Pierce KC, et al. Comparison of the effects of three different weight-training programs on the one repetition maximum squat. J Strength Condition Res. 2000;14(3):332–7.
109. Rhea MR, Alderman BL. A meta-analysis of periodized versus nonperiodized strength and power training programs. Res Q Exerc Sport. 2004;75(4):413–22.
110. ACSM. Exercise and physical activity guidelines for older adults. Med Sci Sports Exerc. 2009;41(7):1510–30.
111. Porter MM. Resistance training recommendations for older adults. Topic Geriatr Rehabil. 2000;15(3):60–9.
112. Pollock ML, Gaesser GA, Butcher JD, et al. ACSM position stand: the recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in healthy adults. Med Sci Sports Exerc. 1998;30(6):975.
113. Faigenbaum AD, Schram J. Can resistance training reduce injuries in youth sports? Strength Condition J. 2004;26(3):16–21.
114. Faigenbaum AD, Myer GD. Resistance training among young athletes: safety, efficacy and injury prevention effects. Br J Sports Med. 2010;44(1):56–63.
115. Corbin CB. The fitness for life physical activity pyramid for children. Champaign, IL: Human Kinetics; 2003.

# Exercise Precautions for the Female Athlete: Signs of Overtraining

# 23

Jacalyn J. Robert-McComb and Lauren Gates

**Abstract**

Athletes are constantly aiming to be the best they can be by incorporating a strict nutritional and training schedule into their daily lives. When athletes push themselves further than their bodies will allow, a state of staleness will occur. Overreaching, overtraining, and the overtraining syndrome are three different levels of staleness possible if an athlete does not take the proper precautions to prevent these conditions from occurring. Within these conditions, functional, metabolic, psychological, and physiological limitations are commonplace. A full recovery can last anywhere from a few days to years in the most extreme cases. In the past, these three terms were considered one and the same; however, recent research findings have split them into three distinct conditions with defining signs and symptoms. Some of these signs and symptoms overlap with those of clinical depression, so it is imperative for an athlete to be medically evaluated in order to determine the actual cause of their symptoms. If an athlete is diagnosed with overtraining, their symptoms can be used to determine which physiological pathway is causing the condition. Research has discovered a couple of possible new mechanisms for overtraining: the negative feedback system and protein deficiency. It is important to catch overreaching and overtraining in their early stages so that athletes and their coaches can implement a plan of prevention rather than treatment. Coaches and parents need to regularly communicate with their athletes and help maintain a safe and healthy training regimen.

**Keywords**

Overreaching • Overtraining • Hypo-arousal • Hyperarousal • Overtraining syndrome • Negative feedback • Protein

J.J. Robert-McComb, Ph.D., F.A.C.S.M., A.C.S.M., P.D. (✉)
Department of Health, Exercise, and Sports Sciences, Texas Tech University, 3204 Main Street, Lubbock, TX 79409, USA
e-mail: jacalyn.mccomb@ttu.edu

L. Gates, M.S., A.C.S.M., C.E.S.
Department of Health, Exercise and Sport Sciences, Texas Tech University, 50 W. Lakemist Cir, The Woodlands, TX 77381, USA
e-mail: laurengates@yahoo.com

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_23, 

## 23.1 Learning Objectives

- The terms overreaching, overtraining, and the overtraining syndrome
- The differences between the symptoms of overtraining and depression
- Hypo- and hyperarousal overtraining states
- The current perception of overtraining and the appropriate prevention and treatment strategies
- Future areas of research

## 23.2 Introduction

Athletes are always striving to do their best and sometimes are willing to put their bodies through arduous amounts of stress and overload to achieve their goals. Most of the time it is athletes focused on endurance sports, such as cross-country running, swimming, or cycling, that push their bodies to extremes in order to attain that top level of performance. However, athletes alone are not always to blame for their sometimes far-reaching goals. Parents, coaches, and peers can put a great deal of pressure on athletes, causing stress and the constant willingness to please and succeed. Athletes should strive for their best effort but should also know the boundaries between safely training and overtraining.

It is important for athletes to know their full potential, but the consequences to overtraining can be significant. Functional, metabolic, psychological, and physiological limitations are commonplace in athletes who have gone beyond their potential and are suffering from staleness. When these limitations are in place and an athlete's performance is compromised, the athlete either may give up entirely or may see this as a weakness and try to push themselves even harder to compensate for their reduced abilities. Once any of the above-mentioned limitations have occurred due to an increase in training, overtraining has occurred. It is important for coaches and athletes to recognize the signs and symptoms of overtraining to prevent this illness from fully developing.

There are different levels of overtraining depending on how long after the first sign of symptoms the athlete continued to train. The term overtraining relates to a decrease in performance despite continuing or an increase in training [2]. Overtraining usually occurs when an athlete is exposed to high volumes of training with inadequate periods of rest. This ultimately can lead to an altered mood and reduced exercise performance. The collective impairments an athlete experiences during overtraining are also sometimes referred to as the overtraining syndrome.

## 23.3 Research Findings

### 23.3.1 The Terms Overreaching, Overtraining, and the Overtraining Syndrome

The terms overreaching, overtraining, and the overtraining syndrome were once used interchangeably; however, the present understanding is that these three terms are different based on their physiological characteristics. There are a few key differences between overreaching and overtraining that can be explained by grouping athletes into one of three categories: functional overreaching, nonfunctional overreaching (more commonly known as overtraining), and the overtraining syndrome [5]. See Table 23.1 for a review of these terms.

Functional overreaching is the appropriate term to use when an athlete is in the stages just prior to overtraining [1]. This type of overreaching can be a normal variant of athletic training. While functional overreaching does also originate from a training load that exceeds and athlete's capabilities, the effects are short lived lasting approximately a few days to a few weeks for a full recovery. The symptoms associated with functional overreaching include an amplified resting heart rate, a lowered work capacity, unusual fatigue during training, an increase in thirst, and an increase in submaximal heart rate. If an athlete does not acquire an appropriate amount of time for rest and recovery, they could develop into a state of overtraining, or nonfunctional overreaching. The terms nonfunctional overreaching and overtraining can be used interchangeably.

**Table 23.1** Overreaching, overtraining, and the overtraining syndrome

| Condition | Definition | Time to recovery | Signs and symptoms | Normal or abnormal part of training |
|---|---|---|---|---|
| Functional overreaching | Condition that occurs quickly due to an increase in training volume or intensity | Short term: a few days to a few weeks | • Slight fatigue<br>• Slight decrease in work capacity | Normal |
| Nonfunctional overreaching (overtraining) | Condition that occurs slowly over 1–2 months due to an increase in training volume with inadequate time for recovery | Moderate: several weeks to months | • Increase in resting heart rate<br>• Fatigue during training<br>• Decreased work capacity<br>• Increase in heart rate during submaximal workloads<br>• Increase in thirst<br>• Loss of muscle strength and coordination | Abnormal |
| The overtraining syndrome | Severe case of overtraining due to an increase in training volume or intensity with inadequate time for recovery | Long term: several months to years | • Premature fatigue<br>• Decrease in performance<br>• Mood changes<br>• Emotional instability<br>• Decreased motivation<br>• Loss of appetite<br>• Sleep disturbances<br>• Altered cortisol levels<br>• Decreased VO2 max | Abnormal |

Adapted from (1) Bandyopadhyay, A, Bhattacharjee, I, et al. Physiological perspective of endurance overtraining—a comprehensive update. AJMS. 2012; 5: 7–20.; and (2) Ehrman, JK, deJong, A, Sanderson, B, et al., eds. ACSM's Resource Manual for Guidelines for Exercise Testing and Prescription. 6th ed. Baltimore, MD: Lippincott Williams & Wilkins; 2010: 246, 486, 543

Overtraining is simply the term used to describe when an athlete's training load surpasses their ability to recover from that load. This type of overtraining may last from several weeks to several months, and performance capacity is usually restored with adequate amounts of rest.

Lastly, the overtraining syndrome is a serious condition that consists of not only the physical consequences of overtraining but the emotional and behavioral conditions as well [1]. The overtraining syndrome is a chronic condition lasting for weeks or months at a time. Some of the symptoms associated with the overtraining syndrome are a poor performance in competitions, chronic fatigue, an inability to maintain training intensities, reduced immune function, sleep disturbances, an altered mood state, and a reduction in catecholamine production. If not recognized in its early stages, the overtraining syndrome could last anywhere from several months to years. It is important for coaches and athletes alike to be able to recognize these conditions so that the athlete has sufficient time to recover.

### 23.3.2 Overtraining or Depression?

The signs and symptoms of overtraining and the overtraining syndrome may seem to be easily recognizable, but major depression exhibits many of the same signs and symptoms [2]. In some cases, it is hard to differentiate between these conditions. In order for someone to be diagnosed with major depression, they must have the signs and symptoms for at least 2 weeks [5]. The condition of overtraining, or nonfunctional overreaching, also begins at around 2 weeks of exhibiting signs and symptoms. Some of the overlapping signs and symptoms between these two medical conditions include sleep deprivations, a loss of

appetite, a change in mood state, a reduced body weight, and an overall loss of motivation. The overtraining syndrome and major depression additionally share common central nervous system structures, endocrine, and immune pathways that could confuse the two conditions. Because many times athletes of the same sport show different signs and symptoms from their peers, diagnosing them as having symptoms of overtraining versus major depression can be challenging.

It has been reported that about 80 % of athletes with any form of overtraining had a similar makeup as those with major depression [5]. Many athletes, especially those at the elite level, are highly motivated and will put their bodies through almost any form of stress in order to be the top performer in their sport. When these athletes begin to show signs of overtraining, they may not realize they have a medical condition and attribute their poor performance to undertraining. With so much pressure to do well, athletes will typically increase their training regimen even further in order to counteract their loss of momentum. However, in an attempt to increase their capabilities, the opposite effect occurs; physical and emotional fatigue is increased, and their performance continues to diminish. Eventually, this cycle of constantly needing to improve causes mood, sleep, and appetite disturbances and could eventually lead to a diagnosis of major depression in addition to overtraining or the overtraining syndrome.

### 23.3.3 Hypo-arousal and Hyperarousal Overtraining States

The overtraining syndrome is a very serious condition, and researchers are now proposing that there may be two distinct forms of the disorder. The first of these newly proposed states is termed the hyperarousal, or sympathetic, overtraining syndrome [3]. This form occurs mainly in "power" or anaerobically centered athletes such as sprinters and weight lifters. The sympathetic form is considered to occur only in rare circumstances due to an increase in sympathetic tone, or an increased activation of the sympathetic nervous system, during rest [1]. Some signs and symptoms of sympathetic overtraining include fatigue, sleeplessness, weight loss, night sweats, palpitations, an increased basal metabolic rate, a delayed recovery of pulse rate, an increase in ventilation during exercise, reduced coordination, and restlessness.

The hypo-arousal, or parasympathetic, state is much more prevalent and occurs in endurance or aerobically centered athletes such as swimmers and cyclists [3]. In the same manner as the sympathetic state, the parasympathetic overtraining state is a result of increased parasympathetic tone, or an increased activation of the parasympathetic nervous system, during rest and exercise [1]. There are fewer signs and symptoms for the parasympathetic state including fatigue and apathy, depression, an increased number of infections, a decreased lactate response to exercise, and possible amenorrhea in female athletes [1, 3]. It is still unclear from the present research whether or not the hyper- and hypo-arousal syndromes are caused by neurological and endocrine pathways or are simply symptoms of other mechanisms. Yet there is some research emerging linking the overtraining syndrome to neuroendocrine and immune systems [8].

## 23.4 Contemporary Understanding of the Issue

Athletes many times pursue extreme measure to improve their performance with athletes of many sports training twice a day for hours a time [4]. These twice-a-day practices do not always allow for ample recovery and lead to a state of overtraining. Since overtraining has become more and more apparent, different mechanisms have been hypothesized as to the origin of overtraining. One hypothesis is that the body is using the state of overtraining as a negative feedback mechanism for the constant state of overload without recovery [1]. Because of negative feedback, there is a decrease in neuromuscular excitability as well as a decrease in adrenocorticotropic

**Table 23.2** Prevention strategies and treatment options for overtraining [1, 3, 6]

| Strategies for prevention | Strategies for treatment |
|---|---|
| • Individualized training protocols | • Allow ample time for recovery |
| • Progressive increases in training load by about 10 % per week | • Reduce training volume and/or intensity |
| • Training volume and intensity should be inversely related | • Work different sets of muscles on alternating days |
| • Resist performing every exercise session to absolute exhaustion | • Increase the amount of sleep |
| • Maintain appropriate technique with the appropriate number of repetitions for specific muscle and joint exercises | • Get a deep-tissue or sports massage for affected muscles |
| • Take frequent breaks during the training session | • Cryotherapy or thermotherapy |
| • Switch up the training sessions to avoid monotony | • Temperature or contrast therapy |
| • Maintain a balanced diet with adequate carbohydrates, proteins, fats, nutrients, and electrolytes | • Hydrotherapy |
| • Maintain proper hydration | • Make sure calories taken in matches calories expended |
| • Have regular medical examinations administered | • Maintain a balanced diet with the appropriate macronutrient ratios |
| • Administer an interactive coaching style | • Take daily vitamins for any vitamin deficiencies |
| • Athletes should listen to their bodies | • Cross-training |

Adapted from (1) Bandyopadhyay, A, Bhattacharjee, I, et al. Physiological perspective of endurance overtraining—a comprehensive update; (2) Hackney, AC, Battaglini, C. The overtraining syndrome: neuro-endocrine imbalances in athletes. BRJB. 2007; 34–44; and (3) Overtraining and athletes: Learn the signs and symptoms of overtraining syndrome in athletes. http://sportsmedicine.about.com/cs/overtraining/a/aa062499a.htm. Accessed April 18, 2012

hormone (ACTH) sensitivity. This ultimately results in a decrease in cortisol release and a lowered metabolic response.

A second possible mechanism related to overtraining has been suggested as protein deficiency [1]. It has been observed that with overtraining, even though amino acids are consumed, the relationship between supply and demand is unbalanced. Amino acids are used more quickly than they are supplied. A suggestion could be made that increasing protein consumption could counteract the effects of overtraining [4]. With the addition of protein to a predominantly carbohydrate-rich meal, enhanced glycogen synthesis and exercise performance have been witnessed as compared to a carbohydrate-only meal. Because athletes and coaches rely on a carbohydrate-rich meal pre-competition to better performance, adding small amounts of protein may help to relieve the negative effects associated with overtraining.

It is essential that coaches and athletes are able to recognize the signs and symptoms of overtraining so that they can assist in preventing and treating this condition [1, 3]. The ever-emerging understanding of this condition is that it is not only a physical manifestation, as previously thought, but a psychological and behavioral condition as well. See Table 23.2 for a list of prevention strategies and treatment options.

If early stages of overreaching are seen, it is important for the athlete to take immediate measure to prevent the overtraining syndrome rather than start treatment after signs and symptoms have emerged [1, 3]. For coaches and athletes, it is imperative to not only look at the training preventions but preventions in the athlete's home and social life as well. The signs and symptoms can be difficult to recognize since the normal symptoms of training may mask any adverse signs. Also, because each individual athlete progresses at a different rate, individualized training programs are necessary to help prevent overtraining. As such, interactive coaching is essential. Coaches must talk to their athletes on a daily basis to see if they are progressing and improving at a healthy pace. Many times athletes will give small clues that they are frustrated or unhappy with their performance which could lead them to try even harder for success. In the same way, it is

just as important for the athlete to listen to his or her own body and speak up if they feel they are being overworked or are starting to feel an unusual amount of fatigue.

## 23.5 Future Directions

The understanding of overtraining has steadily become clearer with new appreciation about this serious condition. While the current physical and physiological prevention and treatment strategies are up to date, emotional considerations should be the focus of current research efforts [5]. The use of questionnaires is an easy and efficient way to determine an athlete's emotional standing and capabilities. Questionnaires such as the Profile of Mood States (POMS), Training-Induced Stress Scale, and other overtraining questionnaires are recommended. In addition, the additional testing of physiological markers can assist in the recognition of overtraining. Markers such as immunoglobulins (IgA), testosterone and cortisol levels, heart rate variability, and heart rate recovery are just a few. The combined results from emotionally based questionnaires and physiological markers can provide a better insight into how emotional, mood variations, and physiological markers work together.

## 23.6 Concluding Remarks

From the current research, it is becoming apparent that overtraining is not simply a physical condition but a condition that includes relationships, diet, sleep, and various other behavioral connections [7]. Early recognition of the signs and symptoms of overreaching and the beginning stages of overtraining are essential to prevent an athlete from developing the overtraining syndrome. Because the overtraining syndrome can take months to years to overcome, if an athlete does exhibit the signs and symptoms, the coach should immediately implement treatment strategies. An athlete may not be willing at first to lower their level of training, but rest and recovery is crucial to their healing. As the research surrounding overtraining carries on, further understanding and possible new prevention and treatment strategies will transpire.

## References

1. Bandyopadhyay A, Bhattacharjee I, et al. Physiological perspective of endurance overtraining—a comprehensive update. Al Ameen J Med Sci. 2012;5:7–20.
2. Ehrman JK, deJong A, Sanderson B, et al., editors. ACSM's resource manual for guidelines for exercise testing and prescription. 6th ed. Baltimore, MD: Lippincott Williams & Wilkins; 2010. p. 246. 486, 543.
3. Hackney AC, Battaglini C. The overtraining syndrome: neuro-endocrine imbalances in athletes. Braz J Biomotricity. 2007;1:34–44.
4. Lowery L, Forsythe CE. Protein and overtraining: potential applications for free-living athletes. J Int Soc Sports Nutr. 2006;3:42–50.
5. Matos N, Winsley RJ. Trainability of young athletes and overtraining. J Sports Sci Med. 2007;6:353–67.
6. Overtraining and athletes: Learn the signs and symptoms of overtraining syndrome in athletes. http://sportsmedicine.about.com/cs/overtraining/a/aa062499a.htm. Accessed 18 Apr 2012.
7. Palazzetti S, Margaritis I. Does overtraining occur in triathletes. Biol Sport. 2003;20:139–46.
8. Smith LL. Cytokine hypothesis of overtraining: a physiological adaptation to excessive stress. Med Sci Sports Exerc. 2000;32:317–31.

# Excessive Exercise and Immunity: The J-Shaped Curve

24

Carolina Chamorro-Viña, Maria Fernandez-del-Valle, and Anna M. Tacón

**Abstract**

Exercise modulates the innate and specific arms of the immune system with a marked intensity-dependent response. This response might be influenced by sex differences and other factors including age, nutrition status, and overall level of psychological stress.

Exercise immunology is the field that studies this area. In the 1990s, Dr. Nieman formulated the controversial "J-shaped hypothesis" to describe the relationship between exercise intensity and the risk of acquiring upper respiratory tract infections (URTI). This hypothesis suggests that moderate exercise has the ability to improve immune function above sedentary levels while high intensity exercise depresses the immune system. However, some methodological problems exist in studies of the J-curve which makes evidence more anecdotal than evidence-based regarding the role of moderate and intense exercise in the incidence of URTI. These limitations are presented in the chapter.

**Keywords**

Upper respiratory tract infection • J-Curve • Immune system • Exercise • Female athletes • Dietary supplements and exercise

C. Chamorro-Viña, Ph.D. (✉)
Faculty of Kinesiology, Biomedicine and Health's Sciences, University of Calgary, 2500 University Dr NW, Calgary, AB, Canada T2N 1N4
e-mail: carolinachamorro1@hotmail.com

M. Fernandez-del-Valle, Ph.D.
Department of Health, Exercise, and Sport Sciences, Texas Tech University, Exercise and Sport Sciences Building, 3204 Main, Lubbock, TX 79401, USA
e-mail: maria.fernandez-del-valle@ttu.edu

A.M. Tacón, Ph.D.
Texas Tech University, HESS, Main Street, Lubbock, TX 79409, USA
e-mail: anna.tacon@tu.edu

## 24.1 Learning Objectives

After completing this chapter you should be able to:

1. Understand the J-Shaped curve model.
2. Describe the effects of moderate and intense exercise on immune system.
3. Understand the possible mechanisms of exercise-induced change in immunosurveillance.
4. Distinguish most important sex differences in the immune system.

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*, DOI 10.1007/978-1-4614-8884-2_24, 

5. Describe the effects of some dietary supplements on the immune system function.
6. Realize the limitations in this research field.
7. Know the expertise recommendations to perform exercise training during and after upper respiratory tract infection (URTI).

## 24.2 Introduction

Exercise modulates the innate and specific arms of the immune system with a marked intensity-dependent response that might be influenced by sex differences [1] and other factors including age, nutrition status, and overall level of psychological stress.

Exercise immunology is the field that studies this area. This is a brand new scientific discipline that has experienced increased growth in the last decade with more than 80 % of articles published from 1990 onward [2]. One of the factors underlying the fast growth of this discipline is the commonly held belief that the frequency of URTIs is high in elite athletes after a single bout and/or during periods of intense training [3, 4]. On the other hand, URTIs, usually described as the common cold, are the most frequent occurring illness among humans worldwide. More than 200 viruses cause URTI with influenza, rhinovirus, and coronaviruses being the most common [5].

Despite preventive efforts, influenza epidemics are responsible for substantial morbidity and mortality every year in the United States. A huge economic burden, as described by Molinari et al., includes outpatient visits, hospitalization, and mortality, as well as time lost from work; indeed, estimated costs are $87.1 billion annually [6].

The first epidemiological study in the 1980s documented a twofold to sixfold increase in the risk of developing respiratory symptoms by participants in a marathon [3, 7]. This observational study motivated Nieman in the 1990s to formulate the controversial "J-shaped hypothesis" to describe the relationship between exercise and URTI. The term J-curve is used in several fields, which, in our case, describes the relationship between exercise intensity and susceptibility to infections [8, 9]. This hypothesis suggests that moderate exercise has the ability to improve immune function above sedentary levels while high intensity exercise depresses the immune system [4, 10, 11] (Fig. 24.1).

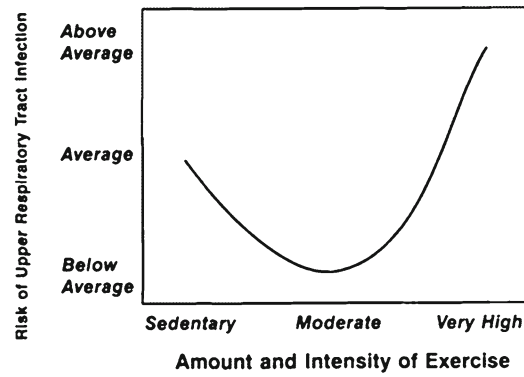



**Fig. 24.1** "J"-shaped model of relationship between varying amount of exercise and risk of URTI. This model suggests that moderate exercise may lower risk of respiratory infection, while excessive amounts may increase the risk. Reproduced from Nieman D.C: Exercise, infection and immunity. International Journal of Sports Medicine 1994 15: S131–S141, with kind permission of Georg Thieme Verlag

Intense exercise-induced immunodepression has a multifactorial origin. This includes (1) increased number of neutrophils with decreased number of lymphocytes in the blood; (2) impaired phagocytosis and neutrophil function; (3) decreased oxidative burst activity; (4) decreased natural killer cell cytolytic activity (NKCA); and (5) diminished immunoglobulin levels [8]. However, none of these functions reflect immune function as whole, and the immune system has multiple functions to protect the human body against pathogens. Therefore, measurements of blood leukocyte subtypes (e.g., Europhiles, lymphocytes) by number and function (e.g., natural killer cell activity) may not reflect the immunocompetence as a whole. Also, the host immune system is sensitive to many factors such as age, gender, nutrition state, and stress; consequently, it is difficult to predict the overall effects of small to moderate changes on immune parameters on host resistance. For this reason, the incidence of URTI is the most useful outcome from a clinical point of view [8].

Some methodological problems exist in studies of the J-curve, which makes evidence more anecdotal than evidence-based as to the role of moderate and intense exercise in the incidence of URTI [4]. A recent systematic review conducted

by Moreira et al. identified 162 relevant publications using the key search terms of URTIs and exercise. From these, only 30 were categorized as high quality observational studies that used the Newcastle-Otawa scale "star system," and studies using multiple doses of exercise are limited. As a result, the "J" curve hypothesis has been built based on a combination of results from observational and case series surveys [8]. Herein lies the controversy of this hypothesis.

## 24.3 Research Findings

### 24.3.1 Moderate Exercise and Risk of URTI

The evidence suggests that moderate regular exercise may be beneficial to diminish the risk of URTI. However, research in this field is scarce compared to the evidence documented regarding heavy exercise and URTI [8]. When the "J-shaped curve" hypothesis was first proposed, little had been published about moderate exercise-induced alteration in immune system. Activity in this field has emerged in recent years in response to an increasing interest to find factors that may lessen influenza's incidence and severity and/or improve influenza's vaccine efficacy [12].

Most epidemiological studies support the notion that moderate levels of physical activity reduce URTI incidence [13]. However, important methodological limitations have been described and will be discussed later in this chapter.

A study performed in mice by Lowder et al. showed a protective effect of moderate treadmill exercise at 65–70 % of their maximal oxygen uptake ($VO_{2max}$) in mice who exercised 3 days after influenza virus inoculation yet before the onset of flu symptoms. Meanwhile, prolonged exercise (2.5 h) led to increased morbidity and decreased survival, and moderate exercise significantly decreased mortality rates, even when compared with sedentary controls; specifically, 83 % survived in moderate when compared with 43 % survived in the control group [12].

The American Colleague of Sport Medicine's (ACSM) physical activity guidelines for healthy adults recommend that most adults should engage in moderate-intensity cardiorespiratory exercise training for ≥30 min/day on ≥5 day/week for a total of ≥150 min/week (see Chap. 20). The effects of a moderate bout of 30 min brisk walking on the immune system function were investigated by Nieman et al., in healthy non-obese women. Results showed that walking between 60 and 65 % of $VO_{2max}$ compared with the same participant sitting as a non-exercise control induced discrete and short-lived increases primarily in Natural Killer cells (NK) and neutrophils [14].

Mitogen-induced leukocyte proliferation has been observed immediately after walking due to an increase in serum T cells. Furthermore, IL-6 cytokine showed a small yet significant increase, while cortisol levels, salivary IgA or plasma IL-1ra concentration remained unchanged. These changes are inconsistent with those reported during prolonged and intensive exertion such as large increases in leukocyte, neutrophil and monocytes counts, plasma cortisol concentration, plasma IL-6 and IL-1ra concentration, and extensive postexercise decline in IgA secretion rate, NK count and activity, and mitogen-induced lymphocyte proliferation [14, 15]. Several findings support the stance that moderate exercise induces favorable changes in immunosurveillance [16–21], yet the mechanisms underlying exercise reduced URTI risk have not been fully identified [14, 22]. One study revealed that lung macrophages play an important role in mediating the beneficial effects of moderate exercise on susceptibility to respiratory infections. Specifically, mice were assigned to one of four groups: exercise and resting control with and without clodronate encapsulated liposomes ($CL_2$MDP-lip) (a substance used to deplete tissue macrophages). The results showed that regular moderate exercise decreased morbidity by 36 %, mortality by 61 %, and symptom severity score. Davies et al., however, demonstrated in another study with mice that alveolar macrophage antiviral resistance is suppressed after exercise until fatigue, and also after 30 min of a single bout of exercise. Mice were virus inoculated after the exercise session with suppression after 30 min of

exercise lasting for 3 h, and then resolved after 8 h. Increased mortality rate was only observed in mice that exercised to the point of fatigue. At first glance, these in vitro results may appear to contradict findings of Murphy et al., yet the different results could be explained by differences in immune system regulation between a single acute bout of moderate exercise and a regular moderate bout of moderate exercise performed at least 6 days before inoculation.

Data from animal studies have been difficult to apply to human participants, but, in general, are consistent with the finding that heavy exercise bouts after virus inoculation may lead to high morbidity and mortality rates [12, 22–24]. Nevertheless, data in this area are still insufficient [24].

In a large epidemiological study conducted in 641 healthy nonathlete adults, moderate exercise was found to decrease the risk of URTI in 20–30 %—especially during the summer and fall. Unfortunately, the use of self-report methods to assess URTI and physical activity, as well as the absence of a formally validated tool to assess URTI, limits the reliability of these findings [13]. Additionally, no differences were reported between exercise and sedentary control groups in an intervention study where 50 moderate fit young adults reported severity and duration of Rhinovirus. Specifically, the exercise group trained 10 days at 70 % of heart rate reserve during 40 min sessions, beginning the first day of virus inoculation. This well-designed control intervention study did not find any difference in the self-reported outcomes of severity and duration of the virus infection between exercise and sedentary control participants [25]. These results indicate that exercise may be safe during virus infection if the exercise is performed at moderate intensity.

Additionally, several case–control studies regarding the chronic effect of exercise training performed in elderly participants have been conducted. Specifically, comparison between active and sedentary subjects showed that the latter have a greater risk of developing URTIs [8]. These findings suggest that moderate regular exercise may protect the host against URTIs.

### 24.3.2 Exhaustive Exercise and Risk of URTI

Epidemiological research suggests that athletes engaged in strenuous exercise (e.g., a marathon) or an intense training period, such as near a competition, are at greater risk of URTI. After prolonged or intensive exercise bouts, many components of the immune system reflect physiological stress and immunodepression. This period commonly lasts 3–72 h, and commonly is known as the "Open window."

Nieman, a recognized researcher in the field, conducted an epidemiologic survey-based study of Los Angeles Marathon (LAM) participants to investigate the relationship between self-reported infectious episodes, previous training performed, and the intensity at which the race was run. Results showed that runners under heavy training—defined as more than 97 km/week—may be at double risk of infectious episodes compared with those who trained equal to or less than 32 km/week. Also, runners who participated in the LAM race experienced a greater incidence of URTI compared to runners who did not run in the race [26]. These data suggest that other than exercise intensity, cumulative training affects URTI risk. This is consistent with findings from other studies [27, 28].

A recent epidemiological study conducted by Ekblom et al., did not show any relationship between training volume 6 months previous to the Stockholm Marathon in Sweden and the increase to acquire an infection in the weeks following this competition [27]. Specifically, the rate of URTI was 16 % before the competition, which remained stable 3 weeks afterwards in the runners without URTI symptoms before competition. The researchers did suggest that exercise stress may play an important role in virus reactivation for those participants who had a virus 3 weeks before competition; this is because they showed a 33 % risk of URTI following competition [27]. Also, faster finishing time in relation to prerace fitness seems to be a risk factor, especially in younger runners.

Cross-sectional study compared immune function and infections rates in nonathletic females vs. female's elite rowers [29]. Findings showed

significantly higher phytohemagglutinin-induced lymphocyte proliferation and NKCA in the elite rowers. However, the numbers of days with URTI symptoms during the spring season did not differ between both groups [29]. Also, another study of 12 national-level swimmers under intense exercise training showed that neutrophil oxidative activity was significantly lower compared to sex-matched sedentary participants. Nevertheless, URTI rates did not show differences between the swimmers and the control group [30]. In sum, findings are not consistent in this area. Indeed, Moreira et al. suggest that the risk of URTI may be dependent upon each participant and not solely on exercise intensity [8].

The results do suggest that although exercise induces changes in immune functions, these not always are associated with risk of URTI [30, 31].

### 24.3.3 Effects of Exercise on Innate Immune Cell Count and Function

This section will focus primarily on the influence of acute exercise on cellular components of innate immunity due to regular exercise training, which does not appear to alter peripheral blood leukocyte counts [4]. General information as to the influence of chronic exercise also will be discussed.

Leukocytosis (increase in leukocyte number) in peripheral blood is noticed during an exercise bout. Leukocytosis depends on exercise type, intensity, duration, and is attenuated by exercise training [4, 11]. Exercise up to 30 min leads to an increased leukocyte cell count, which then returns to baseline levels within 10–30 min after exercise. This period of leukocyte level returning to baseline may be longer if exercise duration is longer [1]. Researchers originally attributed this leukocytosis to hemoconcentration. Nonetheless, this theory was rejected because the fluid loss from plasma during exercise does not fully explain the large increase in peripheral blood leukocytes, which may be more than double at maximal aerobic exercise [32]. A biphasic response characterized this leukocytosis. A redistribution of leukocytes from marginal pools has been proposed to explain this sharp increase that occurs in the first phase. At least two factors have been postulated to be responsible for this redistribution: (1) the increase in cardiac output that induces a greater mechanical force that drags leukocytes from blood vessels walls; and (2) catecholamine release that might be responsible for a down regulation of leukocytes and/or endothelial cells adherence capacity [4, 11, 32]. However the second increment also called "delay leukocytosis" is present only in extensive aerobic exercise (>1 h) and 2–4 h after a short bout of exercise. This occurs almost exclusively due to an increased number of neutrophils that are released from bone marrow [11]. This increase in neutrophils, moreover, likely is mediated by an increase in granulocyte colony stimulating factor (G-CSF) rather than from epinephrine or cardiac output [1]. Previous studies in which cortisol was infused in healthy participants demonstrate that increments in cortisol plasma levels seem to be responsible for the delayed leukocytosis. Therefore, recent evidence shows that short bouts of intense exercise may cause this delay in the release of neutrophils from the bone marrow [11, 33]. Exercise intensity of less than 50 % of $VO_{2max}$ does not elevate plasma cortisol levels.

Monocytes and macrophages cells seem to be affected by patterns similar to neutrophils. Indeed, research has demonstrated that counts of neutrophils and monocytes circulating in blood may increase by about 90 % after an intensive bout of exercise [32, 33]. Neutrophils are the predominant circulating leukocytes with importance in the nonspecific immune function by phagocyte bacteria, virus, and protozoa, which is followed by intracellular digestion mediated by granular hydrolytic enzymes and reactive oxygen species. Moderate intensity exercise may enhance neutrophils' respiratory burst activity, mediated by increase in inflammatory cytokine IL-6 [11]. Degranulation appears to be mediated by exercise because a postexercise plasma elevation in concentrations of elastase and myeloperoxidase has been found [11]. Prolonged and acute exercise do not appear to provoke neutrophil degranulation changes during exercise, yet a great

reduction of elastase release per neutrophils has been observed 2.5 h postexercise in response to bacterial stimulation. Also, intense or long duration exercise may suppress the production of reactive oxidants via elevated circulating concentrations of epinephrine (adrenaline) and cortisol [34]. In principle, improved responsiveness of neutrophils to stimulation following exercise of moderate intensity could mean that individuals participating in moderate exercise may have improved resistance to infection. On the other hand, competitive athletes undertaking regular intense exercise may be at greater risk of URTI. To investigate the long-term effect of endurance training (>10 years) on immunity, a study assessed phagocytic activity of circulating neutrophils at rest and after a submaximal bout of exercise in well-trained cyclists ($VO_{2max}$ 61.0 ml/kg/min) and sedentary age-matched controls ($VO_{2max}$ 37.4 ml/kg/min). Results showed that circulating neutrophil phagocytic capacity was approximately 70 % lower in trained individuals at rest compared to that of control participants. Therefore, prolonged periods of endurance training may lead to increased susceptibility to opportunistic infections by diminishing immune function at rest [35]. Table 24.1 reflects the incidence of different types of exercise on neutrophil function [11, 15, 34]. Moreover, the effects of exercise on the neutrophil function have been discussed in detail by Peake [34].

Monocytes/macrophages make up around 10–15 % of leukocytes, and their main function is phagocytic and to kill pathogens. Additionally, they play an important role in mediating the acquired immune system response to an antigen presenting cell. Basically, an antigen presenting cell is responsible for displaying a fragment of the antigen that is bound to a class II MHC molecule on their membrane. Specifically, this is accomplished by either phagocytosis or receptor-mediated endocytosis. This way, a T cell can recognize and react to the antigen; however, T cells can only recognize antigens that have been processed and presented by cells via an MHC molecule. After a prolonged bout of intense exercise, the expression of some Toll-like receptors (TLR) on monocytes decreases. TLRs are receptors that recognize molecules, which are broadly shared by pathogen that allow antigen presenting cells to recognize pathogens [11]. The decrease of TLR 1, 2, 4 was described by Lancaster et al. [36]. Further studies are needed to clarify whether this decrease is real or merely reflects a decrease in monocytic count. A decrease in IL-6, IL-1α, and TNF-α production also has been associated with a reduction in TLR expression [4].

### 24.3.4 Role of NK Cells in Immunosurveillance

The mechanism underlying moderate exercise-induced decreases in URTI has not been identified completely; however, improved NKCA may play a role. NK cells are a heterogeneous subpopulation of lymphocytes that are the most responsive immune cells to acute exercise. They are part of innate immunity and their main function is to destroy virus and tumor cells. Two main NK cell subtypes have been described; that is, NK cells bright ($CD56^{bright}$) and NK cells dim ($CD56^{dim}$). NK cells dim are the expressed high level of CD16 and are the most cytotoxic subtype. NK cells bright express absent or low levels of CD16 resulting from high levels of cytokine production. They appear to play an important role in early immune challenge by coordinating the action between the innate and adaptive arms of the immune system. The decision to lyse a target cell is made by these cells via a complex signal system in which activating and inhibiting signals or the Killer immunoglobulin-like receptor (KIR) is involved. NK cell activating signals should dominate over inhibiting signals to lyse the target cell. If an NK cell is engaged with a major histocompatibility complex (MHC) class 1 molecule, the inhibitory KIR is activated, and the presenting signal prevents NK cells from killing host cells (non-MHC restricted) [37]. In response to an aerobic or anaerobic exercise, NK cells are mobilized faster into the peripheral blood circulation. A review published by Timmons et al. in 2008 states that this mobilization is associated with improved the immune system function, which reduces the risk

**Table 24.1** Effect of acute and chronic exercise on neutrophils functions

| Neutrophils function | Acute moderate exercise | Acute exhaustive exercise | Chronic exercise at rest | Description |
|---|---|---|---|---|
| Chemotaxis | ↑ | ↔ | ↓ ↔ | Ability to migrate to other tissue guided by chemical signals |
| Adherence | ↔ | ↔ | ↓ | First stage of diapedesis |
| Phagocytosis | ↑ | ↓ | ↓ | Action to engulf the pathogen |
| Neutrophils degranulation | ↑ | ↓ | ↔ ↓ | Digestion of microorganism by releasing granular lytic enzymes |
| Oxidative burst activity | ↑ | ↔ | ↔ | Digestion of microorganism by generating reactive oxygen species (ROC) |

Increase, ↑; decrease, ↓; no change, ↔

References: Gleeson et al. [11]; Walsh et al. [4, 10]; and Pedersen and Hoffman-Goetz [15]

of virus acquisition [37]. However, a biphasic response in the NK cell function has been described after intense exercise. This NK cell cytolytic response may result from an exercise-induced imbalance in Th1/Th2 lymphocytes. Research shows that Th1 lymphocytes are suppressed after intense exercise due to corticosteroid effect. IL-2, mainly secreted by Th1 lymphocytes, is an important cytokine responsible for stimulating NKCA [11, 38, 39]. This NKCA reduction appears to make the host more susceptible to invasion by microorganisms—especially to viruses [40, 41]. Moderate regular exercise has been found to increase NK cell function in both sedentary individuals and cancer patients [39, 42, 43]. A study conducted in obese women showed that 6 weeks of walking for 45 min, 5 days/week, produced a 57 % increase in NKCA compared with the 3 % increase found in the control group [44]. A marked increase in NK cell count, generally, is documented at the end of exercise, which may be due to a catecholamine-mediated demargination of cells. The opposite occurs several hours after exercise; specifically, the NK cell counts drops about 50 % compared with normal levels. In general, the normal resting value is restored in a couple of hours or within 24 h, except in prolonged intense and stressful exercise which might take longer. Also if activity is both prolonged and vigorous, the decrease in NK cell counts and NKCA may begin during the exercise session [39]. This decrease in NK cell number and cytolytic activity seems to be irrelevant for recreational participants, but becomes more important in athletes who might experience this immunosuppression several times per week.

### 24.3.5 Possible Mechanisms Underlying Immune Suppression Through Intensive Exercise

The role that intense exercise plays in the risk of URTI has multiple potential explanations. Commonly a temporary drop in circulating NK cells is observed after a vigorous exercise bout, which generally lasts for several hours. At first sight, this period of time seems to allow easier access to infected microorganism and is commonly known as "open window." NK cells count and activity typically have been described as being depressed for only a few hours. The new technology has revealed that high intensity exercise does not destroy NK cell. Instead, high intensity exercise is responsible for catecholamine secretion that increases the activation of adhesion molecules; therefore, NK cells are redistributed to reservoir sites such as walls of peripheral veins [4]. Information about the role of NK cells and moderate exercise was reviewed earlier in this chapter.

Changes in salivary IgA (s-IgA) immunoglobulin concentration and secretion rates have been postulated as the only consistent immune measures to date responsible for the increase in upper respiratory symptoms (URS) [3, 4, 11, 32]. The immunoglobulin-A is the predominant Ig in mucosal secretions that, in concert with innate mucosal defenses, provide the "first line of defense" against pathogens and antigens presenting at the mucosa [45]. Meanwhile, research has shown that prolonged high intensity exercises provoke a fall in s-IgA concentration, while short and moderate exercise increases it [45–52]. Decreased secretion rate of s-IgA have been implicated as risk factors for subsequent episodes of URTI and URS in athletes and non-athletes [4]. The mechanism underlying exercise-induced modifications in s-IgA remain unclear. Furthermore, adrenaline seems to be partially responsible. Chronic stress, like intensive training, is associated with diminished functioning of the hypothalamic–pituitary–adrenal axis (HPA), suppressed effects on IgA synthesis, and/or transcytosis (process by which various macromolecules are transported across the interior of a cell) [4].

In 2011, a group of highly recognized researcher in the field announced by consensus that high incidence of infections are reported in individuals with selective deficiency of s-IgA very low saliva flow rates. Also, decreases in s-IgA may occur during intensive periods of training. The main limitation in the field is that

most of the studies were conducted in military populations. Military intensive training introduces a wide range of bias because this intensive training usually is accompanied with dietary energy deficiency, sleep deprivation, and psychological stress. The majority of these factors also may alter the immune response. These stressful factors appear to amplify the exercise-induced alteration.

### 24.3.6 Role of Exercise on the Acquired Immune System

The T- and B lymphocytes are part of the acquired immune system and play an important role in the control of viral infection. Lymphocytes express a high density of $\beta_2$-adrenergic receptors, and the density of these receptors increase due to exposure to exercise or catecholamines. The NK cells are the lymphocytes that express most of the density of these receptors followed by Lymphocytes cytotoxic T cells ($CD8^+$) and Lymphocyte B cells ($CD19^+$); and Lymphocyte T helper cells ($CD4^+$) express the least amount. For that reason NK cells are more sensitive to exercise modulation, while $CD4^+$ shows the lowest response to exercise. Lymphocytosis was observed during and immediately after an acute bout of exercise followed by decreased below resting levels during early stages of recovery. The decrease is largely observable in T lymphocytes, especially Th1 subtype, while B lymphocyte showed a lesser effect. The extent of exercise mobilization is primarily dependent on exercise intensity and duration [53]. Adrenaline seems to be responsible for this biphasic response. It is not clear as to whether the decrease is due to apoptosis or redistribution. Also, the decrease in Th1 T lymphocytes count alone does not necessarily imply reduced host immunosurveillance.

The position Stand (2011) concludes that acute intensive exercise produces a depression of acquired immune system function. This depression is usually short-lived and will resolve within 24 h unless insufficient recoveries between exercise sessions cause chronic depression of acquire immunity [4]. Also, the combined effect of small detrimental changes in several aspects of host defense may compromise resistance to minor illness such as URTI [4].

## 24.4 Contemporary Understanding of the Issues

### 24.4.1 Methodological Problems that Limit the Quality of Research

Abundant anecdotal and survey data exist supporting the J-shaped curve hypothesis [23]. The limitations in this research field come from many factors that include the following: the use of self-report data, nonclinically diagnosed URTI, poorly defined exercise intensity, and lack of control over important research variables [24].

The reliability of the data collected from survey-based epidemiology studies may have been influenced by several variables of the experimental design: the participants may have been aware of the objective of the survey and consequently altered their responses; also, recall information over a long period of time may have produced potential error due to participant's boredom [11].

Most of the studies did not clinically confirm URTI infections. This is a common bias in this field of research. Therefore a sore throat reported as a URTI symptom would be the consequence of a noninfectious airways inflammation due to drying of the mucosal surfaces and or/inhalation of pollutant or dry air [4, 8, 11].

Most human studies in the exercise immunological field are limited to immune measures derived from the blood, and immunosurveillance is not fully represented by serum measures. Therefore, it is difficult to determine the impact of changes on the human immune system's ability to fight against infections [4].

Studies with athletes or those who describe the effect of acute bouts of exercise-induced change in the immune system rarely report an objective measure of exercise intensity (e.g., intensity assessed by heart rate, lactate or $VO_{2max}$ percentage) [8]. This makes the comparison of

result among studies much more difficult. Also, control of other important variables such as nutrition status, overall level of stress, and the use of nutritional supplement among others were rarely performed [8, 24].

### 24.4.2 Role of Nutrition in Diminishing the Risk of URTI

A healthy and balanced diet may supply all the required nutrients for non-athletes engaging in moderate physical activity. However, athletes might benefit from immunonutritional support to enhance immunity during heavy training periods [10].

Some studies have investigated the efficacy of vitamin C supplementation in decreasing the risk of URTI after an ultra marathon race. Peters et al. found that the consumption of 600 mg of vitamin C supplement 21 days before the race enhanced runners' resistance to post race URTI infections, a common occurrence in these competitive runners. During 14 days after the race, a significant difference was revealed with 68 % of the placebo control group reporting URTI symptoms vs. 32 % of runners who had received vitamin C supplementation [54]. Another study conducted by the same author showed that supplementation of more than 500 mg of Vitamin C supplementation least 3 weeks before a race was effective in diminishing URTI symptoms in 90 km ultra marathon runners. The ratio of URTI infections after vitamin C supplementation alone, or in combination with Vitamin E or beta-carotene, was almost halved [55].

Vitamin C (ascorbic acid) has been described as an antioxidant responsible in part for mediating free radicals neutralization. In simple terms, a reactive and possibly harmful free radical can interact with vitamin C. The reactive free radical is reduced, and the ascorbyl radical formed in its place is less reactive. This process is called free radical scavenging or quenching [56]. It is known that free radical cause inhibition of chemotaxis, phagocytosis, proliferation of T and B lymphocytes, and cytotoxic activity of natural killer cells. But the data in this area are scarce and inconclusive because other studies failed to show significant differences in the risk of URTI after a marathon by Vitamin C supplementation by runners [57]. Also, a recent publication regarding immune health by a panel of world-leading experts [10] did not recommend the use of vitamin E or Vitamin C as a supplement to improve immunosurveillance in athletes during exertive training phases. The researchers explained that vitamin E might be pro-oxidative in heavy exertion, and also, that the existing data have not shown vitamin C to be consistently different from placebo effects.

Another area where consensus is lacking is the consumption of carbohydrate beverage to attenuate immunosuppressive effect of prolonged exercise [55]. Maintaining blood glucose levels during exercise appears to diminish the secretion of stress hormones, which therefore diminishes the immune inflammatory response [10]. In terms of practical application, experts recommend to ingest up to 60 g of carbohydrate per hour of heavy exertion exercise.

Glutamine and amino acid supplement also are not recommended due to the suggestion that large glutamine body store can easily exceed exercise-lowering effects.

A recent research interest in the field of exercise immunology involves the effects of dietary polyphenols (such as quercetin) in immune system modulation because of their antioxidative, anti-inflammatory, anti-pathogenic, anticarcinogenic, and mitochondrial stimulatory activities. Forty trained male cyclists were randomized to quercetin ($N=20$) or placebo ($N=20$) groups and, under double-blind procedures, received 3 weeks quercetin (1,000 mg/day) or placebo before, during, and for 2 weeks after a 3-day period in which subjects cycled for 3 h/day at approximately 57 % Watts max. Results showed less incidence of URTI during the 2-week postexercise period in quercetin group vs. the placebo group. Although quercetin vs. placebo ingestion did not alter exercise-induced changes in several measures of immune function, it significantly reduced URTI incidence in cyclists during the 2-week period following intensive exercise. Thus, initial data appears to support the use of quercetin supplementation in athletes. Some foods rich in quercetin include black and green tea, capers, apples,

red onion, tomato, broccoli, and other leafy green vegetables.

Multiple supplements have been studied. Please refer to Table 24.2 for a summary of findings on other supplements. More details about immunonutritional can be found in the Position stand: part two [10].

### 24.4.3 Sex Differences

Notable sex differences have been documented regarding immune function. At rest, females have (1) a higher percentage of activate neutrophils and macrophages circulating (studies in rats) [58, 59]; (2) greater T lymphocyte percentage (from the total pool of lymphocytes) [60]; and (3) higher Th2 cytokine in vitro production without differences along menstrual cycle. These differences might explain why females have a lower mortality rate than males for certain types of infections, and why females have higher rates of autoimmune disease [1].

As NK cells are the most responsive immune cell to exercise, IL-6 is the most responsive cytokine. Another recent study showed that this cytokine was secreted by muscles, had a potent anti-inflammatory effect due to the up-regulation of IL-1ra and IL-10 while avoiding TNF-α (alpha) release. Also, it has been shown to regulate fatty acid oxidation and glucose uptake [61]. The secretion of this interleukin is dependent upon exercise intensity (increased response) and fitness level (decreased response).

Overall, research shows that a significant difference in cytokine release between sexes. However, a study controlling for menstrual phase and oral contraceptive (OC) found a trend of increased IL-6 in women with and without consumption of oral contraceptive at follicular phase [62]. Previous studies have found an association of the follicular phase of the menstrual cycle with greater carbohydrates consumption during exercises (see Chap. 4). Also, a decrease in carbohydrates was associated with an increase in IL-6 release [1].

The majority of research conducted regarding sex differences in leukocyte response to exercise have not found any difference between the sexes. However, important variables such as menstrual cycle, fitness level, and use of OC were not controlled. A study performed by Timmons et al. showed that after pedalling 90 min at 65 % of $VO_{2max}$ females under OC treatment showed a greater increase of lymphocytes and neutrophils compared to males and females who did not use OC. No fluctuations due to menstrual cycle were experienced in females compared to non-OC users. However the response in the follicular and luteal phases seemed to affect exercise-induced changes in leukocytes. These results seem to indicate that sex hormones are not responsible for those changes. Rather, an increase in plasma levels in women under OC has been proposed to explain that [62].

In well-controlled studies, NK cell activity appears to be sex and menstrual phase dependant with greater response in women at the follicular phase. From animal studies, estrogens appear to inhibit the inflammatory response, which limits neutrophil infiltration by acting as a cell membrane stabilizer and antioxidant. When taking this into account, it makes sense that the greatest increase in neutrophils occurs during luteal phase when estrogens levels are lower [1].

It has been proposed that the highest amount of adipose tissue in females may be responsible for the greater IL-6 release in women compared to males. Previous research demonstrates the role of the adipose tissue in IL-6 release. Also the disparity in the leukocyte response between males and females might be explained by females presenting more β-2 adrenergic receptors than males.

Estrogens do not seem to be the primary factor responsible for many of the sex differences observed in research. Thus, more research is needed to clarify cytokine release differences between the sexes, while at the same time, controlling variables like menstrual cycle, OC use, fitness level, and exercise intensity.

### 24.4.4 Recommendation to Exercise During and After a URTI

Acute URTI is the most common medical condition affecting athletes at summer and winter Olympics games [10]. Heavy training

**Table 24.2** Summary of rationale and findings for selected immunonutritional supplements

| Immunonutritional supplement | Proposed rationale | Recommendation based on current evidence |
|---|---|---|
| Vitamin E | Quenches exercise-induced reactive oxygen species (ROS) and augments immunity | Not recommended; may be pro-oxidative with heavy exertion |
| Vitamin C | Quenches ROS and augments immunity | Not recommended; not consistently different from placebo |
| Multiple vitamin and minerals | Work together to quench ROS and reduce inflammation | Not recommended; not different from placebo; balance diet is sufficient |
| Glutamine | Important immune cell energy substrate that is lowered with prolonged exercise | Not recommended; body stores exceed exercise-lowering effects |
| Branched chain amino acids (BCAAs) | BCAAs (valine, isoleucine, and leucine) are the major nitrogen source for glutamine synthesis in muscle | Not recommended; data inconclusive, and rationale based on glutamine |
| Carbohydrates | Maintain blood glucose during exercise, lowers stress hormones, and thus counters immune dysfunction | Recommended; up to 60 g/h of heavy exertion helps dampen immune inflammatory responses, but not immune dysfunction |
| Bovine colostrums | Mixture of immune, growth and hormonal factors improve immune functions and the neuroendocrine system; lower illness risk | Jury still out, with mixed results |
| Probiotics | Improve intestinal microbial flora, and thereby enhance gut and systemic immune function | Jury still out, with mixed results |
| *N*-3 PUFA (fish oil) | Exert anti-inflammatory effects postexercise | Not recommended; not different from placebo |
| β-Glucan | Receptors found on immune cells, shows supplementation improve innate immunity and reduces infection rate | Not recommended; human studies with athletes do not show any benefits |
| Herbal supplements (e.g., Ginseng, Echinacea) | Contain bioactive molecules that augment immunity and counter infection rates | Not recommended; human studies do not show consistent support within an athletic context |
| Quercetin | In vitro studies show strong evidence for anti-inflammatory antioxidative and anti-pathogenic effects. Animal data indicate increase in mitochondrial biogenesis and endurance performance; reduction in illness | Recommended when mixed with other flavonoids and nutrients; human studies show strong reduction in illness rates during heavy training and mild stimulation of mitochondrial biogenesis and endurance performance in untrained subjects; anti-inflammatory and antioxidative effects when mixed with green tea extract and fish oil |

Reproduced from Walsh NP, Gleeson M, Pyne DB, Nieman DC, Dhabhar FS, Shephard RJ, Oliver SJ, Bermon S, Kajeniene A: Position statement. Part two: Maintaining immune health. Exerc Immunol Rev 2011, 17:64–103, with kind permission of Dr. Hinnak Northoff

can increase the severity and duration of the infection. Although rare, myocarditis could develop with life-threatening consequences [11]. For that reason the tables below show practical guideline for athletes and coaches once infections have been acquired (Tables 24.3 and 24.4).

## 24.5 Future Directions

The role of moderate exercise as a preventive and therapeutic tool to treat common cold has to be explored in detail. There is increasing evidence that moderate exercise might have the capability to

**Table 24.3** Guidelines for exercise during episodes of URTI in athletes

| Day of illness | Recommendations |
|---|---|
| 1st | No strenuous exercise or competitions when experiencing URTI symptoms like sore throat, coughing, runny or congested nose<br>No exercise when experiencing symptoms like muscle/joint pain and headache, fever, generalized feeling of malaise, diarrhea or vomiting<br>Drink plenty of fluids, keep from getting wet and cold, and minimize life-stress<br>Consider use of topical therapy with nasal drainage, decongestants and analgesics if feverish<br>Report illness to team physician or health care personnel and keep away from other athletes if you are part of a team training or travelling together |
| 2nd | If fever is >37.5–38 °C, or coughing increases as well as diarrhea or vomiting: *no training*<br>If no fever or malaise and no worsening of "above the neck" symptoms: *light exercise (pulse <120 bpm*) for 30–45 min, indoor during winter and by yourself |
| 3rd | If fever and URTI or gastrointestinal infections (GI) symptoms are still present: consult your physician. Quinolones should be avoided whenever possible because of an increased risk of tendinopathy<br>If no fever or malaise and no worsening of initial symptoms: *moderate exercise (<150 bpm)* for 45–60 min, preferably indoor and by yourself |
| 4th | If no symptom relief: do not to exercise but make an office visit to your doctor<br>If first day of improved condition, follow the guideline below |

Adapted from Walsh NP, Gleeson M, Pyne DB, Nieman DC, Dhabhar FS, Shephard RJ, Oliver SJ, Bermon S, Kajeniene A: Position statement. Part two: Maintaining immune health. Exerc Immunol Rev 2011, 17:64–103, with kind permission of Dr. Hinnak Northoff

**Table 24.4** Guidelines for return to exercise after infections

| Recommendations |
|---|
| • Wait 1 day without fever and with improvement of URTI symptoms before returning to exercise |
| • Stop physical exercise and consult your physician if a new episode with fever or worsening the initial symptoms or persistent coughing and exercise-induced breathing problems occur |
| • Use the same numbers of days to step up to normal training as spent off regular training because of illness |
| • Observe closely your tolerance to increased exercise intensity and take extra day off if recovery is incomplete |
| • Use proper outdoor clothing and specific cold air protection for airways when exercising in temperatures below −10 °C the first week after URTI |

Adapted from Walsh NP, Gleeson M, Pyne DB, Nieman DC, Dhabhar FS, Shephard RJ, Oliver SJ, Bermon S, Kajeniene A: Position statement. Part two: Maintaining immune health. Exerc Immunol Rev 2011, 17:64–103, with kind permission of Dr. Hinnak Northoff

improve immunosurveillance against URTI. More interventional research is needed to determine how this effect can be maximized [3, 14]. Future studies should make an effort to improve methodological issues such as randomization, selection of subjects, and report adverse events. Additionally, future investigations need to improve the description physical activity and/or exercise intensity and to identify the source of cells producing the high amounts of cytokine in response to a muscle contraction, which will aid in identifying their role in the repair and growth of muscle [15]. Also, it is time to examine the role of exercise on clinical outcomes in various groups including patients with immune disorder or malignant disease [15].

## 24.6 Concluding Remarks

The concluding remarks that the authors feel are most noteworthy have been bulleted.

- Although the "J-shaped curve" hypothesis is generally accepted by consensus, the available experimental evidence is not enough to support it.
- Not only does exercise intensity or duration seem to be responsible for increasing the risk

of URTI, other individual factors such as age, fitness condition, nutritional status, psychological wellbeing, and previous health status increase the exposure to pathogens.

- Strenuous exercise produces quick leukocytosis that mainly is mediated by neutrophils and lymphocytes demargination from others pools. Also, delayed neutrophilia mainly provoked by neutrophils, which was released from the bone marrow was observed.
- Exercise has a biphasic response regarding numbers of circulating lymphocytes and lymphocyte subsets with an increase in the number of cells occurring during exercise as well as a decrease in cell quantity after exercise. The degree of this change is intensity and duration dependent.
- Evidence regarding immunonutritional supplements remains controversial. There is not enough evidence to recommend most of them.
- Estrogens alone are not responsible for immune sex differences in exercise-induced immune system changes. More research is needed to clarify cytokine release differences between the sexes that control variables such as menstrual cycle, OC use, fitness level, and exercise intensity.
- Cessation of exercise or reduction in the amount and intensity of exercise may improve the time of recovery from a URTI infection. However, athletic competition or training maintained at high intensity levels may increase the severity of the disease or even compromise the life of the athlete.

## References


1. Gillum TL, Kuennen MR, Schneider S, Moseley P. A review of sex differences in immune function after aerobic exercise. Exerc Immunol Rev. 2011;17:104–21.
2. Nieman DC. Current perspective on exercise immunology. Curr Sports Med Rep. 2003;2(5):239–42.
3. Shephard RJ. Development of the discipline of exercise immunology. Exerc Immunol Rev. 2010;16:194–222.
4. Walsh NP, Gleeson M, Shephard RJ, Woods JA, Bishop NC, Fleshner M, Green C, Pedersen BK, Hoffman-Goetz L, Rogers CJ, et al. Position statement. Part one: Immune function and exercise. Exerc Immunol Rev. 2011;17:6–63.
5. Nieman DC. Risk of upper respiratory tract infection in athletes: an epidemiologic and immunologic perspective. J Athl Train. 1997;32(4):344–9.
6. Molinari NA, Ortega-Sanchez IR, Messonnier ML, Thompson WW, Wortley PM, Weintraub E, Bridges CB. The annual impact of seasonal influenza in the US: measuring disease burden and costs. Vaccine. 2007;25(27):5086–96.
7. Peters EM, Bateman ED. Ultramarathon running and upper respiratory tract infections. An epidemiological survey. S Afr Med J. 1983;64(15):582–4.
8. Moreira A, Delgado L, Moreira P, Haahtela T. Does exercise increase the risk of upper respiratory tract infections? Br Med Bull. 2009;90:111–31.
9. Nieman DC. Exercise, infection, and immunity. Int J Sports Med. 1994;15 Suppl 3:S131–41.
10. Walsh NP, Gleeson M, Pyne DB, Nieman DC, Dhabhar FS, Shephard RJ, Oliver SJ, Bermon S, Kajeniene A. Position statement. Part two: Maintaining immune health. Exerc Immunol Rev. 2011;17:64–103.
11. Gleeson M. Immune function in sport and exercise, vol. 1. China: Elsevier; 2006.
12. Lowder T, Padgett DA, Woods JA. Moderate exercise protects mice from death due to influenza virus. Brain Behav Immun. 2005;19(5):377–80.
13. Matthews CE, Ockene IS, Freedson PS, Rosal MC, Merriam PA, Hebert JR. Moderate to vigorous physical activity and risk of upper-respiratory tract infection. Med Sci Sports Exerc. 2002;34(8):1242–8.
14. Nieman DC, Henson DA, Austin MD, Brown VA. Immune response to a 30-minute walk. Med Sci Sports Exerc. 2005;37(1):57–62.
15. Pedersen BK, Hoffman-Goetz L. Exercise and the immune system: regulation, integration, and adaptation. Physiol Rev. 2000;80(3):1055–81.
16. Davis JM, Murphy EA, Brown AS, Carmichael MD, Ghaffar A, Mayer EP. Effects of oat beta-glucan on innate immunity and infection after exercise stress. Med Sci Sports Exerc. 2004;36(8):1321–7.
17. Kohut ML, Boehm GW, Moynihan JA. Moderate exercise is associated with enhanced antigen-specific cytokine, but not IgM antibody production in aged mice. Mech Ageing Dev. 2001;122(11):1135–50.
18. Woods JA, Davis JM, Kohut ML, Ghaffar A, Mayer EP, Pate RR. Effects of exercise on the immune response to cancer. Med Sci Sports Exerc. 1994;26(9):1109–15.
19. Mooren FC, Bloming D, Lechtermann A, Lerch MM, Volker K. Lymphocyte apoptosis after exhaustive and moderate exercise. J Appl Physiol. 2002;93(1):147–53.
20. Ortega E, Collazos ME, Maynar M, Barriga C, De la Fuente M. Stimulation of the phagocytic function of neutrophils in sedentary men after acute moderate exercise. Eur J Appl Physiol Occup Physiol. 1993;66(1):60–4.
21. Brown AS, Davis JM, Murphy EA, Carmichael MD, Carson JA, Ghaffar A, Mayer EP. Susceptibility to

HSV-1 infection and exercise stress in female mice: role of estrogen. J Appl Physiol. 2007;103(5):1592–7.
22. Murphy EA, Davis JM, Brown AS, Carmichael MD, Van Rooijen N, Ghaffar A, Mayer EP. Role of lung macrophages on susceptibility to respiratory infection following short-term moderate exercise training. Am J Physiol Regul Integr Comp Physiol. 2004;287(6):R1354–8.
23. Nieman DC. Is infection risk linked to exercise workload? Med Sci Sports Exerc. 2000;32(7 Suppl):S406–11.
24. Murphy EA, Davis JM, Carmichael MD, Gangemi JD, Ghaffar A, Mayer EP. Exercise stress increases susceptibility to influenza infection. Brain Behav Immun. 2008;22(8):1152–5.
25. Weidner TG, Cranston T, Schurr T, Kaminsky LA. The effect of exercise training on the severity and duration of a viral upper respiratory illness. Med Sci Sports Exerc. 1998;30(11):1578–83.
26. Nieman DC, Johanssen LM, Lee JW, Arabatzis K. Infectious episodes in runners before and after the Los Angeles Marathon. J Sports Med Phys Fitness. 1990;30(3):316–28.
27. Ekblom B, Ekblom Ö, Malm C. Infectious episodes before and after a marathon race. Scand J Med Sci Sports. 2006;16(4):287–93.
28. Heath GW, Ford ES, Craven TE, Macera CA, Jackson KL, Pate RR. Exercise and the incidence of upper respiratory tract infections. Med Sci Sports Exerc. 1991;23(2):152–7.
29. Nieman DC, Nehlsen-Cannarella SL, Fagoaga OR, Henson DA, Shannon M, Hjertman JM, Schmitt RL, Bolton MR, Austin MD, Schilling BK, et al. Immune function in female elite rowers and non-athletes. Br J Sports Med. 2000;34(3):181–7.
30. Pyne DB, Baker MS, Fricker PA, McDonald WA, Telford RD, Weidemann MJ. Effects of an intensive 12-wk training program by elite swimmers on neutrophil oxidative activity. Med Sci Sports Exerc. 1995;27(4):536–42.
31. Nieman DC. Immune response to heavy exertion. J Appl Physiol. 1997;82(5):1385–94.
32. Gleeson M. Immune function in sport and exercise. J Appl Physiol. 2007;103(2):693–9.
33. Gabriel H, Urhausen A, Kindermann W. Mobilization of circulating leucocyte and lymphocyte subpopulations during and after short, anaerobic exercise. Eur J Appl Physiol Occup Physiol. 1992;65(2):164–70.
34. Peake JM. Exercise-induced alterations in neutrophil degranulation and respiratory burst activity: possible mechanisms of action. Exerc Immunol Rev. 2002;8:49–100.
35. Blannin AK, Chatwin LJ, Cave R, Gleeson M. Effects of submaximal cycling and long-term endurance training on neutrophil phagocytic activity in middle aged men. Br J Sports Med. 1996;30(2):125–9.
36. Lancaster GI, Khan Q, Drysdale P, Wallace F, Jeukendrup AE, Drayson MT, Gleeson M. The physiological regulation of toll-like receptor expression and function in humans. J Physiol. 2005;563(Pt 3):945–55.
37. Timmons BW, Cieslak T. Human natural killer cell subsets and acute exercise: a brief review. Exerc Immunol Rev. 2008;14:8–23.
38. Shephard RJ, Rhind S, Shek PN. The impact of exercise on the immune system: NK cells, interleukins 1 and 2, and related responses. Exerc Sport Sci Rev. 1995;23:215–41.
39. Shephard RJ, Shek PN. Effects of exercise and training on natural killer cell counts and cytolytic activity: a meta-analysis. Sports Med. 1999;28(3):177–95.
40. Pedersen BK, Ullum H. NK cell response to physical activity: possible mechanisms of action. Med Sci Sports Exerc. 1994;26(2):140–6.
41. Nieman DC, Cook VD, Henson DA, Suttles J, Rejeski WJ, Ribisl PM, Fagoaga OR, Nehlsen-Cannarella SL. Moderate exercise training and natural killer cell cytotoxic activity in breast cancer patients. Int J Sports Med. 1995;16(5):334–7.
42. Na YM, Kim MY, Kim YK, Ha YR, Yoon DS. Exercise therapy effect on natural killer cell cytotoxic activity in stomach cancer patients after curative surgery. Arch Phys Med Rehabil. 2000;81(6):777–9.
43. Peters C, Lotzerich H, Niemeier B, Schule K, Uhlenbruck G. Influence of a moderate exercise training on natural killer cytotoxicity and personality traits in cancer patients. Anticancer Res. 1994;14(3A):1033–6.
44. Nieman DC, Nehlsen-Cannarella SL, Markoff PA, Balk-Lamberton AJ, Yang H, Chritton DB, Lee JW, Arabatzis K. The effects of moderate exercise training on natural killer cells and acute upper respiratory tract infections. Int J Sports Med. 1990;11(6):467–73.
45. Bishop NC, Gleeson M. Acute and chronic effects of exercise on markers of mucosal immunity. Front Biosci. 2009;14:4444–56.
46. Gleeson M, Pyne DB, Callister R. The missing links in exercise effects on mucosal immunity. Exerc Immunol Rev. 2004;10:107–28.
47. McDowell SL, Chaloa K, Housh TJ, Tharp GD, Johnson GO. The effect of exercise intensity and duration on salivary immunoglobulin A. Eur J Appl Physiol Occup Physiol. 1991;63(2):108–11.
48. Klentrou P, Cieslak T, MacNeil M, Vintinner A, Plyley M. Effect of moderate exercise on salivary immunoglobulin A and infection risk in humans. Eur J Appl Physiol. 2002;87(2):153–8.
49. Akimoto T, Kumai Y, Akama T, Hayashi E, Murakami H, Soma R, Kuno S, Kono I. Effects of 12 months of exercise training on salivary secretory IgA levels in elderly subjects. Br J Sports Med. 2003;37(1):76–9.
50. Blannin AK, Robson PJ, Walsh NP, Clark AM, Glennon L, Gleeson M. The effect of exercising to exhaustion at different intensities on saliva immunoglobulin A, protein and electrolyte secretion. Int J Sports Med. 1998;19(8):547–52.
51. Tiollier E, Gomez-Merino D, Burnat P, Jouanin JC, Bourrilhon C, Filaire E, Guezennec CY, Chennaoui M. Intense training: mucosal immunity and incidence of respiratory infections. Eur J Appl Physiol. 2005;93(4):421–8.

52. Whitham M, Laing SJ, Dorrington M, Walters R, Dunklin S, Bland D, Bilzon JL, Walsh NP. The influence of an arduous military training program on immune function and upper respiratory tract infection incidence. Mil Med. 2006;171(8):703–9.
53. Shek PN, Sabiston BH, Buguet A, Radomski MW. Strenuous exercise and immunological changes: a multiple-time-point analysis of leukocyte subsets, CD4/CD8 ratio, immunoglobulin production and NK cell response. Int J Sports Med. 1995;16(7):466–74.
54. Peters EM, Goetzsche JM, Grobbelaar B, Noakes TD. Vitamin C supplementation reduces the incidence of postrace symptoms of upper-respiratory-tract infection in ultramarathon runners. Am J Clin Nutr. 1993;57(2):170–4.
55. Moreira A, Kekkonen RA, Delgado L, Fonseca J, Korpela R, Haahtela T. Nutritional modulation of exercise-induced immunodepression in athletes: a systematic review and meta-analysis. Eur J Clin Nutr. 2007;61(4):443–60.
56. Padayatty SJ, Katz A, Wang Y, Eck P, Kwon O, Lee JH, Chen S, Corpe C, Dutta A, Dutta SK, et al. Vitamin C as an antioxidant: evaluation of its role in disease prevention. J Am Coll Nutr. 2003;22(1):18–35.
57. Himmelstein SA, Robergs RA, Koheler KM, Lewis SL, Qualls CR. Vitamin C suplementation and upper respiratory tract infections in marathon runners. Int J Exerc Physiol. 1998. Available on: http://faculty.css.edu/tboone2/asep/jan9.htm.
58. Spitzer JA, Zhang P. Gender differences in neutrophil function and cytokine-induced neutrophil chemoattractant generation in endotoxic rats. Inflammation. 1996;20(5):485–98.
59. Spitzer JA, Zhang P. Protein tyrosine kinase activity and the influence of gender in phagocytosis and tumor necrosis factor secretion in alveolar macrophages and lung-recruited neutrophils. Shock. 1996;6(6):426–33.
60. Bouman A, Schipper M, Heineman MJ, Faas MM. Gender difference in the non-specific and specific immune response in humans. Am J Reprod Immunol. 2004;52(1):19–26.
61. Pedersen BK. IL-6 signalling in exercise and disease. Biochem Soc Trans. 2007;35(Pt 5):1295–7.
62. Timmons BW, Hamadeh MJ, Devries MC, Tarnopolsky MA. Influence of gender, menstrual phase, and oral contraceptive use on immunological changes in response to prolonged cycling. J Appl Physiol. 2005;99(3):979–85.

# Screening Tools for Excessive Exercise in the Active Female

25

Maria Fernandez-del-Valle

**Abstract**

Excessive Exercise etiology and diagnosis has been deeply studied with inconclusive results. The criteria used for diagnosis are based on the Diagnostic and Statistical Manual for Mental Disorders (DSM) to diagnose addictive behaviors or substance abuse (alcohol, drugs, etc.). Moreover, the screening tools proposed by different authors have been subjective, inaccurate and co-assess other psychological and psychiatric disorders. Subjective screening tools (questionnaires, inventories, interviews, etc.) show information related to the psychological factors contributing to this behavior, and the information related to the characteristics of the exercise performed is scarce. Further, no validated cut-points exist to assess excessive exercise in different groups (children, adolescents, adults, elders, athletes, females, males, etc.). Therefore, the objective screening tools (accelerometers, pedometers, etc.) could add meaningful knowledge about the real characteristics of the activity performed by individuals.

**Keywords**

Female • Excessive exercise • Exercise addiction • Objective • Subjective • Quantitative • Qualitative • Screening tool

## 25.1 Learning Objectives

After completing this chapter, you should have an understanding of the following:

- What does excessive exercise, exercise addiction, or habit mean?
- Concepts of Primary Exercise Addiction and Secondary Exercise Addiction.
- Concepts of Positive Addiction and Negative Addiction.
- When is exercise considered excessive? Excessive Exercise Thresholds.
- Detecting unhealthy Physical Activity Levels: What are the recommendations?
- Qualitative and quantitative screening tools. Are they diagnosing? Are they reporting exercise characteristics?
- Excessive Exercise Comorbidity.

M. Fernandez-del-Valle, Ph.D. (✉)
Department of Health, Exercise, and Sport Sciences, Texas Tech University, Exercise and Sport Sciences Building, 3204 Main, Lubbock, TX 49423, USA
e-mail: maria.fernandez-del-valle@ttu.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_25, 

- Physical Activity cut-points for Excessive Exercise Syndrome (EES).

## 25.2 Introduction

Physical Activity and Physical Exercise are considered by scientists as a determinant tool to prevent chronic physiological and psychological pathologies [1–5]. Performing and maintaining adequate physical activity levels have positive effects on physical and mental health [6]; therefore knowing the cut-points of excessive or insufficient exercise will allow the community to understand the dose–response relationship between exercise and health.

While sedentary life and its effects have been deeply researched, there are few studies that try to quantitatively or qualitatively analyze excessive exercise behaviors. This research conceptualizes it as Excessive Exercise Syndrome (EES).

## 25.3 Research Findings

### 25.3.1 Excessive Exercise Syndrome (EES)

#### 25.3.1.1 Definition

How are "excessive" and "exercise" defined? Excessive describes a quantity that is more than what is desirable, while exercise is defined as "planned, structured, and repetitive bodily movement done to improve or maintain one or more components of physical fitness" [7]. Therefore "excessive exercise" describes a quantity exercise performed beyond the physical healthy limits.

More than 30 terms have been adopted by researchers to describe the phenomenon [8], and the most frequents are "addiction" [9], "dependence" [10], "obligatory" [11], "abuse" [12], "compulsive" [13], "morbid," and "driven" [14]. From these terms, "excessive exercise" does not reflect any etiological implications such as compulsion, addiction, abuse, etc., and therefore is the most used in the absence of consensus [15].

However, the most popular research etiological viewpoints are "addiction" and "compulsion" [10, 16]. Addiction is defined as "the behavioral process that can provide either pleasure or relief from internal discomfort (stress, anxiety, etc.) and it is characterized by repeated failure to control the behavior (state of powerlessness) and maintenance of the behavior in spite of negative consequences" [17]. Therefore, exercise addiction creates both physical and mental distress [18].

On the one hand, "addiction" seems to incorporate concepts describing the principal characteristics of the disorder, since it incorporates both dependence and compulsion [17]. These concepts add psychological aspects related to mental disorders to the excessive exercise syndrome (EES). "Addicts enjoy what they are doing and do not want to stop (ego syntonic)"while "obsessive-compulsive do not enjoy what they are doing but think they ought to do it (ego dystonic)" [15]. From the beginning, research has been focused solely on the population having both EES and mental disorders; however, there is insufficient research relating EES to the general and physical active population (recreational, amateur, or high performance athletes). Furthermore, no studies exist with a clinical sample of exercise addicts greater than 500 individuals, and those subjects demonstrated to suffer from other disorders [19].

All of these definitions imply negative effects, although a large number of positive effects have been described by authors due to high levels of physical activity [20–22]. It is possible that there is a close tie between such concepts (excessive and addictive) and other implicit concepts like habit. Habit is defined as a recurrent, often unconscious, pattern of behavior that is acquired through frequent repetition. Regularly practicing physical activity improves self-esteem, fitness, and social behavior, all of which promote continuous exercise behavior; such a process is known as intrinsic motivation to practice and results in a habit [23]. What happens when you remove a habit from daily life? Researchers have reported that 1–2 weeks of practice deprivation resulted in depression symptoms, negative mood

**Table 25.1** Primary and secondary exercise addiction symptoms

| Primary exercise addiction (PEA) | Secondary exercise addiction (SEA) |
|---|---|
| Preoccupation with exercise routine | Stereotyped pattern of exercise with regular schedule (one set or more a day) |
| Significant withdrawal symptoms if exercise ceases | Salience with and increasing priority over other daily tasks to maintain the routine |
| Significant distress or impairment in all their areas of functioning | Increased tolerance |
| Preoccupation with exercise cannot be explained by co-occurring with other mental disorder | Withdrawal symptoms if exercise ceases |
| | Relief of withdrawal symptoms if exercise is restarted |
| | Subjective awareness of a compulsion to exercise |
| Continues exercise despite of injuries or physical pain | |
| Loss of weight by dieting to improve performance | |

Berczik et al. [17], Bamber [31], Coverley Veale [27]

states, or fatigue in habitual exercises [24–26]. Do these effects mean that they are addicted or performing excessive exercise?

#### 25.3.1.2 Classifications

Coverley Veale et al. proposed a classification for exercise addiction (EA) depending on the causes or the role of the exercise [27]. They differentiated two types of EA: primary and secondary. On the one hand, in primary EA physical activity is an end in itself (exercise is the objective); hence practitioners are intrinsically motivated to exercise. On the other hand, secondary EA co-occurs with an eating disorder or other compulsive disorders, where individuals are extrinsically motivated to exercise according to their self-image (weight loss is the objective) (Table 25.1). Accordingly to this classification, it is important to lighten whether EES is affecting firstly practitioner life or whether it emerges as a derived problem from another psychological disorder [17, 27].

EA criteria in literature are based on the substance abuse criteria from Diagnostic and Statistical Manual for Mental Disorders-IV (DSM-IV) [28] and other research. DSM does not have a specific exercise dependence standard, thus exercise addiction is conceptualized as a maladaptive pattern of exercise, leading to clinically significant impairment or distress. Diagnosis of the EES remains uncertain due to results obtained using different screening tools have not been deeply correlated with symptoms exposed below. It seems that exercise addiction disorder could manifest three or more of the following criteria (Table 25.2) [29, 30].

In order to present the positive and negative issues of an elevated exercise practice, Glasser (1977) introduced the classification of Positive Addiction (PA) and Negative Addiction (NA) [32]. PA contributes to overall practitioner's fitness by integrating exercise into daily activities. Moreover, individuals with PA schedule their sessions around other aspects of their social life and work commitment, increasing feelings of control, competence, and physical and psychological well-being. Thereupon, practice is not detrimental to a proper conduct of their life. Otherwise, NA involves a compulsive need to exercise that annuls the practitioner's physical and mental regards including wellbeing and social life [32].

#### 25.3.1.3 Etiology

Etiological theories are diverse and multifactorial, and based on physiological (endorphins hypothesis and sympathetic arousal hypothesis), psychological (general theory of addiction), or psychobiological (personality traits, or the anorexia analogue hypothesis) issues.

##### Physiological Hypothesis

During 1980s and 1990s some authors had reported about the intense exercise effect in the endogenous opioid system, resulting in significant higher concentrations in blow stream and spinal fluids: the Endorphins Hypothesis.

**Table 25.2** Exercise addiction criteria adapted from DMS-IV

| Concept | Description |
|---|---|
| Tolerance | "Need for increased amounts of exercise to achieve desired effect; diminished effect with continued use of same amount of exercise" |
| Withdrawal | "Characteristic withdrawal symptoms for exercise (e.g., anxiety, fatigue) or exercise is taken to relieve or avoid symptoms" |
| Intention effect | "Exercise is often taken in larger amounts or over a longer period than was intended" |
| Lack of control | "A persistent desire or unsuccessful effort to cut down or control exercise" |
| Time | "A great deal of time is spent in activities necessary to obtain exercise (e.g., physical activity vacations)" |
| Reduction in other activities | "Social, occupational, or recreational activities are given up or reduced because of exercise" |
| Continuance | "Exercise is continued despite knowledge of having a persisting/recurring physical or psychological problem that is likely to have been caused or exacerbated by the exercise (e.g., continued running despite injury)" |

β-endorphin and catecholamine form part of the brain reward system, and it was thought to be related with exercise addiction due to their capacity to regulate physiological responses to stress and intense exercise [18, 33].

Endorphins are endogenous opioids derived from pro-opiocortin polypeptides. Moreover, endorphins are originated in the hypothalamus, and regulate pain perception increasing pain threshold, and showing a greater effort perception in trained people. Exercise intensity (performed above 60 % of the maximal oxygen uptake) and duration (sustained for at least 3 min) are related to increases plasma β-endorphin concentrations. However, plasma endorphins cannot cross the blood–brain barrier (BBB), whereby there is no evidence that changes in plasma levels could lead to simultaneous brain changes. Notwithstanding, some authors believe that endogenous opiates in plasma also operate in the central nervous system activity [17, 34]. In spite of the lack of sufficient direct evidence of association between exercise addiction and endogenous opioid system, and knowing that aerobic exercise stimulates the release of β-endorphin [33], an animal study with rats reported opioid tolerance and dependence in chronic exercisers [35]. Steinberg et al. established that chronic exercise practice [36]:

– Provides an enjoyable effect that stimulates continuing practice
– Triggers an excessive and compulsive behavior
– Results in a reduced pain sensation dependent on the practitioners
– Causes the emergence of a psychological and physiological withdrawal syndrome

Sympathetic Arousal Hypothesis was first proposed by Thompson et al. in 1987. Increased concentrations of catecholamine (adrenalin, noradrenalin, and dopamine) are induced by intense physiological or psychological stress (exercise or tasks). In addition, researchers have reported 1.5–20 times greater concentrations of catecholamine depending on exercise type, duration and intensity [37]. Catecholamine produces increases in heart rate, blood pressure, and a general reaction of the sympathetic nervous system known as "fight-or-flight response" (first stage of a general adaptation syndrome that regulates stress responses) [33, 38]. However, endorphins seem to attenuate their concentrations affecting the sympathetic nervous system regulation. On the one hand, habitual practitioners show a central effect of exercise that reduces the sensitivity to stress, producing lower concentrations of catecholamine and an increased efficiency of energy utilization [38]. On the other hand, research also has shown that greater physical fitness resulting in attenuated concentrations of these hormones could promote negative feelings such as lethargy, fatigue, depression, and decreased arousal [18, 33]. These findings suggest a possible association

between addiction and catecholamine behaviors, due to the fact that habitual exercisers are motivated to engage in increased levels of exercise in order to achieve the same arousal levels and suppress symptoms [33, 39].

#### Psychological Hypothesis

Szabo et al. proposed a general theory of addiction or cognitive appraisal hypothesis to explain the etiology of exercise addiction. This theory means that habitual exercisers use exercise as a way to cope with stress, learning to need exercise for this purpose (coping mechanism). When the amounts are exaggerated, exerciser explains and justifies the practice, and slowly takes a principal role instead of normal daily activities. Negative psychological feelings (irritability, guilt, anxiousness, etc.) appear when the person is required to reduce or stop exercising, feelings that are believed to represent the withdrawal symptoms. There is also a loss of the coping mechanism where exerciser loses control over stressful situations increasing vulnerability to stress, and amplifying these negative psychological feelings when deprivation of exercise happens. The addicted exerciser is trapped in a vicious circle, exercising more to cope with daily stress that partly is caused by itself [40].

#### Psychobiological Hypothesis

Personality traits or anorexia analogue hypothesis shows to be the more postulated to explain EA despite the limited research support. Individuals addicted to exercise share common personality traits and behavioral dispositions with anorectic patients such as compulsiveness [41], neuroticism [42], low self-esteem [43], perfectionism [43–45], high trait anxiety [46], high self-expectations, denial of potentially serious debility, and tendency towards depression [33]. These traits and dispositions seem to be more pathological in patients with anorexia nervosa than in addicted exercisers [47].

The main effects of EA in female are: concern about body image and appearance, development of anxiety and depression disorders, as well as the emergence of other behaviors as compulsive buying [48], whereas male have an uncertain identity, low self-esteem, AND anxiety about physical ineffectiveness [49]. Some authors have reported that exercise addiction coexist with eating disorders [50], results supported by animal models demonstrating that running-wheel is induced when rats are food-restricted 1 day. This vicious cycle is reinforced by a reward mechanism [51]. However, no evidence was reported in human runners compared with anorexic patients [47].

### 25.3.1.4 Prevalence

The prevalence of EA is variable and uncertain, owing the lack of research of clinical cases methodologically comparable (heterogeneity of the instruments used to assess EA, the insufficient sample size, and heterogeneity of the population studied). However, Sussman et al. showed a prevalence of 3 % at risk of EA, results that were supported by other authors that reported a 2.5 % [52, 53] and 3.6 % of general exercisers [54]. A greater prevalence (7 %) was found by Szabo et al. among university sport science students. "Maybe those results were induced by the awareness about the benefits of exercise on the well-being" [54].

Lejoyeux et al. analyzed exercise behaviors on 300 practitioners from a fitness room (18 years and older). A total of 125 (42 %) presented risk factors of EA, and from those risk "dependants" spent more hours each day in the fitness center compared with "no-dependants"(2.1 vs. 1.5 h/day), and they went more often each week (3.5 vs. 2.9 days/week). Moreover, exercise addicts smoked less and were significantly more compulsive buyers (63 % vs. 38 %) [48].

### 25.3.1.5 Exercise Addiction in Active Female

Gender incidence remains unclear, although some researchers reported equal prevalence in both males and females, while others have shown a higher prevalence of a primary EA in males, compared with an increased secondary EA in female [53, 55].

Villella et al. reported results in behavioral addictions in adolescents and young adults using the Exercise Addiction Inventory (EAI).

This inventory was validated for university students, not high school students [56, 57]. Participants with scores of 24 or more were identified as at risk for exercise addiction. From a total of 2,853 high school students (1,142 girls—40 %) ranged between 13 and 20 years old, 8.5 % were at risk of EA. Segregating the sample in adolescents and young adults, both groups presented similar percentages (8.7 % and 8.3 % respectively), and females presented lower percentage (6.3 %) compared to males (10.1 %) [58].

EAI was used by Griffiths et al. who identified 3 % of the sample ($n = 200$) of adults between 18 and 40 years old at risk of EA scoring above 24, but no gender differences were reported [57].

Johnston et al. recruited 32 women (16–77 years old) from exercise facilities, weight-loss organizations, and school and university classes [15]. Participants were engaged in a wide variety of activities (hockey, diving, exercise classes, running, weight training, etc.), where the active time spent weekly ranged from 1 to 16 h (mean of 5 h/week). A total of 18.75 % scored above cutoff points of the Obligatory Exercise Questionnaire (OEQ), and half of them were defined as chronic dieters. They also showed that behavioral criteria such as frequency and amount of exercise (quantitative) are as important as psychological factors such as effort and enthusiasm (qualitative).

Exercise addiction in adult runners has been reported, showing that the more they exercise the more addicted they are to exercise with no gender differences. In addition, these results were constantly significant in exercisers of health club [59].

Authors such as Crossman et al. reported no exercise addiction in preadolescents, adolescents, and young adult runners (13–26 years) and swimmers (10–19 years) of different competitive levels (from international to regional) [60]. Results reported that 1–5 days of layoff are perceived by athletes as positive, showing greater positive mood states when competition level is lower, and when female group is analyzed compared to male [60].

Edgar et al. recruited a total of 102 female athletes where 47 were dancers, 39 runners and 16 hockey players. EA is lower in women who participate in collaborative sports (hockey, or soccer), followed by endurance practitioners (marathon or ultra-marathon), with a higher rates in women practicing activities such as ballet or modern dance. Higher addiction in dancers and ballerinas could be due to the expectations of technical, aerobic and anaerobic fitness, intensity, body image, and weight control requirements [61, 62].

There are few studies with a low sample size that differentiate the prevalence of this disorder based on the level of performance (high performance, amateur or physical activity for health), with higher levels of dependence in high performance and professionals (64.3 %) compared to amateur athletes (43.3 %). Moreover, athletes who presented eating disorders associated to exercise addiction where 34 % of 203 recruited (50 % in female, 27 % in male). In addition, eating disorders had a greater presence in amateurs compared to professionals (35.7 % vs. 31.5 %) [55]. According to these results, it seems that the volume of exercise (time, frequency, and intensity) has no validated cut-points for excessive exercisers [55, 60].

One study analyzed the exercise addiction using the Obligatory Exercise Scale (OES) in a group of 183 active female from 18 to 71 years. From all 7.1 % met exercise addiction criteria presenting scores equal or above to 50, and there were no differences between age groups: Group 1: 6.6 % (18–25 years); Group 2: 3.3 % (26–35 years); Group 3: 16.1 % (36–45 years); Group 4: 3.1 % (46–55 years); Group 5: 6.9 % (56–71 years). Furthermore, older showed to be more concerned with their health than younger women. Besides, they reported that from the total of the sample 82 % were concerned with their appearance, 30.6 % with their weight, and 41 % perceived themselves as being overweight [63].

### 25.3.2 Screening Tools for Excessive Exercise Syndrome (EES)

As EES could trigger in serious psychological and physiological consequences, it is necessary to detect excessive practice, and reeducate the practitioner to healthy exercise habits.

Detecting the disorder could be accomplished by different screening tools. Which kind of screening tools could help? There are many tools in the literature that have been developed since 1970s, and therefore the most updated and used in the literature have been explained.

#### 25.3.2.1 Classification of the Screening Tools

Choosing one screening tool over the other could give the researchers different validity levels of data from the most objective to the most subjective measurements. When is a screening tool considered objective or subjective? A screening tool is considered to be highly objective when it measures what it intends to, and when it approaches the fact. Subjective screening tools approximate the data by delayed information where the perception of researchers or participants could alienate results. Using objective or subjective screening tools depend on which characteristics of the excessive exercise are aimed to be analyzed: minutes per day, week or month, intensity of exercise, mood state, or eating disturbances. Researchers are more likely to use objective tools when the assessment does not need from the participation of individuals, or subjective tools when the participation of one or both researcher and individual is needed. Therefore, screening tools like mechanical devices are shown as objective tools, and inventories, questioners, self-report diaries, and interviews are shown as subjective tools [64, 65].

Given that, screening tools could be classified by what they are measuring (quantity or quality of excessive exercise). Therefore, quantitative characteristics assess the minutes, intensity, or time spent exercising, while qualitative characteristics assess the psychological effects such as mood state, anxiety, or eating disturbances. Daily logs, questionnaires, inventories, and observations are the most used subjective instruments due to their easy application and low cost. Nevertheless, they are limited by their validity and reliability depending on the sample size and targeted population (children and elder) [66] that obstruct the validation process. When these screening tools are compared with criteria methods, the results presented become overestimated. What is more, when the variables assessed are physiological they result in a greater overvalue, because they do not analyze all dimensions of physical activity [65].

Regarding objective instruments, pedometers, heart rate devices, and accelerometers are the most common devices used to assess spontaneous activity during prolonged periods of time. Of these, the accelerometer is a practical and precise device which has a friendly cost [67].

Finally, criteria instruments are used as a reference to validate all the instruments mentioned above. Doubly Labeled Water (DLW) is considered as the "Gold Standard," and is used to determine daily life energy uptake, nevertheless it is not often used due to its elevated cost [68–70].

Both subjective and objective screening tools can be classified as qualitative [Obligatory Exercise Questionnaire (OEQ), Exercise Dependence Scale Revised (EDS-R) or Exercise Addiction Inventory (EAI)], and quantitative [International Physical Activity Questionnaire (IPAQ), and mechanical devices as accelerometers or pedometers]; all of which are subjective except mechanical devices.

#### 25.3.2.2 Qualitative Screening Tools

Qualitative screening tools report information about the characteristics of the exercise regarding to psychological and physiological issues. The main characteristics of these instruments (QEQ, EDS-R, and EAI), as well as principal sources, can be found at http://www.knowmo.ca.

##### Obligatory Exercise Questionnaire (OEQ)

The OEQ [11] was the first scale to measure obligatory exercise. It was modified from the Obligatory Running Questionnaire [47], and its psychometric properties have been well established [71].

This is a self-report questionnaire consisted on 20 items with a 4 point Likert scale scored at the extremes with never (1) to always (4); scores can add up to a total of 20 [72], and it can be used to identify psychological characteristics of committed adult and adolescent athletes, covering

a wide range of exercise behavior such as running and weight lifting.

The OEQ also looks at the relationship between exercise behavior, eating disturbance, and body image in obligatory exercisers. Therefore, is often used to detect anorexia athletica or eating disorder induced by exercise abuse. Besides, OEQ has three subscales: exercise fixation (items associated with missed exercise and exercise to compensate for perceived overeating), exercise frequency (addressing frequency and type of exercise), and exercise commitment (indicating a sense of routine which cannot be missed) [73]. The primary weaknesses of this excessive exercise screening tool consist of different versions that have been developed, and the lack of validated cut-points.

#### Exercise Dependence Scale Revised (EDS-R)

The EDS was created by Downs et al. [74], and revised from the Hausenblas et al. Exercise Dependence Scale (EDS) [10]. Criterion was based upon DSM-IV [28]. The EDS-R provides the following information: mean score of exercise dependence symptoms, differences between at-risk for exercise dependence, nondependent-symptomatic, and nondependent-asymptomatic, and specifies if there is evidence of physiological dependence or no physiological dependence [75].

The EDS-R can be administered in adults 18 years and older, indicating responses to 21-items (28 items initially) of a Likert scale scored at the extremes with never (1) and always (6). EDS-R can provide information about the mean of each one of the symptoms or of the mean total score, allowing to differentiate individuals in three groups: at risk of exercise dependence (scores of 5–6 on the Likert scale in at least three of the seven criteria), nondependent symptomatic (scores of 3–4 on the Likert scale in at least three criteria, or scores of 5–6 combined with scores of 3–4 in three criteria, but without meeting the at-risk conditions), and nondependent asymptomatic (scores of 1–2 on the Likert scale in at least three criteria, without meeting the conditions of the nondependent symptomatic). The primary strength is the large sample size (college students), whereas weaknesses are the lack of validated cut-points, and the instrument length.

#### Exercise Addiction Inventory (EAI)

The EAI was developed by Terry et al. in 2004 [56]. This inventory was developed as a self-report to examine beliefs towards exercise in habitual adult exercisers. The inventory is made up of six statements: the importance of exercise for the individual, personal conflicts due to exercise, how mood changes with exercise, the amount of time spent exercising, the outcome of a missing workout, and the effects of decreasing physical activity. Individuals are asked to rate each statement from 1 (strongly disagree) to 5 (strongly agree). If an individual scores 24 or greater they are at-risk for exercise addiction, from 13 to 23 they are symptomatic individuals, and from 0 to 12 they are asymptomatic individuals. While this is an instrument quick and easy to use, validity data on cut-points are limited.

### 25.3.2.3 Quantitative Screening Tools

#### International Physical Activity Questionnaire (IPAQ)

The purpose of the International Physical Activity Questionnaires (IPAQ) is to provide a set of well-developed instruments that can be used internationally to obtain comparable estimates of physical activity. IPAQ was created as a tool to universalize research worldwide in different human races, age, and gender to enable the comparison of data [76]. This questionnaire was the first international rule for subjective assess of physical activity, and it has two versions (short and long). The short version is suitable for use in national and regional prevention and the long version provides more detailed information [77, 78]. This instrument brings detailed information about the amount of physical activity (recreational, work, sports, or transportation activity), and has great weaknesses such as its length taking more than 30 min in the long version [79].

The scoring protocol presents three categories of physical activity: Low, Moderate, and

High. First, those individuals who do not meet criteria of 2 or 3 days of practice are considered low physical active or inactive. Second, is the moderate physical activity level where individuals participate in 3 or more days of vigorous activity of at least 20 min, 5 or more days of moderate activity or walking at least 30 min, or 5 or more days of any combination of walking, moderate or vigorous activity achieving a minimum of 600 MET-min/week. Finally, high physical activity individuals show vigorous activity on at least 3 days accumulating 1,500 MET-min/week, or 7 or more days of any combination of walking, moderate or vigorous activities achieving a minimum of 3,000 MET-min/week. Cut-points for excessive exercise were not studied. MET values for each domain and intensity are reported in http://www.ipaq.ki.se/ for free use as well as questionnaire in different languages.

**Accelerometry**

Provides information about objective quantification of physical activity, researchers have been using multiple movement devices: accelerometers, pedometers, etc. Among them accelerometers are most popular, and Actigraph has been the most used to quantify free-living physical activity levels and patterns (Fig. 25.1). Moreover, Actigraph accelerometer has been used not only in healthy children, adolescents, adults and elders both genders, but also in different pathologies owing to its low cost, storage capacity (up to 22 days), programming, data download, validity, and reliability [68, 70].

Actigraph has to be placed at waist using a belt (Fig. 25.2). Participants have to where it for a period of 7 days, at least 10 h/day to consider it a valid measure. This device needs to be programmed and downloaded using a software.

The data obtained is as follows: sedentary activity (min/day), and active levels (min/day) (light, moderate, vigorous, and very vigorous levels). These levels have been correlated with metabolic equivalent of task (MET), where sedentary is 1–2 METs, light level is <3 METs, moderate level between 3 and 5.9 METs, vigorous level between 6 and 9 METs, and very vigorous level >9 METs [80, 81].

**Fig. 25.1** Actigraph

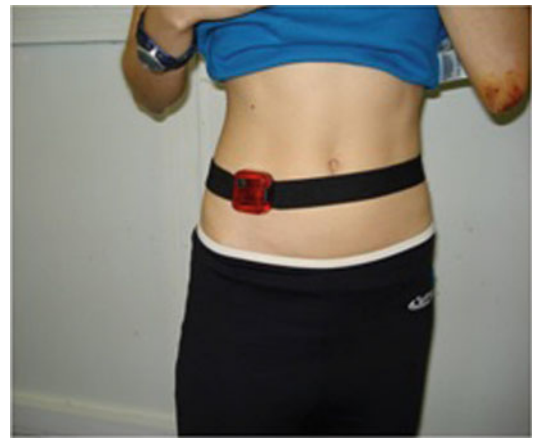


**Fig. 25.2** Actigraph placement

Depending on the age, physical activity intensities are determined using different cut-points, because the physical activity patterns change from childhood to elder. The cut-points refer to the daily minutes that individual spent depending on the intensity, and knowing the age [82–84].

#### 25.3.2.4 Choosing an Appropriate Screening Tool

Considerations for choosing an appropriate screening tool:

- Application: easy to apply and interpret.
- Validity: capable to measure what it was created for, and reproducible.
- Reliability: inter- and intra-subject.
- Cost–benefits: sample size could limit to choose one or other.

– Objectivity: the most objective instrument has to be used.
– Reactivity: participants tend to change behaviors.

Choosing an appropriate screening tool has to be made taking in account strengths and weaknesses of the instruments (Table 25.3) [85].

### 25.3.3 Cut-Points to Detect Unhealthy Exercise Levels

Since "health" is considered a state of complete physical, mental, and social well-being and not merely the absence of disease, physical activity levels could determine the healthy status of the population [86]. Quantification of free-living physical activity could be used to detect and establish the cut-points of exercise behaviors in different healthy and clinical population regarding to absence or excess of active lifestyle. However, while sedentary lifestyle has been deeply researched using accelerometers, excess of physical activity has been poorly studied. Only a few studies related with eating disorders has been developed; all of them have shown no abuse of exercise at the moment of assessment compared to healthy lifestyle recommendations and eating disorders cut-points for excessive exercise [87–89].

The Center for Disease Control and Prevention and American College of Sport Medicine reported the first physical activity recommendations, 30 or more minutes of moderate–vigorous physical activity in almost every day [7, 90]. Currently this recommendation has been increased, especially in children, where at least 60 min of moderate to vigorous physical activity (MVPA) should be performed 5 days a week and ideally every day (see Chaps. 21 and 22).

Related to the cut-points some authors established that more than 6 h of physical activity per week during at least four consecutive weeks indicate excessive exercise (EE) practice [91]. This cut-point was proposed by Davis et al. regarding to eating disorder use of physical activity as a compensatory behavior, therefore it is related with secondary exercise addiction [90]. Later, the intensity of this EE (first postulated by Davis et al.), was defined by Bratland-Sanda et al. in 2010 to be moderate to vigorous intensity. Therefore, those who perform more than 52 min/day of MVPA will be considered as SEA. Nevertheless, this cut-point has to be taken carefully, since it has been established for a mental disorder [92].

Comparing Davis et al. recommendations (1997) with ACSM recommendations [93] is contradictory because healthy population is expected to practice at least 60 min every day, while those who practice more than 52 min every day of MVPA are considered addicted to exercise. There is a lack of studies comparing both healthy and disordered populations that could put as in acquaintance of how much activity is performed by addicted individuals. Such lack of knowledge is extended to other population groups such as amateur or high performance athletes which lack reference values that could clear how much activity is too much for such groups. In relation to the level of practice, physical activity thresholds seem to change, therefore cut-points for EES are different depending on the level of practice and expertise.

An article of 2001 reported a direct relationship between greater increases in physical health parameters and the number of weekly hours spent exercising (from 2 to 7 weekly hours). Lower cardiovascular risks of mortality were reported in those who practiced between 4 and 7 weekly hours of regular physical activity. However, some cardiovascular risks were reported when more than 7 weekly hours where performed. By contrast, lower risk of cancer, respiratory disease, or other diseases where addressed when the practice was more than 7 h/week of MVPA [20]. Other authors show that physical activity benefits depend on the intensity of the practice, where greater benefits and lower mortality rates where associated to vigorous activity, nor light activity practice [94].

## 25.4 Contemporary Understanding of the Issue

Up to date, the research about the excess of exercise as a syndrome has been insufficient to fully understand, detect, diagnose, and manage exercise abuse-related problems. During the last 20 years different authors have shown that each group

**Table 25.3** Screening tools population applicability, strengths, and weaknesses

| Instrument | Population | Strengths | Weaknesses |
|---|---|---|---|
| Obligatory exercise questionnaire (OEQ) | Adolescents<br>Adults | Cheap applicability (public domain)<br>Exercise fixation, frequency and commitment | Lack of validated cut-points<br>Different versions developed<br>Instrument length<br>Subjective (self-report) |
| Exercise dependence scale revised (EDS-R) | Adults | Cheap applicability (public domain)<br>DMS-IV addiction criteria (withdrawal, continuance, lack of control, reductions in other activities, time, intention) | Lack of validated cut-points<br>Instrument length<br>Subjective (self-report) |
| Exercise addiction inventory (EAI) | Adults (university students) | Cheap (public domain), and quick applicability<br>General components of addiction (salience, mood modification, tolerance, withdrawal, conflict, relapse) | Lack of validated cut-points<br>Subjective (self-report) |
| International physical activity questionnaire (IPAQ) | 15–69 years | Cheap applicability (public domain)<br>Worldwide instrument<br>Self or telephone report | Length in long form<br>Details in short form<br>Subjective (self-report) |

behave different, and in many cases the EES is accompanied or caused by a mental disease. There is much more work to be done, because it is necessary to detect when the practice of exercise is an acquired habit, or when is turning into a pathology depending on the group that is analyzed.

## 25.5 Future Directions

### 25.5.1 Reference Values for Female and Clinical Population

There is a lack of research on reference values for excessive exercise in healthy and clinical population in all ages. Validation, association, and correlation studies are needed to establish cut-points and reference values using different instruments and technics: physical activity levels using accelerometers or pedometers, plasma endogenous opioids level correlations with excessive exercise levels (endorphin, or catecholamine), psychological personality traits, etc.

More research has to be conducted to find out if exercise addiction is a consequence of other mental disorders (obsessive-compulsive, compulsive, addictive disorders, eating disorders, disordered eating, etc.)

### 25.5.2 Female Athletes and EES

Future research is needed to obtain reference values for excessive physical activity levels in high performance due to training requirements in different sports and disciplines which requirements are different from general population, distinguishing professional, and amateur, or international, national, or regional competition level.

## 25.6 Concluding Remarks

There is a fine line between regular and healthy exercise and excessive practice. There is not a clear relationship between high levels of exercise and other mental disorders. Although other mental illnesses may be the source, there are no studies to confirm or deny it.

Excessive exercise is greatly present in high performance compared to recreational; this may be due to the habitual practice, frequency and quantity of exercise performed. When 15 years of experience are exceeded, the rate of excessive exercisers decreases, likely because practice is highly integrated in daily life. Moreover, when excessive exercisers are females the features are different compared to males, such as weight preoccupation, appearance, body image and body composition commitment. These features are closely related with eating disturbances and eating disorder symptoms.

Screening tools need to be more specific and detailed, and validated as well. No cut-points for excessive exercise were established in any of the instruments purposed. However, there are some recommendations reported for eating disorders, weekly physical activity practice, and mortality research. Additionally, a deep analysis (using one or more screening tools) of quantitative and qualitative is needed to set the most appropriate assessment.

## References

1. Ness AR, Leary SD, Mattocks C, et al. Objectively measured physical activity and fat mass in a large cohort of children. PLoS Med. 2007;4(3):e97.
2. Ekelund U, Brage S, Froberg K, et al. TV viewing and physical activity are independently associated with metabolic risk in children: the European Youth Heart Study. PLoS Med. 2006;3(12):e488.
3. Landers DM. The influence of exercise on mental health. Washintong, DC: President's Council on Physical Fitness and Sports; 1997.
4. Ströhle A. Physical activity, exercise, depression and anxiety disorders. J Neural Transm. 2009;116(6):777–84.
5. Oeland AM, Laessoe U, Olesen AV, Munk-Jørgensen P. Impact of exercise on patients with depression and anxiety. Nord J Psychiatry. 2010;64(3):210–7.
6. WHO. The prevention of mental disorders: effective interventions and policy options. Geneva: World Health Organization; 2004.
7. Pate RR, Pratt M, Blair SN, et al. Physical activity and public health. JAMA. 1995;273(5):402.
8. Allegre B, Souville M, Therme P, Griffiths M. Definitions and measures of exercise dependence. Addict Res Theory. 2006;14(6):631–46.

9. Goodman A. Addiction: definition and implications. Br J Addict. 1990;85(11):1403–8.
10. Hausenblas HA, Symons DD. Exercise dependence: a systematic review. Psychol Sport Exerc. 2002;3(2): 89–123.
11. Pasman L, Thompson JK. Body image and eating disturbance in obligatory runners, obligatory weightlifters, and sedentary individuals. Int J Eat Disord. 1988;7(6):759–69.
12. Davis C. Exercise abuse. Int J Sport Psychol. 2000;31(2):278–89.
13. Dalle Grave R, Calugi S, Marchesini G. Compulsive exercise to control shape or weight in eating disorders: prevalence, associated features, and treatment outcome. Compr Psychiatry. 2008;49(4):346–52.
14. Fairburn CG. Cognitive behavior therapy and eating disorders. New York: The Guilford Press; 2008.
15. Johnston O, Reilly J, Kremer J. Excessive exercise: from quantitative categorisation to a qualitative continuum approach. Eur Eat Disord Rev. 2011;19(3):237–48.
16. Adkins EC, Keel PK. Does "excessive" or "compulsive" best describe exercise as a symptom of bulimia nervosa? Int J Eat Disord. 2005;38(1):24–9.
17. Berczik K, Szabó A, Griffiths MD, et al. Exercise addiction: symptoms, diagnosis, epidemiology, and etiology. Subst Use Misuse. 2012;47:403–17.
18. Cockerill IM, Riddington ME. Exercise dependence and associated disorders: a review. Couns Psychol Q. 1996;9(2):119–29.
19. Sussman S, Lisha N, Griffiths M. Prevalence of the addictions: a problem of the majority or the minority? Eval Health Prof. 2011;34(1):3–56.
20. Rockhill B, Willett WC, Manson JE, et al. Physical activity and mortality: a prospective study among women. Am J Public Health. 2001;91(4):578.
21. Myers J, Prakash M, Froelicher V, Do D, Partington S, Atwood JE. Exercise capacity and mortality among men referred for exercise testing. N Engl J Med. 2002;346(11):793–801.
22. Heyward C, editor. Evaluacion de la aptitud física y Prescripción del ejercicio. 5th ed. Editorial Médica Panamericana; 2008
23. Adams J, Kirkby RJ. Excessive exercise as an addiction: a review. Addict Res Theory. 2002;10(5) :415–37.
24. Gauvin L, Szabo A. Application of the experience sampling method to the study of the effects of exercise withdrawal on well-being. J Sport Exerc Psychol. 1992;14:361–74.
25. Berlin AA, Kop WJ, Deuster PA. Depressive mood symptoms and fatigue after exercise withdrawal: the potential role of decreased fitness. Psychosom Med. 2006;68(2):224–30.
26. Kop WJ, Weinstein AA, Deuster PA, Whittaker KS, Tracy RP. Inflammatory markers and negative mood symptoms following exercise withdrawal. Brain Behav Immun. 2008;22(8):1190–6.
27. Coverley Veale DMW. Exercise dependence. Br J Addict. 1987;82(7):735–40.
28. APA. Diagnostic and statistical manual of mental disorders: DSM-IV-TR. Arlington, VA: American Psychiatric Publishing Inc.; 2000.
29. Freimuth M, Moniz S, Kim SR. Clarifying exercise addiction: differential diagnosis, co-occurring disorders, and phases of addiction. Int J Environ Res Public Health. 2011;8(10):4069–81.
30. Vina J, Sanchis-Gomar F, Martinez-Bello V, Gomez-Cabrera M. Exercise acts as a drug. Pharmacological benefits of exercise. Br J Pharmacol. 2012;167(1):1–12.
31. Bamber D, Cockerill I, Rodgers S, Carroll D. Diagnostic criteria for exercise dependence in women. Br J Sports Med. 2003;37(5):393–400.
32. Glasser W. Promoting client strength through positive addiction. Can Counsellor. 1977;11(4):173–5.
33. Hamer M, Karageorghis CI. Psychobiological mechanisms of exercise dependence. Sports Med. 2007;37(6):477–84.
34. Biddle S, Mutrie N. Psychology of physical activity and exercise. London: Springer; 1991.
35. Smith MA, Yancey DL. Sensitivity to the effects of opioids in rats with free access to exercise wheels: μ-opioid tolerance and physical dependence. Psychopharmacology (Berl). 2003;168(4):426–34.
36. Steinberg H, Sykes EA, LeBoutillier N. Exercise addiction: indirect measures of "endorphins"? In: Annett J, Cripps B, Steinberg H, editors. Exercise addiction: motivation for participation in sport and exercise. Leicester: The British Psychological Society; 1995. p. 6–14.
37. Zouhal H, Jacob C, Delamarche P, Gratas-Delamarche A. Catecholamines and the effects of exercise, training and gender. Sports Med. 2008;38(5):401–23.
38. Tsatsoulis A, Fountoulakis S. The protective role of exercise on stress system dysregulation and comorbidities. Ann N Y Acad Sci. 2006;1083(1):196–213.
39. Glass JM, Lyden AK, Petzke F, et al. The effect of brief exercise cessation on pain, fatigue, and mood symptom development in healthy, fit individuals. J Psychosom Res. 2004;57(4):391–8.
40. Szabo P, Tury F. [Prevalence of clinical and subclinical forms of anorexia and bulimia nervosa among working females and males]. Orv Hetil. 1995; 136(34):1829–35.
41. Thome JL, Espelage DL. Obligatory exercise and eating pathology in college females: replication and development of a structural model. Eat Behav. 2007;8(3):334–49.
42. Carron AV, Hausenblas HA, Estabrooks PA. The psychology of physical activity. New York: McGraw Hill; 2003.
43. Hall HK, Hill AP, Appleton PR, Kozub SA. The mediating influence of unconditional self-acceptance and labile self-esteem on the relationship between multidimensional perfectionism and exercise dependence. Psychol Sport Exerc. 2009;10(1):35–44.
44. Hausenblas HA, Downs DS. How much is too much? The development and validation of the exercise dependence scale. Psychol Heal. 2002;17(4):387–404.

45. Hall HK, Kerr AW, Kozub SA, Finnie SB. Motivational antecedents of obligatory exercise: the influence of achievement goals and multidimensional perfectionism. Psychol Sport Exerc. 2007;8(3):297–316.
46. Spano L. The relationship between exercise and anxiety, obsessive-compulsiveness, and narcissism. Personal Individ Differ. 2001;30(1):87–93.
47. Blumenthal JA, O'Toole LC, Chang JL. Is running an analogue of anorexia nervosa? JAMA. 1984;252(4): 520–3.
48. Lejoyeux M, Avril M, Richoux C, Embouazza H, Nivoli F. Prevalence of exercise dependence and other behavioral addictions among clients of a Parisian fitness room. Compr Psychiatry. 2008;49(4):353–8.
49. Yates A, Leehey K, Shisslak CM. Running—an analogue of anorexia? N Engl J Med. 1983;308(5):251–5.
50. Davis C, Kennedy SH, Ralevski E, et al. Obsessive compulsiveness and physical activity in anorexia nervosa and high-level exercising. J Psychosom Res. 1995;39(8):967–76.
51. Duclos M, Bouchet M, Vettier A, Richard D. Genetic differences in hypothalamic-pituitary-adrenal axis activity and food restriction-induced hyperactivity in three inbred strains of rats. J Neuroendocrinol. 2005;17(11):740–52.
52. Hausenblas HA, Fallon EA. Relationship among body image, exercise behavior, and exercise dependence symptoms. Int J Eat Disord. 2002;32(2): 179–85.
53. Hausenblas HA, Downs DS. Relationship among sex, imagery and exercise dependence symptoms. Psychol Addict Behav. 2002;16(2):169.
54. Szabo A, Griffiths MD. Exercise addiction in British sport science students. Int J Ment Heal Addict. 2007;5(1):25–8.
55. Blaydon MJ, Lindner KJ. Eating disorders and exercise dependence in triathletes. Eat Disord. 2002;10(1):49–60.
56. Terry A, Szabo A, Griffiths M. The exercise addiction inventory: a new brief screening tool. Addict Res Theory. 2004;12(5):489–99.
57. Griffiths M, Szabo A, Terry A. The exercise addiction inventory: a quick and easy screening tool for health practitioners. Br J Sports Med. 2005;39(6):e30.
58. Villella C, Martinotti G, Di Nicola M, et al. Behavioural addictions in adolescents and young adults: results from a prevalence study. J Gambl Stud. 2011;27(2):203–14.
59. David MF, Germone K. Negative addiction in male and female runners and exercisers. Percept Mot Skills. 1993;77(1):192–4.
60. Crossman J, Jamieson J, Henderson L. Responses of competitive athletes to lay-offs in training: exercise addiction or psychological relief? J Sport Behav. 1987;10:28–38.
61. Edgar FP, Daleng ML, Robert WM. Scores on exercise dependence among dancers. Percept Mot Skills. 1993;76(2):531–5.
62. Anshel MH. Sources of disordered eating patterns between ballet dancers and nondancers. J Sport Behav. 2004;27(2):115–33.
63. Freitag-Honsberger S. The relationship between body image and obligatory exercise behavior among physical active women of various ages. Montreal: Physical Education, McGill University; 2001.
64. Ainsworth BE. How do I measure physical activity in my patients? Questionnaires and objective methods. Br J Sports Med. 2009;43(1):6–9.
65. Prince SA, Adamo KB, Hamel ME, Hardt J, Gorber SC, Tremblay M. A comparison of direct versus self-report measures for assessing physical activity in adults: a systematic review. Int J Behav Nutr Phys Act. 2008;5(1):56.
66. Sallis J, Saelens B. Assessment of physical activity by self-report: status, limitations, and future directions. Res Q Exerc Sport. 2000;71(2 Suppl):S1–14.
67. Staudenmayer J, Pober D, Crouter S, Bassett D, Freedson P. An artificial neural network to estimate physical activity energy expenditure and identify physical activity type from an accelerometer. J Appl Physiol. 2009;107(4):1300–7.
68. Abel MG, Hannon JC, Sell K, Lillie T, Conlin G, Anderson D. Validation of the Kenz Lifecorder EX and ActiGraph GT1M accelerometers for walking and running in adults. Appl Physiol Nutr Metab. 2008;33(6):1155–64.
69. Pate RR, Almeida MJ, McIver KL, Pfeiffer KA, Dowda M. Validation and calibration of an accelerometer in preschool children. Obesity. 2006; 14(11):2000–6.
70. Gallardo C. Cuantificacion objetiva de los niveles y patrones de actividad física en poblacion infantil con patologia cronica controlada. Madrid: Escuela de Doctorado e Investigación, Uniersidad Europea de Madrid; 2011.
71. Coen SP, Ogles BM. Psychological characteristics of the obligatory runner: a critical examination of the anorexia analogue hypothesis. J Sport Exerc Psychol. 1993;15(3):338–54.
72. Thompson JK, Blanton P. Energy conservation and exercise dependence: a sympathetic arousal hypothesis. Med Sci Sports Exerc. 1987;19:91–9.
73. Ackard DM, Brehm BJ, Steffen JJ. Exercise and eating disorders in college-aged women: profiling excessive exercisers. Eat Disord. 2002;10(1):31–47.
74. Downs DS, Hausenblas HA, Nigg CR. Factorial validity and psychometric examination of the exercise dependence scale-revised. Meas Phys Educ Exerc Sci. 2004;8(4):183–201.
75. Hausenblas HA, Symons DD. Exercise dependence scale-21 manual. Gainesville: University of Florida; 2002. Unpublished manuscript.
76. Craig CL, Marshall AL, Sjöström M, et al. International physical activity questionnaire: 12-country reliability and validity. Med Sci Sports Exerc. 2003;35(8):5.

77. Mäder U, Martin BW, Schutz Y, Marti B. Validity of four short physical activity questionnaires in middle-aged persons. Med Sci Sports Exerc. 2006;38(7):12.
78. Wolin KY, Heil DP, Askew S, Matthews CE, Bennett GG. Validation of the International physical activity questionnaire-short among blacks. J Phys Act Health. 2008;5(5):746.
79. Booth ML. Assessment of physical activity: an international perspective. Res Q Exerc Sport. 2000;7(2):7.
80. Ainsworth BE, Haskell WL, Whitt MC, et al. Compendium of physical activities: an update of activity codes and MET intensities. Med Sci Sports Exerc. 2000;32(9):S498–516.
81. Ridley K, Ainsworth BE, Olds TS. Development of a compendium of energy expenditures for youth. Int J Behav Nutr Phys Act. 2008;5(1):45.
82. Ekelund U, Aman J, Yngve A, Renman C, Westerterp K, Sjostrom M. Physical activity but not energy expenditure is reduced in obese adolescents: a case-control study. Am J Clin Nutr. 2002;76(5):935–41.
83. Freedson P, Sirard J, Debold E, et al. Calibration of the computer science and applications, inc. (CSA) Accelerometer 256. Med Sci Sports Exerc. 1997;29(5):45.
84. Kim Y, Beets MW, Welk GJ. Everything you wanted to know about selecting the "right" actigraph accelerometer cut-points for youth, but…: a systematic review. J Sci Med Sport. 2012;15(4):311–21.
85. Rennie KL, Wareham NJ. The validation of physical activity instruments for measuring energy expenditure: problems and pitfalls. Public Health Nutr. 1998;1(04):265–71.
86. WHO. Preamble to the Constitution of the World Health Organization as adopted by the International Health Conference, New York, 19–22 June, 1946; signed on 22 July 1946 by the representatives of 61 States (Official Records of the World Health Organization, no. 2, p. 100) and entered into force on 7 April 1948. Retrieved March. 1948;16.
87. del Valle MF, Perez M, Santana-Sosa E, et al. Does resistance training improve the functional capacity and well being of very young anorexic patients? A randomized controlled trial. J Adolesc Health. 2010;46(4):352–8.
88. Klein DA, Mayer LE, Schebendach JE, Walsh BT. Physical activity and cortisol in anorexia nervosa. Psychoneuroendocrinology. 2007;32(5):539–47.
89. Bouten CV, Verboeket-van deVenne WP, Westerterp KR, Verduin M, Janssen JD. Daily physical activity assessment: comparison between movement registration and doubly labeled water. J Appl Physiol. 1996;81(2):1019–26.
90. CDC. Centers for Disease Control and Prevention. Making physical activity a part of a child's life. 2011. http://www.cdc.gov/physicalactivity/everyone/getactive/children.html
91. Davis C, Kennedy SH, Ravelski E, Dionne M. The role of physical activity in the development and maintenance of eating disorders. Psychol Med. 1994;24(4):957–67.
92. Bratland-Sanda S, Sundgot-Borgen J, Ro O, Rosenvinge JH, Hoffart A, Martinsen EW. Physical activity and exercise dependence during inpatient treatment of longstanding eating disorders: an exploratory study of excessive and non-excessive exercisers. Int J Eat Disord. 2010;43(3):266–73.
93. Pollock ML, Gaesser GA, Butcher JD, et al. ACSM position stand: the recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in healthy adults. Med Sci Sports Exerc. 1998; 30(6):975.
94. Lee IM, Paffenbarger Jr RS. Associations of light, moderate, and vigorous intensity physical activity with longevity. Am J Epidemiol. 2000;151(3):293–9.

# Exercise Prescription and Pregnancy

26

Claudia Cardona Gonzalez, Elvis Álvarez Carnero, and Jacalyn J. Robert-McComb

**Abstract**

Sports and physical activity participation are an increasingly widespread habit among women of reproductive age. It is interesting that a pregnant woman has the opportunity to continue her exercise program without having to postpone it until after the puerperium. The knowledge about physiological adaptations either pregnancy or exercise must be the cornerstone which drive the exercise prescription on this specific population. Before implementing an exercise program, physical activity professionals need a deep knowledge about the physiological changes and the effects of exercise during the pregnancy either for women and fetus. This chapter focuses mainly on the effects of physical activity in pregnancy which would promote positive adaptations without increasing the risk of mother and the fetus. Additionally, we offer a review of the main exercise prescription guidelines from the most important institutions in physical activity area. Finally, practical ideas for exercises are suggested.

**Keywords**

Gravid women • Exercising during pregnancy • Exercise guidelines during pregnancy

C.C. Gonzalez, Ph.D., B.Sc.
Department of Sports Fundamentals,
Universidad Europea de Madrid, Madrid, Spain

Department Fundamentos de la moticidad y el Entrenamiento Deportivo, Universidad Europea de Madrid, Madrid, Spain
e-mail: claudiaandrea.cardona@gmail.com

E. Álvarez Carnero, Ph.D.
Biodynamic and Body Composition Laboratory,
Sports and Physical Activity Sciences,
University of Malaga, Málaga, Málaga, Spain
e-mail: ecarnero@uma.es

J.J. Robert-McComb, Ph.D., F.A.C.S.M., A.C.S.M., P.D. (✉)
Department of Health, Exercise, and Sports Sciences,
Texas Tech University, Lubbock, TX 79409, USA
e-mail: jacalyn.mccomb@ttu.edu

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_26, 

## 26.1 Learning Objectives

After completing this chapter, you should have an understanding of:

- The most important acute and chronic adaptations during pregnancy as a consequence of physical activity practice and/or exercise training;
- The benefits of regular physical activity for gravid women and fetus;
- How to avoid risks for fetus and mother associated with exercise training;
- Recommendations and guidelines to prescribe rational exercise programs for pregnant women during pregnancy and post-delivery.

## 26.2 Introduction

While pregnancy involves changes in women's physiology, several adaptations from regular physical activity (PA) practice must occur during this period of a life cycle. Although there has been a large discrepancy about the effect/consequences of exercise in the past, research has highlighted evidence about the benefits of exercise and how to decrease risks. So PA and exercise training must provide several benefits for both the mother and the fetus: blood pressure control, blood glucose regulation, healthy birth weight, and prevention of obesity and maintenance of pre-gravid physical fitness. Nevertheless, there appears to be some risks (hyperthermia, hypoglycemia, chronic fatigue, abortion, etc.) associated with excessive and poorly planned exercise regimens. These risks occur mainly when environmental conditions, volume and intensity thresholds of exercise load, and contraindications are not respected. Associations of obstetricians and gynecologists from different countries have collected scientific knowledge since the last century to develop useful guidelines to design safe and effective PA and exercise training programs.

## 26.3 Research Findings and Exercise Physiology During Pregnancy

### 26.3.1 Physiology of Pregnancy

The pregnancy period is a vital life cycle of the women that last 40 weeks in order to gestate a new human body. Parturition is the rather formal term for birth, and labor is the sequence of events that occur during birth. Classical pregnancy is divided into trimesters, each of them characterized by milestones. Weeks are also used to quantify gestational age at delivery, anatomical and physiological development of the fetus, or an assessment agenda (ultrasound or blood samples). In order to support the life growing inside her body, the pregnant woman undergoes several organic morphologic and functional changes (Table 26.1). The purpose of this section is to describe the most important physiological changes and mechanisms operating during pregnancy which are relevant to physical performance and health.

#### 26.3.1.1 Cardiovascular Function

The most striking changes occur at the level of the cardiovascular system. The excess heart load (caused by increased-weight bearing) should promote myocardium hypertrophy, either volumetric or wall thickness [1]. By the end of the first trimester, the raising of the diaphragm increases resting cardiac output throughout the pregnancy and peaks around the 20th week (40 % more than in the nonpregnant state), remaining steady during the final 3 months. The latter adaptation induces several hemodynamic changes at rest [2]. Systolic *blood pressure* (SBP) during pregnancy is completely stable, while the diastolic blood pressure (DBP) falls from 5 to 10 mmHg; this may be a consequence of a reduction in peripheral resistance and the development of circulation in the uterus and placenta up to 22 weeks.

**Table 26.1** Physiological changes during pregnancy

| System | Function | Mechanism |
|---|---|---|
| Cardiovascular | Higher peripheral oxygen demands (from 50 to 500 ml/min)<br>Cardiac Output increases by 40 % (Heart rate increases at the beginning up to 10–15 bpm and systolic volume by 10–12 %)<br>Peripheral resistance decreases from week 12 to week 24, becoming normal later<br>Blood returning to the heart is more oxygenated<br>Resting respiratory rate is reduced whilst vital capacity is preserved | Oxygen uptake increases from 15 to 20 % during the second half of pregnancy. This is caused by growing oxygen uptake from the uterus, placenta, and fetus<br>Resting HR in the pregnant woman increases by the increase in gonadotropin hormone, the lower activity of the parasympathetic system, and reduced concentration of blood catecholamine<br>This is caused by vasodilatation produced by hormones<br>Minute volume increases more than oxygen uptake<br>This is caused by a slight increase in inspiratory capacity |
| Blood | Plasma volume increases gradually until the 32 weeks (30–60 %)<br>Red blood cells number and size increase<br>The veins increase their capacity and peripheral vascular resistance decreases | This causes a hemodilution of the blood causing the physiological anemia of pregnancy<br>Produced by increased renal erythropoietin<br>Produced by effect of progesterone |
| Respiratory | Resting hyperventilation to compensate alkalosis (increased ventilation from 6 to 9 L/min) | This is produced by the increment of the tidal volume, which removes more $CO_2$ from blood, this raises PH. Also, it helped by chemoreceptors enhanced sensitivity to $CO_2$ in order to prevent fetal ischemia and acidosis |
| Renal and urinary | Dilated ureters and renal pelvis producing an increase of the dead space and a delay in the elimination of urine<br>Increased kidney size<br>Diastolic decreases 5–10 mmHg<br>Increased renal plasma flow, in the first quarter (600 to 836 ml/min) | This is caused by aldosterone and estrogen release, which balances progesterone<br>Caused by progesterone activity<br>Increased renin secretion and activation of the axis renin–angiotensin–aldosterone<br>The increased glomerular filtration. Later, it decreases |
| Gastrointestinal | Nausea, vomiting<br>Predisposition to tooth decay and gum hyperemia<br>Delay in time for gastric evacuation producing obstipation<br>Pyrosis | Associated with hormone secretion (gonadotropins and estrogens)<br>Related to hormone concentration in saliva<br>The growing of the uterus, moves bowel and stomach<br>Cardiac sphincter relaxation causes the hydrochloric acid in the stomach to reflux into the esophagus |

(continued)

**Table 26.1** (continued)

| System | Function | Mechanism |
|---|---|---|
| Metabolic | Diabetogenic effect of pregnancy<br>Change in blood lipid profile<br>Increased resting metabolic rate | This is due to some hormones like cortisol, estrogens, and lactogen from placenta can have blocking effects on insulin (insulin resistance). Pancreas can naturally produce more insulin, causing gestational diabetes<br>Lipids increase from 600 to 900 mg/ml. Produced by the influence of estrogens and cortisol<br>Caused by the increased demands from gestational state |
| Water metabolism | Increment in total body water | Hydrostatic vessels pressure<br>Increase in lower limb blood flow return<br>Capillary permeability<br>Sodium retention |
| Dermatological | Increased pigmentation<br>Possible appearance of stretch marks<br>Increased sweat secretion | Caused by estrogen activity<br>Hormonal activity produces muscle distension and low ligament elasticity<br>Sweating glands tend to have a higher activity due to elevated hormonal secretion |
| Skeletal system | Ligaments become more relaxed (Sacroiliac, sacrococcygeal, and pubic joints)<br>Increased lumbar dorsal curvature (lordosis)<br>Pain in zones around peripheral innervations<br>Frequent muscle cramps in the third term, especially in legs | Caused by relaxin<br>Produced by the displacement of the center of mass<br>Produced by liquid retention and relaxation of ligaments by hormonal increase<br>Related to sodium depletion |
| Hormonal changes | Human chorionic gonadotropin<br>Estrogens<br>Progesterone | Human chorionic gonadotropin develops the placenta<br>Estrogens increase the size of the uterus and prepare milk ducts for breastfeeding<br>Progesterone retains pregnancy and develops the lobules of the breast |
| Body weight | Increase between 9 and 12 kg | This is due to fetus growth; also mother gains fat mass, liquid, uterus blood volume, amniotic liquid, and placenta and breast tissue |

**Table 26.2** Rates of weight gain during pregnancy, as related with the pre-gravid (pre-pregnancy) weight status

| Pre-gravid BMI groups | Recommended total weight gain (kg) | Recommended rate of weight gain (kg/month)[a] |
|---|---|---|
| Underweight (<19.8 kg/m$^2$) | 12.5–18 | 2.3 |
| Normal weight (19.8–26.0 kg/m$^2$) | 11.5–16 | 1.8 |
| Overweight (>26.0–29.0 kg/m$^2$) | 7–11.5 | 1.2 |
| Obese (>29.0 kg/m$^2$) | 7.0 minimum | 2.0–0.9 |

Adapted from Gunderson, E. P. (2003). Nutrition during pregnancy for the physically active woman. *Clin Obstet Gynecol*, *46*(2), 390–402
*BMI* body mass index
[a]Rate of gain applies to gain during the second and third trimesters

The *blood volume* increases up to 40 %, peaking during weeks 32–34. However, plasma volume increases more than the blood cells, which produces hemodilution and less blood viscosity; as a consequence the circulation time is reduced, resulting in physiological anemia. Even the oxygen content difference between arterial and mixed venous blood (a-V $O_2$ difference) decreases during the first few months. Oxygen uptake ($VO_2$) increases between 15 and 20 % at the second half of pregnancy, mainly by a larger amount of oxygen consumed by the uterus, placenta, and growing fetus [3]. The a-V $O_2$ difference returns to previous levels (normally) in the third quarter.

#### 26.3.1.2 Pulmonary Function

During pregnancy higher ventilation (VE, L/min) is observed at rest and during incremental exercise than during the nonpregnant status [4]. Residual respiratory capacity is reduced while vital capacity is not altered as a consequence of a slight increase in inspiratory capacity. Tidal volume increases, even though the respiratory rate is held constant. *Gravidarum* hyperventilation leads to a compensated respiratory alkalosis, which decreases the concentration of carbon dioxide in the blood and increases pH slightly [3].

#### 26.3.1.3 Endocrine System

The endocrine system of pregnant women undergoes significant changes due to hormone production in the placenta. So the stimulation of the pituitary–adrenal axis raises the production of corticotropin (ACTH). This is associated with an increase of total and free cortisol [5]. The thyroid function is relatively stable during pregnancy, although the concentrations of T3 and T4 in plasma are increased [6]. However, thyrotropin (TSH) secretion may be reduced in early pregnancy.

PA promotes positive acute and chronic effects on insulin resistance syndrome, and pregnant women are not an exception [7]. Under resting conditions, catecholamine levels remain as before pregnancy; however, a misbalance between hormones from the endocrine pancreas can be observed (glucagon and insulin). This is due to the antagonized diabetogenic hormones of the placenta (human lactogen). Both alterations would contribute to insulin insensitivity, commonly diagnosed during pregnancy. Peak concentrations of glucose and insulin are progressively higher, and insulin sensitivity reduces up to 80 % accordingly to the tolerance test glucose (TTG). This previous alteration would state a grade of insulin resistance during the late phase of pregnancy.

#### 26.3.1.4 Metabolism, Energy Expenditure, and Weight Control

A physiological weight gain must happen during pregnancy to support the growth of the fetus at the beginning of the third trimester, so there is an increase between 3.5 and 5 kg of fat deposits (mainly from the 10th to 30th week). This alteration is associated with a normal range of increased weight, which is suggested to be dependent of the pre-pregnant body mass index (BMI [see Table 26.2]).

A proportional increase of energy intake from a balanced diet must be ensured. There must be

enough of a weight gain to support the needs of fetus. During pregnancy, a daily intake around 300 kcal of additional energy is required to maintain metabolic homeostasis. Pregnant women performing exercise should eat a proper diet well balanced in energy [8] and micronutrients [9]. Women who are exercise training should increase their energy intake proportionately to meet the energy costs of the exercise [8].

Connected with weight and body composition modifications, there are some normal alterations in energy expenditure, fat and carbohydrate metabolism [10]. Regarding the components of total daily energy expenditure (TDEE), basal metabolic rate is elevated during pregnancy. The increase in TDEE could be related to the additional cardiac and renal costs observed during the first part of pregnancy, in addition to the growth of the placenta, uterus, and fetus during the second part of pregnancy [11]. Both of these factors contribute to the increased TDEE described in women under gestation [10]. Other mechanisms which could explain body composition and TDEE alterations are related to changes in fat metabolism. There is an increase in the production of triglycerides in the plasma up to 3 times normal. The increased concentration of free fatty acids during late pregnancy is 2–4 times above normal [5]. This is a consequence of protective mechanism, where the mother stores carbohydrates and uses more fat as an energy source in order to preserve placental, fetus, and uterus demands. The increment in cortisol and impairment of insulin sensitivity help to meet the glucose needs for a healthy growth of the fetus [5]. So during the third trimester acetone in the plasma is increased and fasting plasma glucose is decreased, a metabolic state characterized by hypoglycemia, hypoinsulinemia, and hypoketonemia can occur [1].

PA energy expenditure may not be a primary cause to increase energy intake, since improved daily task efficiency has been well documented in gestating [10].

#### 26.3.1.5 Musculoskeletal System

Several adaptations during pregnancy and labor will promote changes in musculoskeletal tissues. The action of the hormone relaxin progressively leads a softening of the ligaments, particularly at the region of the pubic symphysis and sacroiliac joints. This softening reaches the peak at the beginning of the third trimester. So the growth of uterus size induces great pressure against the lumbar spine. This increases lordosis, joint angle changes, and joint relaxation, which lead to lumbar pain. Finally, relaxed pubic symphysis can move a few millimeters causing pain when walking or standing. All of these adaptations will be important concerns when selecting activity to prescribe exercise during pregnancy.

## 26.4 Contemporary Understanding of the Issues

### 26.4.1 Benefits of Exercise During Pregnancy

Since practices of PA and exercise training have positive effects on the health of nonpregnant women, the outcomes highlighted in Table 26.3 could be expected during pregnancy (see Table 26.3). Sadly, a decrease in daily PA has been widely reported along gestation [10]. Misperceptions about the risks of exercise during pregnancy might be one of the most important reasons explaining this behavior [12]. For example, Zhang and Savitz [13] have shown that 60 % of pregnant women were sedentary, which represented twice the sedentarism of the US adult population.

Despite old beliefs about the harmful consequences of exercise during pregnancy, there is more than ample evidence about the healthy outcomes of exercise for both the mother and the fetus [14]. The risks associated with exercise training are well known and easily managed [14, 15]. Table 26.3 highlights the benefits associated with regular PA or exercise training during gestation.

#### 26.4.1.1 Improved Aerobic Fitness

Hormonal and physiological changes occurring in pregnancy will affect the cardiovascular system during exercise [16]. Cross-sectional evidence

**Table 26.3** Possible benefits of exercise during pregnancy

| | |
|---|---|
| Improvement of cardiovascular fitness | • It decelerates heart rate |
| | • It improves circulation |
| | • It helps prevent varicosities |
| | • It help to regulate blood pressure |
| Improvement of muscle fitness | • It improves muscle tone |
| | • It reduces cramps |
| | • It corrects posture |
| | • It eases back pain |
| Prevention of excessive weight gain | • It improves the general physical condition of the pregnant woman and it reduces the risks of pregnancy and labor |
| | • It helps to manage cellulite |
| | • It reduces fluid retention |
| Digestive system regulation | • It reduces digestive discomfort |
| | • It reduces obstipation |
| Psychological well-being enhanced | • It reduces: Fatigue, depression, and insomnia |
| | • It helps control anxiety |
| | • It helps to reduce stress |
| | • It creates healthy lifestyle habits |
| Prevention of gestational diabetes | • It helps to regulate glucose and insulin needs |
| | • It prevents excessive weight gain |
| Enhancement of postpartum recovery | • It reduces hospitalization time |
| | • It reduces cesarean section risk |
| | • It helps restore the physical appearance |

suggests that aerobic fitness as assessed by $VO_{2max}$ can be reduced in women who do not practice aerobic exercise during pregnancy. However, if women continue to exercise during their pregnancy, aerobic fitness should be maintained as long as they are active during their pregnancy [17]. Female athletes (athletes vs. sedentary) are generally able to maintain their level of fitness ($VO_{2max}$, power output, heart rate [HR] at anaerobic thresholds) during pregnancy if they continue to train [18]. As a general rule, $VO_{2max}$ will not be reduced during pregnancy if women maintain their exercise training, at least when expressed by L/min [19]. However, if the unit is expressed in relative terms (ml/kg/min) rather than absolute terms (L/min), a slight reduction of 9 % would be observed during the first weeks of postpartum. In athletes, this diminution might be recovered 4 months after delivery [20].

A well-reported adaptation is improved efficiency during pregnancy for both weight-bearing and weight supported activities. This effect is correlated with the enlargement of non-consuming-oxygen tissues during exercise such as fat mass, placenta or extracellular fluid. Therefore, absolute $VO_2$ (L/min) remains unchanged during gestation [3]. During pregnancy it is unusual to see improvements in $VO_{2max}$ or aerobic power; therefore, the purpose of aerobic training should be to induce healthy physiologic outcomes in child bearing women that will augment the welfare of the mother and the fetus, such as a reduction of insulin resistance [21]. Another example is that trained pregnant women have a lower resting HR than untrained pregnant women which allows active gravid women to have a greater cardiac reserve than untrained gravid women [22]. However, during pregnancy there is an increase in HR (about 15 beats per minute [bpm]) compared to the nonpregnant state, regardless of the exercise regimen.

#### 26.4.1.2 Decreased Lumbar Pain

Lumbar pain is a common event of pregnancy. At least 50 % of pregnant women suffer lumbar pain [23]. It seems that this incidence is lower in female athletes, due to improved muscular fitness. However, this does not mean that exercise prevents all lower back pain in pregnancy [24]. It does seem that exercise during the second half of pregnancy reduces the *intensity* of back pain; the mechanism proposed was an enhanced flexibility of the spine without altering the lordosis.

A second paradigm related with the etiology of lumbar pain has been focused on weight gain and the parallel loss of stability of the pelvic girdle. This loss of stability of the pelvic girdle is due to hormonal changes which causes weight gain in the abdominal region. During pregnancy, women experience hormonal changes that occur in preparation for delivery. The hormone relaxin is released, which loosens joints and ligaments, which can cause hip pain and other aches. As the

baby grows and extra weight is placed on the pelvis, the pelvis can shift, which causes pain. Changes in a woman's posture can also contribute to hip pain, as her back and muscles are pulled in a different way to carry the baby. This places strain on the muscles and causes the pelvis to tilt out of alignment. Back exercise treatment has implications not only for pregnant women, but also for health care costs and labor productivity. Since one goal of the exercise program is to restore joint biomechanics, lumbo-pelvic stabilization following proper posture training must be a cornerstone strategy for exercise prescription during gestation. A study published in 2005 reported significant reductions in the intensity of lumbar pain and improved mobility of the spine, in spite of no observed changes in lordosis [23]. So it appears that internal mechanisms, more than the lordosis angle, are responsible for low back and pelvic pains; the hormone relaxin has been suggested as an important factor [25].

#### 26.4.1.3 Weight Control

Exercise promotes important benefits for weight control during gestation and postpartum. Preventing a body weight increase >10 % of gestational mass reduces the risk of diabetes or hypertension, and the probability of delivering a macrosomic baby [14]. In accordance with traditional studies, exercise combined with diet is the best way to control weight gain during pregnancy. Thus, gravid women who performed physical exercise gained less weight, without impacting negatively on the fetus [26]. However, it seems that only individualized nutrition and PA programs are successful to avoid increasing body weight during pregnancy. Some examples of successful interventions were aqua-aerobics 1–2 days/week; supervised walking/biking at 60 % of $VO_{2max}$; walking 3–4 days/week at 30 % of HR reserve; or resistance training with a personal trainer [14].

Additionally, controlling weight prevents gestational diabetes and possible future obesity (next paragraph). Also, a healthy weight facilitates the delivery; hence, women who exercise during pregnancy had shorter labors as well as labors that were faster and easier [27, 28].

#### 26.4.1.4 Prevention of Gestational Diabetes

Gestational diabetes is a temporary condition which occurs around the end of pregnancy as a result of the action of insulin and placental hormones. These women develop a physiological insulin resistance state and generate macrosomias in the fetus. A lack of PA as well as being overweight or obese can lead to diabetes mellitus and an increased risk of preeclampsia [29].

Some studies have indicated that women involved in 30 min (min) of moderate PA on most days of the week during pregnancy reduced the risk of gestational diabetes compared to sedentary women 50–75 % [30]. Even, among overweight and obese gravid women with gestational diabetes mellitus, a simple exercise program (25 min 3–4 days/week, and increments of 2 min/week until 40 min) showed important benefits in glucose and insulin regulation [14]. Likewise, exercise has been shown to improve the lipid profile, hence lowering the risk of diabetes [17].

#### 26.4.1.5 Hypertension and Preeclampsia

There are several disorders related with high blood pressure during gestation; the most prevalent are gestational hypertension, chronic hypertension, and preeclampsia/eclampsia [31]. Preeclampsia is a disorder related to hypertension, which occurs in 3–7 % of pregnancies. Women with preeclampsia may have glucose intolerance, hypertriglyceridemia, systemic chronic inflammation, and endothelial dysfunction. Furthermore, preeclampsia has been associated with perinatal complications and it is one of the most important reasons for maternal mortality [32]. The risk of preeclampsia appears to be reduced about 30 % when performing PA before and during gestation [33, 34]. Moreover, it seems that pregnant women who suffer anxiety or depression are 3 times more at risk for preeclampsia. Therefore, regular PA would be an additional benefit for them [26, 34]. Although several studies have shown the positive effect of PA on blood pressure regulation during pregnancy, several variables could influence the results. A recent review analyzed the influence of

exercise on hypertensive disorders and it concluded that exercise seems to protect against these complications, but more randomized control trials must be done to confirm a causal relation. As a suggestion, data from large cohort studies suggest that >25 times/month or 270–419 min/week of leisure PA can help gravid women reduce the likelihood of suffering pre-eclampsia [14].

#### 26.4.1.6 Psychological Benefits

Some psychological dimensions have been studied in pregnant women undergoing exercise training: Body image was shown to improve, symptoms of depression were reduced [35], self-esteem increased [36], and level of stress may be lowered in exercising pregnant women [37].

Regarding female athletes, additional psychological benefits could be attained among well-trained women, namely, it seems that they return to competition sooner and they have more confidence and self-motivation [38]. Improved performances observed after having children have been attributed to either physiological or psychological reasons [39]. Table 26.3 highlights both the physiological and psychological benefits of exercise for pregnant women.

#### 26.4.1.7 Benefits for the Fetus

##### Less Complications During Labor

A research conducted in 1984 showed that women who performed resistance exercises at the same level as before pregnancy and continued to exercise until the third trimester of the gestation period, gained less weight, and gave birth faster. Additionally, their babies were thinner than those from women who quit their exercise regimen before 28 weeks [40]. It seems that women who trained at medium intensity gave birth later than those who did exercise at high intensity exercise, although the training duration was less in the first (training at medium intensity) than in the second group of active women (training at high intensity). In addition to this, most studies concluded exercise reduces the duration of the active stage of labor and diminishes the incidence of obstetric difficulties during labor [41]. However, a meta-analysis on the incidence of obstetric difficulties during labor found no difference between mothers who did exercise programs and control groups in duration of labor, birth weight or APGAR score [15]. The APGAR score is a system of assessing the general physical condition of a newborn infant based on a rating of 0, 1, or 2 for five criteria: HR, respiration, muscle tone, skin color, and response to stimuli. The five scores are added together, with a perfect score being 10. Nonetheless, it must be difficult to isolate the effects of exercise on the characteristics of the fetus at birth, since it must be influenced by many other factors such as genetics, nutrition, socioeconomic elements, and environmental factors [42].

##### More Active Children

Children activity must be a key component for a healthy development. An interesting study examined children between 1 and 5 years and analyzed the mother's level of PA influence on motor and intellectual capacities of their children. The authors concluded that at 1 year of age, children whose mothers exercised during pregnancy showed improved motor skills, but mental abilities and morphological characteristics were identical to those of mothers who were not involved in training. When 5-year-old children were assessed the children of exercising women were much thinner and had much better levels of intelligence (mainly in oral skills) than the children whose mothers were not involved in training. The consequences of these data on future life remains to be elucidated [26].

Studies conducted for 5 days in children of mothers who maintained their exercise programs during pregnancy resulted in differences in the profiles of babies compared to sedentary mothers: the babies of exercising mothers were more responsive to environmental stimuli and bright light, with better motor organization according to the scale of humor [43]. However, all of these positive outcomes need to be confirmed by other studies with different samples.

### 26.4.2 Risks of Exercise in Pregnant Women

As pointed out in the previous paragraphs, being physically active during pregnancy can result in

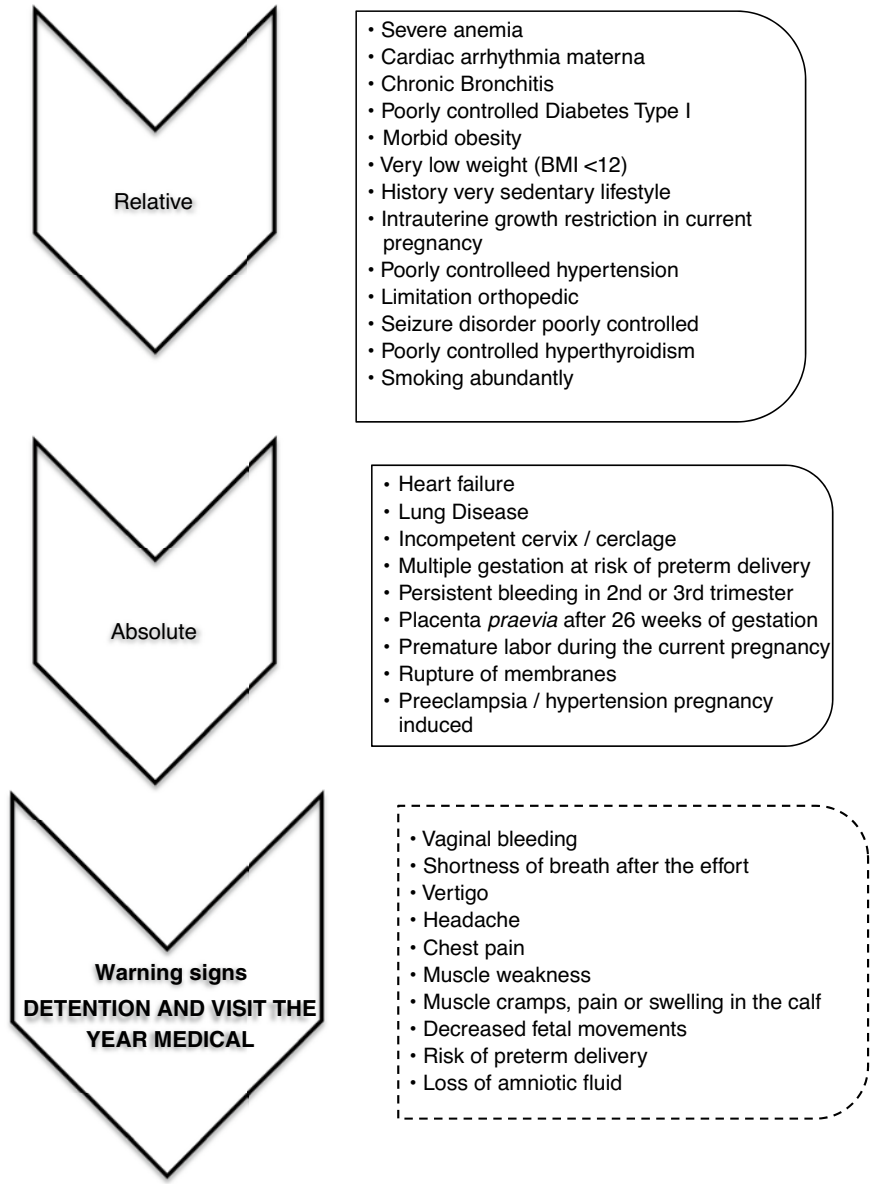



**Fig. 26.1** Absolute and relative contraindications for and during practice exercise. Adapted from Artal, R., & O'Toole, M. (2003). Guidelines of the American College of Obstetricians and Gynecologists for exercise during pregnancy and the postpartum period. *Br J Sports Med*, *37*(1), 6–12; discussion 12

several positive outcomes. Nevertheless, there are many ways in which sport and PA during pregnancy may induce risk to the mother and fetus. Therefore, certain exercises must be avoided or strenuous PA when specific conditions appear. The American College of Obstetricians and Gynecologists (ACOG) has delineated several guidelines in order to guide pregnant women and PA professionals to prevent risks associated with exercise and PA practice [44, 45] (see Fig. 26.1).

Additionally, complications related with poorly planned exercise have also been reported. For example, severe *hypoglycemia* can overcome gravid women after a sharp and intense exercise,

which if repeated chronically can prompt malnutrition and low birth weight in the fetus [46]. *Chronic Fatigue* is other common symptom associated with erroneous exercise prescriptions; this must be a main concern when planning PA for gravid women, because the physiological characteristics of pregnancy can induce early fatigue. The excess body weight gained during gestation must be a factor inducing fatigue from low workloads. Also, due to increasing human chorionic gonadotropin hormone (better known as hCG), the hemodynamic changes and lower parasympathetic activity of pregnant women induce a higher HR of about 15 bpm than while nonpregnant; hence, it should be normal that gravid females got more tired than in the normal state [47]. The hormone hCG is made by cells that form the placenta, which nourishes the egg after it has been fertilized and becomes attached to the uterine wall. Moreover, all strenuous activities performed mainly during the third trimester of pregnancy can lead to chronic fatigue, thus special care must be used when prescribing weight bearing activities. *Musculoskeletal injury* is other risk related with the augment of 15–30 % body mass during pregnancy. Also, biomechanical modifications happening in the pelvic/abdominal region, greater elasticity of the ligaments, and changes in the musculature (abdominal diastasis) all can promote inefficiency of movement skills [48]. So all these factors together can make it easier to get a musculoskeletal injury while performing basic movements such as walking for a long time or of moderate intensity [49].

### 26.4.3 Risks of Exercise During Pregnancy for the Fetus

Similar with the mother, if PA were not well conducted the fetus would suffer risks related with exercise. This section describes events affecting the fetus as a consequence of excess or wrong exercise.

#### 26.4.3.1 Acute Hypoxia

It has been hypothesized that the fetus may have hypoxia during aerobic exercise by the redistribution of blood flow, and thus more oxygen goes to the muscles instead of going to the uterus. The fetal HR, which reflects cardiac output, is usually between 120 and 160 bpm. An exercise event that induces a HR elevation higher than 160 bpm for 10 min is designated as tachycardia, and lower than 120-bpm as bradycardia. Parer [50] reported an increase of 10–30 bpm in the fetus during maternal exercise. The HR responses in the fetus could reflect tissue oxygenation. The a-V $O_2$ difference (indicating the difference of the oxygen content of her arterial blood and her venous blood) was improved in the active mother that suggests more oxygen was being delivered to her tissues [36].

#### 26.4.3.2 Acute Hyperthermia

Excessive elevation of temperature primarily during the first weeks of pregnancy may be a risk to development defects and fetal death [51]. Also, the fetal temperature is about 0.5 °C higher than the maternal temperature [52]. However, the pregnant woman has thermoregulatory mechanisms that increase the circulation to the skin to lose heat, so the increase in temperature of the fetus is tightly regulated to prevent fetus hyperthermia. Regardless, it is not advisable to perform exercise with high environmental temperatures (above 40 °C). Also it is mandatory to drink enough water in order to avoid dehydration and internal warming [53, 54].

#### 26.4.3.3 Low Glucose Availability

The use of carbohydrates by skeletal muscle in pregnant women increases significantly during strenuous exercise [55]. This may limit the ability to extend vigorous exercise, and may predispose pregnant women to hypoglycemia [56]. This effect may be the result of the insulin resistance that develops in the latter half of pregnancy [29]. However, a drop in blood glucose levels, which can limit the consumption of glucose by the fetus, may be a consequence more probable of long-term nutritional mistakes than exercise [57]. Nonetheless, this should induce low weight or alterations in the growth of fetal organs and tissues, so carbohydrate intake after exercise training should be considered, mainly gravid women training longer than 60 min.

#### 26.4.3.4 Abortion in the First Quarter

Beliefs of PA as a promoter of abortion have not been supported by literature [54]. Clearly, the risk of spontaneous abortion was not found to be higher in athletes than in healthy controls [58]. However, exercise in the first trimester can lead to an early abortion, so avoiding strenuous PA is one of the most important restrictions to avoid fetus death prematurely [59].

#### 26.4.3.5 Risk of Preterm Delivery

Acute exercise may induce premature birth because it increases the secretion of catecholamines, especially norepinephrine, which in its turn causes uterine contractions after exercise [60]. This hypothesis was analyzed in a study that recruited more than 7,000 women. The researchers found that 8 h a day in a standing position increased the risk of preterm delivery. However, 4 h a day of work or exercise was not associated with preterm delivery. Also, there were no significant differences between sedentary and physically active jobs for the percentage of premature babies. Moreover, the time of PA was not associated with a higher risk of preterm delivery [61]. However, women at risk of preterm birth are advised to avoid exercise training [44].

#### 26.4.3.6 Reduced Birth Weight

As previously explained, there may be a possible mechanism related with fetal hypoxia, which explains low birth weight of the newborn. In accordance with this hypothesis, there appears to be a dose–response relationship between days/energy expenditure per week of training and low birth weight among athletes. As a suggestion, exercise energy expenditure less than 2,000 kcal/week or intense exercise 1 h a day performed 5–7 days/week, must avoided in order to reduce the probability to deliver a low-weight baby, mainly after 28th week of gestation [20]. Conversely, recreational exercisers or those who meet the American College of Obstetricians and Gynecologists (ACOG) guidelines gave birth to babies with normal weight, even when vigorous intensity was performed [14, 62]. Thus, children of high-level female athletes (and ex-participants in the Olympic Games) have been of normal weight [63]. It seems that individual exercise and nutritional prescriptions need to be followed for a healthy birth weight. There seems to be a lower (>120 min/week) and upper thresholds (intense exercise 1 h a day performed 5–7 days/week) for the quantity of exercise to deliver a healthy baby [14, 20].

### 26.4.4 Recommendations for Exercise During Pregnancy

#### 26.4.4.1 General Recommendations for Programming Exercise During Pregnancy

As pointed out in the previous sections, PA has several positive effects for both the pregnant women and fetus. A recent review focused on daily PA interventions (exercise training and unstructured PA) analyzed the positive and negative effects of PA during pregnancy. The results of this review suggested that PA performed without supervision or prescription could result in lower benefits than prescribed exercise [14]. Furthermore, the analysis of gestational physiology and possible complications as consequence of excessive workload makes us think that care must be put into designing exercise or PA advice for pregnant women. In the previous sections, we have given some specific guidelines for particular cases; however, exercise prescription requires an integration of all determinants of the workout.

Load (duration and intensity), mode (contraction pattern and metabolic pathway), type (activities), periodization, nutrition, and environment have been the variables most commonly studied in order to define specific guidelines by the representative professional/academic associations of pregnancy or exercise [44, 45, 59, 64–68]. These recommendations have been updated during the last decades. We have summarized the most recent guidelines (see Table 26.4) which have been synthesized from the ACOG guidelines and the Physical Activity Readiness Examination (PARmed-X for Pregnancy).

**Table 26.4** Recommendations for exercise prescription during pregnancy

| Exercise prescription variables | Level of performance/practice | | | |
|---|---|---|---|---|
| | General | Sedentary | Recreational | Elite/athlete |
| Bout volume | At least 15 min | 30 min | 30–60 min | Strength: high repetitions (15–20)<br>60–90 min |
| Bout intensity | 140–160 bpm (safe for walking and biking)<br>%$HR_{max}$: 70 % (bike and aerobics)<br>%$VO_{2max}$: 70 % (swimming and water exercises); lower HR than biking<br>"Talk Test"<br>High intensity[a] | RPE: moderately hard<br>%$HR_{max}$: 65–75 % | RPE: moderately hard to hard<br>%$HR_{max}$: 65–80 % | Strength: Light weights<br>RPE: hard<br>%$HR_{max}$: 75–80 % |
| Frequency | 3 d/wk | 3 d/wk | 3–5 d/wk | 3–5 d/wk |
| Mode | Low impact<br>Extensive isometric contractions, anaerobic exercises[a]<br>Strength training without Valsalva maneuver | Low impact | Low and moderate impact | Low and moderate impact<br>Maximum and isometric strength exercises[a]<br>Exercise in supine position first quarter[a] |
| Type (activities) | Childbirth preparation (minimum)<br>Start with no weight-bearing exercises (cycling, swimming, etc.)<br>Walking and brisk walking<br>Aerobics<br>Water exercises are recommended to relieve back pain. Exercise increases blood mobilization and reduction of edema<br>Pilates under individual supervision | Walking, cycling, swimming, and water aerobics | Low impact, and progress to moderate as jogging/running, tennis | Jogging/running, tennis, and similar, progress to racing activities<br>Change races by elliptical device<br>Resistances machines<br>Water exercises to prevent back pain |

(continued)

**Table 26.4** (continued)

| Exercise prescription variables | Level of performance/practice | | | |
|---|---|---|---|---|
| | General | Sedentary | Recreational | Elite/athlete |
| Avoid[a] | Participating in competitions, contact sports or risk of trauma[a]<br>Exercises that could overload the lower back[a]<br>Exercise at moderate altitude (2,500 m over the sea)[a]<br>Frequently shallow diving (never deep)[a]<br>Sport contacts (team sports or martial arts)[a]<br>Horse riding, skating, skiing, climbing and others, which increase fall risk[a] | | | Training with infection, fever, or fatigue[a]<br>Competition events[a]<br>Contact sports[a]<br>Quick changes of direction (ligamentous laxity)[a]<br>Anaerobic exercises[a]<br>Stop training with symptoms such as pain, bleeding, etc. |
| Environment | Avoid high temperatures[a] | | | Avoid high temperatures[a] |
| Nutrition and supplementation | Adequate nutrition and hydration | | | Dehydration[a] |
| Periodization | Supine position during the first quarter; start the training program first quarter[a] | Begin with 15 min and progress to 30 min<br>From 3 to 5 d/wk | | 3 d/wk in the first and third quarter<br>5 d/wk in the second quarter |

Adapted from: (a) Artal, R., & O'Toole, M. (2003). Guidelines of the American College of Obstetricians and Gynecologists for exercise during pregnancy and the postpartum period. *Br J Sports Med*, *37*(1), 6–12; discussion 12; (b) Paisley, T. S., Joy, E. A., & Price, R. J., Jr. (2003). Exercise during pregnancy: a practical approach. *Curr Sports Med Rep*, *2*(6), 325–330; (c)Wolfe, L. A., &Weissgerber, T. L. (2003). Clinical physiology of exercise in pregnancy: a literature review. *J Obstet Gynaecol Can*, *25*(6), 473–483

$HR_{max}$ maximal heart rate, $VO_{2max}$ maximal oxygen uptake, *RPE* rate of perceived exertion, *bpm* beats per minute (heart rate), *d/wk* days/week

[a]Indicate practices that must be avoided

Before starting any exercise program, the general state of the individual pregnant woman should be kept in mind. It is therefore necessary to consider the contraindications that exercise may have, both absolutely and relatively—Meaning an absolute contradiction to exercise and a relative contradiction to exercise: Absolute means DO NOT EXERCISE UNDER ANY CONDITIONS and a relative contraindication means that a case-by-case decision must be made depending on the pros and cons of exercise for this situation. Additionally, several physical signs must be under control in order to stop the practice and proceed with an emergency protocol if necessary (see Fig. 26.1).

The most important aspect of exercise prescription in pregnancy is intensity. Aerobic training appears to be the mode of training most studied and used when prescribing exercise for gravid women. The HR is the classical parameter used to quantify the intensity of continuous aerobic activities; so several thresholds or percentages of maximum HR have been proposed to get benefits and diminish the risks. The main physiological variable determining HR values during PA must be $VO_2$; hence, estimation of $VO_{2max}$ has been an important concern in order to establish training goals.

A meta-analysis on the effects of exercise on pregnancy outcomes did not find evidence of harm to the mother or the fetus with training up to 80 % of $HR_{max}$ (144 beats in women around 26 years old), 43 min/session, up to 3 days/week [15]. Nevertheless, these values can be influenced by the level of sport and activity practice before gestation.

Athletes can continue with exercise training during pregnancy, but intensity should be lowered [69]. They also might be able to perform PA with lower HR than sedentary women. However, it is difficult to define the limits of training with pre-gravid athletes participating in different sports, so more research is needed to find HR ranges for specific modalities [20].

Sedentary pregnant women who performed an exercise training program during quarter 2 and 3 (140–150 beats, 25 min/session, 3 times/week) had decreased submaximal HR when compared to a control group with a sedentary lifestyle [15].

An attenuated response of the sympathetic system during pregnancy induces reductions of $HR_{max}$ in response to exercise. This has lead to a reduction in the importance of HR as a sensitive indicator of intensity. Likewise ACOG recommends the use of RPE to monitor exercise intensity, since HR is affected by the hemodynamic changes during gestation [44, 45].

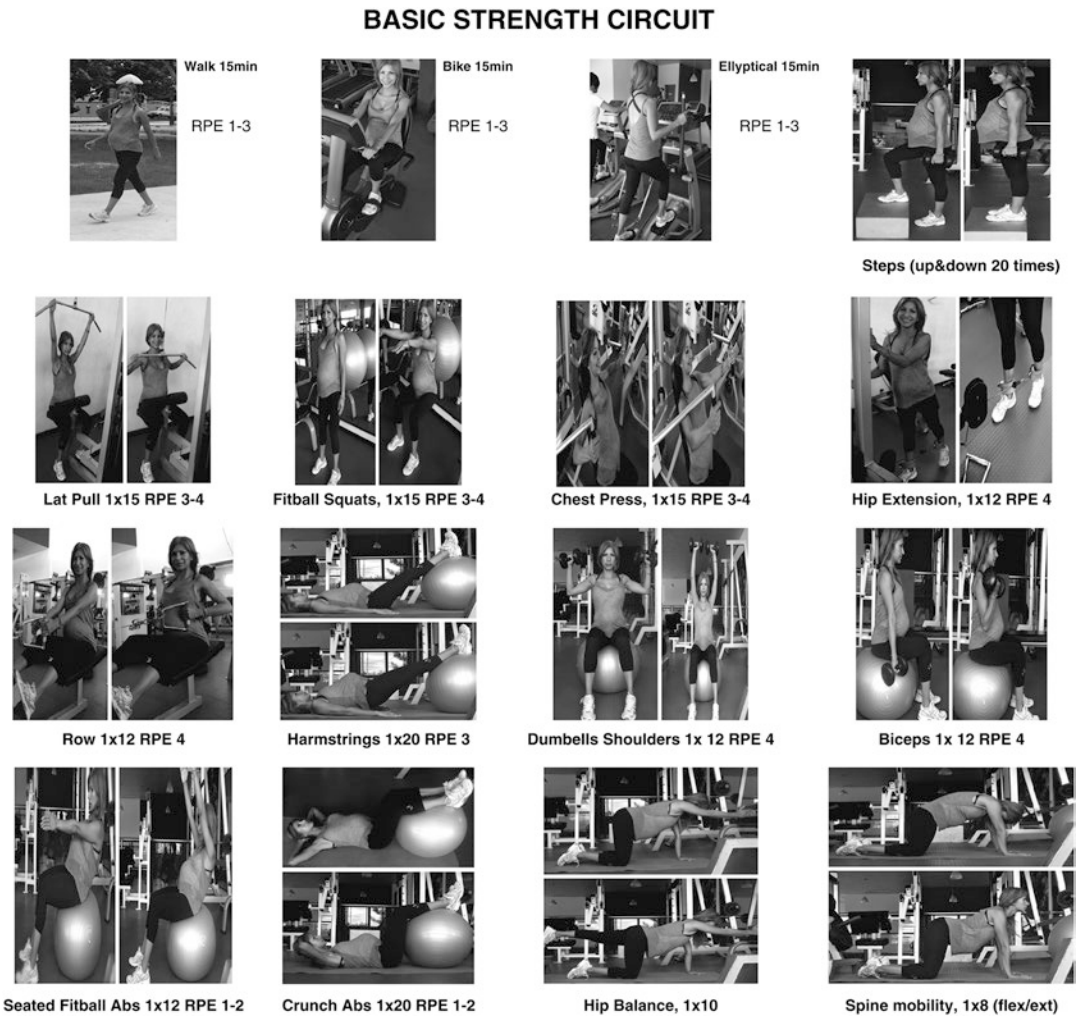
Methods to estimate $VO_{2max}$ are traditionally useful in clinical and field settings to avoid time consuming and expensive laboratory tests. Bike tests using external load and the Astrand nomogram are widely applied by exercise physiologists, clinicians, and coaches [70]. However, the Astrand nomogram has been shown to overestimate the $VO_{2max}$ by about 9 %, in pregnant women. Other methods such as the linear regression from the relationship between $VO_2$-HR using submaximal loads (such as the YMCA protocol), overestimated $VO_{2max}$ by 6 %. These regression methods are procedures well fitted to non-gravid women. However, the necessary assumptions to make valid measurements of $VO_{2max}$ are not met during gestation, mainly when submaximal $VO_2$ is estimated and not measured.

Currently, there are not many well-validated methods to estimate $VO_{2max}$ during pregnancy. One of the few methods for pregnancy utilizes a single constant workload protocol. It requires the measurement of HR at the end of a 6 min steady-state constant exercise on bike. Afterwards, 26.1 and 26.2 equations are used to estimate $VO_{2max}$ [39].

$$\%VO_{2\max} = \left(0.634 \times FC\right) - 30 \qquad (26.1)$$

$$VO_{2\max} = VO_2 / \%VO_{2\max} \times 100 \qquad (26.2)$$

Another key element of programming exercise is the mode or modality of the activities. There are many aspects to consider when selecting safe exercises; these are summarized in Table 26.4. A basic training circuit is illustrated in panel 26.1




#### 26.4.4.2 Post-delivery

Although there are other important concerns associated with PA, for example, the reestablishment of muscular fitness, the quality of breastfeeding, and the mother's weight gain, however, the first aim should be to perform exercise focused on the recovery of the *strength of perineal muscles and later in the abdominal* region. Nevertheless, several physiological modifications persist at least during the first 4 weeks, especially the cardiorespiratory system, and thus some specific temporal guidelines have been suggested [64, 65]. Few hours of delivery, pelvic floor exercises might be initiated.

- The first 3 weeks, exercises to recover abdominal wall *tonus*. Limit exercise training.
- After 40 days of delivery, moderate aerobic activity outdoors jumping or running must be delayed until after 8–12 weeks after birth (risk of trauma for the pelvic floor).
- Hypotension is common. Restrain sudden changes of position.
- Restart strenuous or competition activity only 8 weeks after delivery.
- The first aim is to perform exercise focused on recovering first strength of perineal muscles and later in the abdominal region.

Regarding weight control, gestational *excessive weight* gain is the strongest predictor of postpartum weight retention. Also, it has been reported that weight retention after delivery and low physical activity, may also contribute to obesity [70]. So it has recently been suggested that individualized diet and exercise training plans are needed in order to manage a healthy weight loss [71]. However, more important than weight loss is an enhanced body composition profile, since exercise training preserves fat free mass (FFM), while dieting alone reduces fat mass and FFM. The best results are ensured incorporating both diet and exercise.

In addition, a balance between training energy expenditure and diet must be maintained in order to ensure healthy baby growth and development. Excessive PA and poor energy intake would impoverish milk production and quality, which does not allow the infant to *gain weight*. General guidelines encourage mothers to intake enough liquids and nutrients to meet increased energy requirements as a consequence of physical activity. Also, nursing women are advised to feed babies before exercise practice; this procedure helps the mother to diminish the discomfort of engorged breasts and balance the acidity of her milk [64]. Reduction of postpartum depression symptoms is an additional benefit of exercise. This has been widely reported, but evidence has not been demonstrated without revocation [72].

## 26.5 Future Directions

Although the knowledge of exercise-applied pregnancy has been improved widely during the last few decades, several concerns remain to be defined. The effects of exercise training on the physiological function and body composition of children are not completely elucidated, so longitudinal studies need to be designed in order to establish this relationship. Also, data of the dose–response relationship between exercise-training load and health outcomes have not been massively collected. Finally, improved methods to assess physical fitness and body composition for gravids need to be developed in order to improve exercise prescriptions in field settings.

## 26.6 Concluding Remarks

The guidelines presented in this chapter should only be used as a general rule. The pregnant woman should be monitored closely to adjust training loads as a function of body mass, blood markers, and the fetus developmental changes. Individual prescriptions should be based on assessing energy requirements and capacity as tight as possible. A flow networking must be established between gynecologist, nutritionist, and exercise physiologist in order to maximize benefits to both the fetus and the mother.

The first aim of exercise program must be to guarantee the safety of the mother and the fetus more than performance or esthetic outcomes during pregnancy and after-delivery. Hence, a checklist of risks must be kept in mind when planning goals of PA before prescribing training loads.

While there are no well-developed methods to perform the assessment of physical fitness, these assessments must be carried out cautiously. Regarding body composition estimations, we have not suggested any models or techniques because of changes of FFM hydration and total body water during pregnancy. The change in these variables during pregnancy invalidates the traditional methods to estimated fat mass, FFM or skeletal muscle mass [73]. Given the choice, we would suggest measuring accurately limb skinfolds and circumferences in order to obtain a general idea of changes in fat mass and FFM.

A precise recall of volume, intensity, and type of exercise, in addition to physical and clinical assessment outcomes, is the best way to upgrade and modify PA programs for gravid women rationally. This will allow professionals to understand the individual dose–response relationship in each specific case and the need for individualized exercise prescription in order to maximize the health benefits for both the mother and the fetus.

## References


1. Revelli A, Durando A, Massobrio M. Exercise and pregnancy: a review of maternal and fetal effects. Obstet Gynecol Surv. 1992;47(6):355–67.
2. Capeless EL, Clapp JF. Cardiovascular changes in early phase of pregnancy. Am J Obstet Gynecol. 1989;161(6 Pt 1):1449–53.
3. O'Toole ML. Physiologic aspects of exercise in pregnancy. Clin Obstet Gynecol. 2003;46(2):379–89.
4. Jensen D, Webb KA, Wolfe LA, O'Donnell DE. Effects of human pregnancy and advancing gestation on respiratory discomfort during exercise. Respir Physiol Neurobiol. 2007;156(1):85–93.
5. Bessinger RC, McMurray RG. Substrate utilization and hormonal responses to exercise in pregnancy. Clin Obstet Gynecol. 2003;46(2):467–78.
6. Bell R, O'Neill M. Exercise and pregnancy: a review. Birth. 1994;21(2):85–95.
7. Artal R. Exercise and diabetes mellitus in pregnancy. A brief review. Sports Med. 1990;9(5):261–5.
8. Gunderson EP. Nutrition during pregnancy for the physically active woman. Clin Obstet Gynecol. 2003;46(2):390–402.
9. Ladipo OA. Nutrition in pregnancy: mineral and vitamin supplements. Am J Clin Nutr. 2000;72(1 Suppl):280S–90.
10. Forsum E, Lof M. Energy metabolism during human pregnancy. Annu Rev Nutr. 2007;27:277–92.
11. Butte NF, Wong WW, Treuth MS, Ellis KJ, O'Brian SE. Energy requirements during pregnancy based on total energy expenditure and energy deposition. Am J Clin Nutr. 2004;79(6):1078–87.
12. Clarke PE, Gross H. Women's behaviour, beliefs and information sources about physical exercise in pregnancy. Midwifery. 2004;20(2):133–41.
13. Zhang J, Savitz DA. Exercise during pregnancy among US women. Ann Epidemiol. 1996;6(1):53–9.
14. Mudd LM, Owe KM, Mottola MF, Pivarnik JM. Health benefits of physical activity during pregnancy: an international perspective. Med Sci Sports Exerc. 2013;45(2):268–77.
15. Lokey EA, Tran ZV, Wells CL, Myers BC, Tran AC. Effects of physical exercise on pregnancy outcomes: a meta-analytic review. Med Sci Sports Exerc. 1991; 23(11):1234–9.
16. Pivarnik JM. Cardiovascular responses to aerobic exercise during pregnancy and postpartum. Semin Perinatol. 1996;20(4):242–9.
17. Gorski J. Exercise during pregnancy: maternal and fetal responses. A brief review. Med Sci Sports Exerc. 1985;17(4):407–16.
18. Lotgering FK, Struijk PC, van Doorn MB, Spinnewijn WE, Wallenburg HC. Anaerobic threshold and respiratory compensation in pregnant women. J Appl Physiol. 1995;78(5):1772–7.
19. Kramer MS, McDonald SW. Aerobic exercise for women during pregnancy. Cochrane Database Syst Rev. 2006;3, CD000180.
20. Pivarnik JM, Perkins CD, Moyerbrailean T. Athletes and pregnancy. Clin Obstet Gynecol. 2003;46(2): 403–14.
21. Wolfe LA, Weissgerber TL. Clinical physiology of exercise in pregnancy: a literature review. J Obstet Gynaecol Can. 2003;25(6):473–83.
22. McAuley SE, Jensen D, McGrath MJ, Wolfe LA. Effects of human pregnancy and aerobic conditioning on alveolar gas exchange during exercise. Can J Physiol Pharmacol. 2005;83(7):625–33.
23. Garshasbi A, Faghih ZS. The effect of exercise on the intensity of low back pain in pregnant women. Int J Gynaecol Obstet. 2005;88(3):271–5.
24. Ostgaard HC, Zetherstrom G, Roos-Hansson E. Back pain in relation to pregnancy: a 6-year follow-up. Spine (Phila Pa 1976). 1997;22(24):2945–50.
25. Kristiansson P, Svardsudd K, von Schoultz B. Serum relaxin, symphyseal pain, and back pain during pregnancy. Am J Obstet Gynecol. 1996;175(5):1342–7.
26. Clapp 3rd JF. Exercise during pregnancy. A clinical update. Clin Sports Med. 2000;19(2):273–86.
27. Clapp 3rd JF. The course of labor after endurance exercise during pregnancy. Am J Obstet Gynecol. 1990;163(6 Pt 1):1799–805.
28. Rice PL, Fort IL. The relationship of maternal exercise on labor, delivery and health of the newborn. J Sports Med Phys Fitness. 1991;31(1):95–9.
29. Damm P, Breitowicz B, Hegaard H. Exercise, pregnancy, and insulin sensitivity–what is new? Appl Physiol Nutr Metab. 2007;32(3):537–40.
30. Dempsey JC, Sorensen TK, Williams MA, et al. Prospective study of gestational diabetes mellitus risk in relation to maternal recreational physical activity before and during pregnancy. Am J Epidemiol. 2004;159(7):663–70.
31. Gaillard R, Bakker R, Willemsen SP, Hofman A, Steegers EA, Jaddoe VW. Blood pressure tracking during pregnancy and the risk of gestational hypertensive disorders: the Generation R Study. Eur Heart J. 2011;32(24):3088–97.
32. Ghulmiyyah L, Sibai B. Maternal mortality from preeclampsia/eclampsia. Semin Perinatol. 2012; 36(1):56–9.
33. Saftlas AF, Logsden-Sackett N, Wang W, Woolson R, Bracken MB. Work, leisure-time physical activity, and risk of preeclampsia and gestational hypertension. Am J Epidemiol. 2004;160(8):758–65.
34. Sorensen TK, Williams MA, Lee IM, Dashow EE, Thompson ML, Luthy DA. Recreational physical activity during pregnancy and risk of preeclampsia. Hypertension. 2003;41(6):1273–80.
35. Demissie Z, Siega-Riz AM, Evenson KR, Herring AH, Dole N, Gaynes BN. Associations between physical activity and postpartum depressive symptoms. J Womens Health (Larchmt). 2011;20(7): 1025–34.
36. Wallace AM, Engstrom JL. The effects of aerobic exercise on the pregnant woman, fetus, and pregnancy outcome. A review. J Nurse Midwifery. 1987;32(5): 277–90.

37. Parker KM, Smith SA. Aquatic-aerobic exercise as a means of stress reduction during pregnancy. J Perinat Educ. 2003;12(1):6–17.
38. Beilock SL, Feltz DL, Pivarnik JM. Training patterns of athletes during pregnancy and postpartum. Res Q Exerc Sport. 2001;72(1):39–46.
39. Sady SP, Carpenter MW. Aerobic exercise during pregnancy. Special considerations. Sports Med. 1989;7(6):357–75.
40. Clapp 3rd JF, Dickstein S. Endurance exercise and pregnancy outcome. Med Sci Sports Exerc. 1984; 16(6):556–62.
41. Hartmann S, Bung P, Schlebusch H, Hollmann W. The analgesic effect of exercise during labor. Z Geburtshilfe Neonatol. 2005;209(4):144–50.
42. Lotgering FK, Gilbert RD, Longo LD. The interactions of exercise and pregnancy: a review. Am J Obstet Gynecol. 1984;149(5):560–8.
43. Clapp 3rd JF, Lopez B, Harcar-Sevcik R. Neonatal behavioral profile of the offspring of women who continued to exercise regularly throughout pregnancy. Am J Obstet Gynecol. 1999;180(1 Pt 1):91–4.
44. ACOG committee opinion. Exercise during pregnancy and the postpartum period. Number 267, January 2002. American College of Obstetricians and Gynecologists. Int J Gynaecol Obstet. 2002; 77(1):79–81.
45. ACOG Committee opinion. Number 267, January 2002: exercise during pregnancy and the postpartum period. Obstet Gynecol. 2002;99(1):171–3.
46. Vadakekut ES, McCoy SJ, Payton ME. Association of maternal hypoglycemia with low birth weight and low placental weight: a retrospective investigation. J Am Osteopath Assoc. 2011;111(3):148–52.
47. Lotgering FK, van Doorn MB, Struijk PC, Pool J, Wallenburg HC. Maximal aerobic exercise in pregnant women: heart rate, O2 consumption, CO2 production, and ventilation. J Appl Physiol. 1991;70(3):1016–23.
48. Mullinax KM, Dale E. Some considerations of exercise during pregnancy. Clin Sports Med. 1986; 5(3):559–70.
49. Borg-Stein JP, Fogelman DJ, Ackerman KE. Exercise, sports participation, and musculoskeletal disorders of pregnancy and postpartum. Semin Neurol. 2011; 31(4):413–22.
50. Parer JT, Court DJ, Block BS, Llanos AJ. Variability of basal oxygenation of the fetus–causes and associations. Eur J Obstet Gynecol Reprod Biol. 1984;18(1–2):1–9.
51. Edwards MJ. Review: hyperthermia and fever during pregnancy. Birth Defects Res A Clin Mol Teratol. 2006;76(7):507–16.
52. Power GG. Biology of temperature: the mammalian fetus. J Dev Physiol. 1989;12(6):295–304.
53. Soultanakis-Aligianni HN. Thermoregulation during exercise in pregnancy. Clin Obstet Gynecol. 2003;46(2):442–55.
54. Artal R, Sherman C. Exercise during pregnancy: safe and beneficial for most. Phys Sportsmed. 1999; 27(8):51–75.
55. Soultanakis HN, Artal R, Wiswell RA. Prolonged exercise in pregnancy: glucose homeostasis, ventilatory and cardiovascular responses. Semin Perinatol. 1996;20(4):315–27.
56. Clapp 3rd JF, Wesley M, Sleamaker RH. Thermoregulatory and metabolic responses to jogging prior to and during pregnancy. Med Sci Sports Exerc. 1987;19(2):124–30.
57. Ebbeling CB, Pearson MN, Sorensen G, et al. Conceptualization and development of a theory-based healthful eating and physical activity intervention for postpartum women who are low income. Health Promot Pract. 2007;8(1):50–9.
58. Clapp 3rd JF. The effects of maternal exercise on early pregnancy outcome. Am J Obstet Gynecol. 1989;161(6 Pt 1):1453–7.
59. Wolfe LA, Davies GA. Canadian guidelines for exercise in pregnancy. Clin Obstet Gynecol. 2003;46(2):488–95.
60. Artal R, Platt LD, Sperling M, Kammula RK, Jilek J, Nakamura RI. Maternal cardiovascular and metabolic responses in normal pregnancy. Am J Obstet Gynecol. 1981;140(2):123–7.
61. Klebanoff MA, Shiono PH, Carey JC. The effect of physical activity during pregnancy on preterm delivery and birth weight. Am J Obstet Gynecol. 1990;163(5 Pt 1):1450–6.
62. Duncombe D, Skouteris H, Wertheim EH, Kelly L, Fraser V, Paxton SJ. Vigorous exercise and birth outcomes in a sample of recreational exercisers: a prospective study across pregnancy. Aust N Z J Obstet Gynaecol. 2006;46(4):288–92.
63. Zaharieva E. Olympic participation by women. Effects on pregnancy and childbirth. JAMA. 1972;221(9):992–5.
64. Artal R, O'Toole M. Guidelines of the American College of Obstetricians and Gynecologists for exercise during pregnancy and the postpartum period. Br J Sports Med. 2003;37(1):6–12. discussion 12.
65. Davies GA, Wolfe LA, Mottola MF, MacKinnon C. Joint SOGC/CSEP clinical practice guideline: exercise in pregnancy and the postpartum period. Can J Appl Physiol. 2003;28(3):330–41.
66. Fazlani SA. Protocols for exercise during pregnancy. J Pak Med Assoc. 2004;54(4):226–9.
67. Paisley TS, Joy EA, Price Jr RJ. Exercise during pregnancy: a practical approach. Curr Sports Med Rep. 2003;2(6):325–30.
68. Snyder S, Pendergraph B. Exercise during pregnancy: what do we really know? Am Fam Physician. 2004;69(5):1053. 1056.
69. Davies B, Bailey DM, Budgett R, Sanderson DC, Griffin D. Intensive training during a twin pregnancy. A case report. Int J Sports Med. 1999;20(6):415–8.
70. Evenson KR, Herring AH, Wen F. Self-Reported and objectively measured physical activity among a cohort of postpartum women: the PIN Postpartum Study. J Phys Act Health. 2012;9(1):5–20.
71. Choi J, Fukuoka Y, Lee JH. The effects of physical activity and physical activity plus diet interventions

on body weight in overweight or obese women who are pregnant or in postpartum: a systematic review and meta-analysis of randomized controlled trials. Prev Med. 2013;56:351–64.
72. Daley AJ, Macarthur C, Winter H. The role of exercise in treating postpartum depression: a review of the literature. J Midwifery Womens Health. 2007;52(1): 56–62.
73. Lederman SA. Pregnancy. In: Heymsfield SBL, Lohman T, Wang ZM, Going S, editors. Human body composition, vol. I. 2nd ed. Human Kinetics: Champaign; 2007. p. 299–312.

# Part V

# Nutrition, Energy Balance, and Weight Control

# Estimating Energy Requirements

# 27

Jacalyn J. Robert-McComb, Elvis Álvarez Carnero, and Eduardo Iglesias-Gutiérrez

**Abstract**

Women energy requirements need be balance with total daily energy expenditure (TDEE). Hence, meeting of energy needs for all components of TDEE must be the most important concern for active females. Resting metabolic rate (RMR), thermic effect of food (TEF), and physical activity energy expenditure (PAEE) are the most important components of TDEE; where the most variable is the PAEE. The quantification of each component implies time consuming and expensive techniques, which are unviable in field and clinical settings. However, indirect methods have been developed to allow coaches, physicians, and nutritionists estimate TDEE. In this chapter, we cover the physiological relevance of RMR, TEF, and PAEE, and the indirect ways to obtain estimations of their values. On the other hand, other variables factors affecting TDEE as adaptive thermogenesis and non-exercise activity thermogenesis (NEAT) are highlighted. Finally, a case study is suggested in order to introduce basic metabolic calculations and physical activity level (PAL) concept.

**Keywords**

Energy • MET • Calorie • Basal metabolic rate • Resting metabolic rate

## 27.1 Learning Objectives

After completing this chapter, you should have an understanding of:

1. The components of total energy expenditure;
2. The difference between the terms: resting energy expenditure, resting metabolic rate basal energy expenditure, and basal metabolic rate;
3. Adaptive thermogenesis and non-exercise activity thermogenesis; and
4. Practical methods of estimating total energy expenditure.

J.J. Robert-McComb, Ph.D., F.A.C.S.M., A.C.S.M., P.D. (✉)
Department of Health, Exercise, and Sports, Texas Tech University, Lubbock, TX 79409, USA
e-mail: jacalyn.mccomb@ttu.edu

E.Á. Carnero, Ph.D.
Biodynamic and Body Composition Laboratory, University of Malaga, Málaga, Spain

E. Iglesias-Gutiérrez, Ph.D., M.Sc.
Department of Functional Biology (Physiology), University of Oviedo, Oviedo, Asturias, Spain

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_27, 

## 27.2 Introduction

Energy is defined as *the capacity to do work*. Energy requirements are based on the energy needs for optimal growth and development for each individual at their stage in life in order to maximize long-term good health. Energy requirements for humans are not the same as nutritional requirements (nutritional requirements are discussed in Chaps. 28–29 and 31–32 of this book). Even though we do discuss the difference between nutritional requirements and energy requirements briefly, the focus of this chapter is on the estimation of energy requirements. The Food and Agricultural Organization of the United Nation's (FAO) report, *FAO Food and Nutrition Technical Report Series 1* define energy requirements as:

> the amount of food energy needed to balance energy expenditure in order to maintain body size, body composition and a level of necessary and desirable physical activity consistent with long-term good health. This includes the energy needed for the optimal growth and development of children, for the deposition of tissues during pregnancy, and for the secretion of milk during lactation consistent with the good health of mother and child [1].

## 27.3 Research Findings and Nutritional Terminology

It can be confusing for the novice reader when terms are readily exchanged from one source to another when discussing energy. The terms *Calorie*, *calorie*, *and kcal* are often used when discussing energy. Other interchangeably used terms when discussing TEE are *basal metabolic rate* (BMR) rather than BEE, and *resting metabolic rate* (RMR) rather than REE. In the next section that follows, we will clarify some of the terms used when discussing energy requirements.

### 27.3.1 Terms Used When Discussing Total Energy Expenditure (TEE)

#### 27.3.1.1 Calorie (Capital C or Uppercase C), Calories (Lowercase c), and Kilocalorie

The difference in expressing heat energy with an uppercase *C*, *Calorie*, or a lowercase c, *calorie*, can be a source of confusion. The calorie (lowercase c) is defined as the amount of energy required to raise the temperature of 1 ml or 1 g of water at 15 °C by 1 °C, or from 14.5 to 15.5 °C. In the context of foods and nutrition, "large calorie" (i.e., Calorie) with an uppercase C has been used traditionally [2]. When an uppercase C is used to express "Calorie" it is 1,000 cal and is referred to as a kilocalorie (abbreviated as kcal). A Calorie or kcal expresses the quantity of heat needed to raise the temperature of 1 kg (1 L) of water 1 °C (specifically from 14.5 to 15.5 °C). The term kcal is used in the context of food and nutrition because the amount of energy involved in metabolism of food is fairly large.

#### 27.3.1.2 Basal Metabolic Rate (BMR)

Most simply stated, the BMR describes the rate of energy expenditure that occurs in the postabsorptive state (after an overnight fast with no food consumption for 12–14 h), resting comfortably, supine, awake, and motionless in a thermoneutral environment. In this state, food and physical activity have minimal influence on metabolism. The BMR thus reflects the energy needed to sustain the metabolic activities of cells and tissues, plus the energy needed to maintain blood circulation, respiration, and gastrointestinal and renal processing (i.e., the basal cost of living). BMR thus includes the energy cost associated with remaining awake (the cost of arousal). The sleeping metabolic rate (SMR) during the morning is 5–10 % lower than BMR during the morning hours [3]. The verbatim definition for

BMR that appears in the *FAO Food and Nutrition Technical Report Series 1* is as follows:

> The amount of energy used for basal metabolism in a period of time is called the basal metabolic rate (BMR), and is measured under standard conditions that include being awake in the supine position after ten to 12 hrs of fasting and eight hrs of physical rest, and being in a state of mental relaxation in an ambient environmental temperature that does not elicit heat-generating or heat-dissipating processes [1].

From a physiological point of view, the concept of BMR is:

> … the amount of energy in basal (humans) or standard (animals) state, when no work is done and all energy is dissipated ….
>
> the steady-state rate of heat production by a whole organism under a set of "standard" conditions … these conditions are that the individual is an adult and is awake but resting, stress free, not digesting food, and maintained at a temperature that elicits no thermoregulatory effect on heat production … BMR is measured either as heat production, or indirectly as oxygen consumption from which it can be accurately predicted [4].

#### 27.3.1.3 Basal Energy Expenditure (BEE)

The BMR is commonly extrapolated to 24 h to be more meaningful, and it is then referred to as basal energy expenditure (BEE), expressed as kcal/24 h. It is most simply defined as the minimal amount of energy in kcal that is compatible with life over a 24 h period. All of the conditions associated with BMR (no food consumption for 12–14 h, resting comfortably, supine, awake, and motionless in a thermoneutral environment) must be met for BEE [2].

#### 27.3.1.4 Resting Metabolic Rate (RMR)

RMR has traditionally been proposed to be a surrogate of BMR. If any of the conditions for BMR have not been met (early morning assessment following a 12–14 h fast, no physical exercise following awakening, remaining awake but motionless, supine, comfortable and in a thermoneutral environment) then energy expenditure is referred to as RMR. Resting metabolic rate energy expenditure under resting conditions tends to be somewhat higher (10–20 %) than under basal conditions due to increases in energy expenditure caused by recent food intake (i.e., by the "thermic effect of food") or by the delayed effect of recently completed physical activity [2].

#### 27.3.1.5 Resting Energy Expenditure (REE)

When RMR is extrapolated to 24 h, then it is then referred to as resting energy expenditure (REE), expressed as kcal/24 h. It is most simply defined as resting energy expenditure expressed in kcal over a 24 h period [5]. This term is used when all of the standard conditions required for BMR have not been met.

#### 27.3.1.6 Thermic Effect of Food

The thermic effect of food (TEF) is the energy we spend to digest, absorb, distribute and store the nutrients ingested [2]. The type of food that you eat will have an effect on TEF, for example, spicy foods increase TEF.

#### 27.3.1.7 Energy Balance

Daily energy balance fluctuates considerably, yet over the long term, energy balance is very precise. Positive and negative energy balances result in weight gain and weight loss, accordingly, mainly in the form of fat. The amount of fat stored in an adult of normal weight commonly ranges from 13.2 to 44 lb (6–20 kg). Body fat energy reserves range from approximately 50,000–200,000 kcal since 1 g of fat provides 9.4 kcal. This vast store of energy reserves provides a large buffer capacity as well as the ability to provide energy to survive for several months of severe food deprivation. Large deviations of energy balance, both positive and negative, occur daily by several hundred kcal/day in both normal and overweight subjects. Yet over the long term, energy balance is maintained implying that the cumulative error in adjusting energy intake to expenditure amounts to less than 2 % of energy expenditure [2].

#### 27.3.1.8 Estimated Energy Requirement

According to the Dietary Reference Intakes [2], the Estimated Energy Requirement (EER) is

defined as "the average dietary energy intake that is predicted to maintain energy balance in a healthy, adult of a defined age, gender, weight, height, and level of physical activity consistent with good health."

#### 27.3.1.9 Metabolic Equivalent

The Metabolic Equivalent of Task (MET), or simply metabolic equivalent, is a physiological measure expressing the energy cost of physical activities and is defined as the ratio of metabolic rate (and therefore the rate of energy consumption) during a specific physical activity to a reference metabolic rate, set by convention to 3.5 ml $O_2 kg^{-1}$ $min^{-1}$ or equivalently, 1 MET = 1 kcal $kg^{-1}$ $h^{-1}$ [6]. MET is used as a means of expressing the intensity and energy expenditure of activities in a way comparable among persons of different weight. If someone is working at 10 METs, it is implied that they are working ten times above their resting rate.

#### 27.3.1.10 Compendium of Physical Activities

The *Compendium of Physical Activities* was developed for use in epidemiologic studies to standardize the assignment of MET intensities in physical activity questionnaires. Compendium activities are classified by a 5-digit code that identifies the category (heading) as the first two digits and type (description) of activity as the last three digits. Metabolic equivalents are listed for each activity. The calculation from METs to calories is very easy since 1 MET = 1 kcal $kg^{-1}$ $h^{-1}$. For example if an activity has a MET value of 7 METs then 7 METs = 7 kcal $kg^{-1}$ $h^{-1}$. The compendium has been used in studies worldwide to assign intensity units to physical activity questionnaires and to develop innovative ways to assess energy expenditure in physical activity studies. The compendium was published in 1993 and updated in 2000 and 2011 [6]. Appendix 1 has the updated version published in 2011: You can also find the compendium and modified versions of the compendium at https://sites.google.com/site/compendiumofphysicalactivities/[7].

### 27.3.2 Energy Requirements Versus Nutrient Requirements

Recommendations for nutrient intakes are generally set to provide an ample supply of the various nutrients needed for all healthy individuals in a given life stage and gender group. Recommended intakes are thus set to correspond to the median amounts sufficient to meet a specific criterion of adequacy plus two standard deviations to meet the needs of nearly all healthy individuals.

However, this is not the case with energy. Excess energy cannot be eliminated, and this energy is eventually deposited in the form of body fat. This reserve provides a means to maintain metabolism during periods of limited food intake, but it can also result in obesity.

An excellent reference text entitled, Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients) [2] details the nutrient and energy needs of individuals at all stages of life. This project was funded in part by the United States Department of Health and Human Services Office of Disease Prevention and Health Promotion. A free PDF copy can be downloaded at http://www.nap.edu/catalog.php?record_id=10490.

### 27.3.3 Energy Requirements

Energy requirements are directly related to the components of energy expenditure. In 1985, The World Health Organization stated that "as a matter of principle, we believe the estimates of energy requirements should, as far as possible, be based on estimates of energy expenditure." [8] Several modifications are proposed in the updated *FAO Food and Nutrition Technical Report Series 1* published in 2004 from the former report published in 1985 [1, 8]. The 2004 report is a result of an expert consultation held in Rome, October 17–24 in 2001. Representing agencies included The United Nations University, World Health Organization and The Food and Agriculture Organization of the United Nations. This report can be downloaded free of charge at http://www.fao.org/docrep/007/y5686e/y5686e00.htm.

### 27.3.4 The Components of Energy Expenditure

In the human body, TEE is the sum of: (1) BEE, which includes a small component associated with arousal, as compared to sleeping; (2) TEF which is the energy we spend to digest, absorb, distribute and store the nutrients ingested; (3) EEPA which is the energy expended in physical activity; and (4) the energy expended in depositing new tissues and in producing milk. Figure 27.1 depicts the components of TEE using the doubly labeled water technique method. Total energy expenditure from doubly labeled water does not include the energy content of tissue development during normal growth and pregnancy or the milk produced during lactation,.

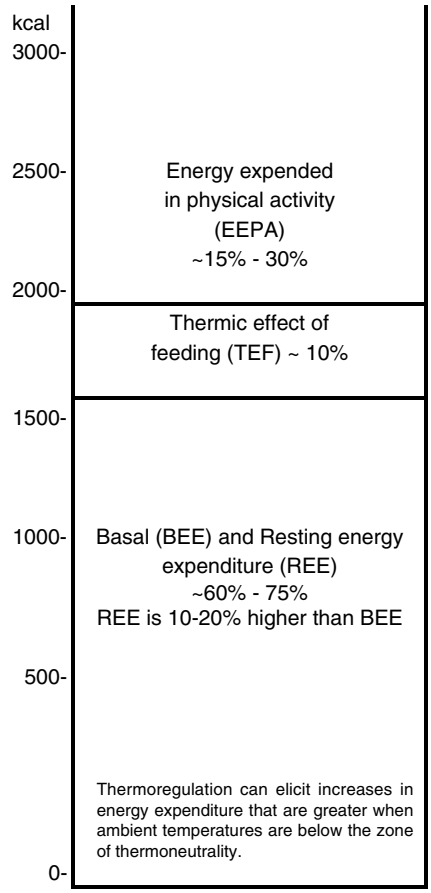



**Fig. 27.1** Components of total energy expenditure from the doubly labeled water method (DLW). *Note*: TEE from DWL does not include the energy content of the maturing tissue constituents during normal growth and pregnancy or the milk produced during lactation

Some sources may state that TEE is composed of: (1) resting energy expenditure (REE); (2) TEF; and (3) EEPA [5]. If all of the conditions required for BEE have not been met then the term REE is used.

Regardless of the term used, the resting or basal component of energy expenditure constitutes the largest portion (60–75 %) of the TEE [9]. The only exception to this would be in extremely active individuals. The TEF represents approximately 10 % of the total daily energy expenditure, although it depends on the macronutrient content of the food ingested. The most variable component of TEE is the contribution of physical activity (EEPA) which varies from approximately 15–30 % depending on the activity level of the individual [2].

There are many factors that affect the components that make up TEE (BEE:TEF:EEPA) whether or not that refers to BEE, REE, BMR, or RMR. From a practical point of view, RMR is usually used in equations to estimate TEE, therefore our discussion will center on the factors that affect REE (RMR extrapolated to a 24 h period) or RMR rather than BEE or BMR. We will also discuss the factors that affect TEF and EEPA.

#### 27.3.4.1 Factors Affecting Resting Energy Expenditure

Numerous factors cause the REE to vary among individuals. Interestingly, three factors, age, sex and fat free mass (FFM), account for about 80 % of the variability in REE [10]. Additional contributing variables include: (1) body size and weight [body surface area (BSA)]; (2) hormonal status [2, 5];(3) age; (4) sex; and (5) fat mass. Age, sex, and FFM are highly correlated and these three factors combined together account for about 80 % of the variability in REE [10].

Body surface area is computed from height and weight, taller individuals who weigh more will have the greatest surface area. Individuals with greater surface area will have the greatest metabolic rate. Various body surface formulas have been developed over the years. There is debate about which is the best formula to use since there is

**Table 27.1** Equations to estimate body surface area

| Name of formula | Formula |
|---|---|
| Boyd | |
| Haycock | BSA ($m^2$) = 0.024265 × Height (cm)$^{0.3964}$ × Weight (kg)$^{0.5378}$ |
| DuBois and DuBois | BSA ($m^2$) = 0.20247 × Height (m)$^{0.725}$ × Weight (kg)$^{0.425}$ |
| Gehan and George | BSA ($m^2$) = 0.0235 × Height (cm)$^{0.42246}$ × Weight (kg)$^{0.51456}$ |
| Mosteller | BSA ($m^2$) = ([Height (cm) × Weight (kg)]/3,600)$^{½}$ |
| | or in inches and pounds: BSA ($m^2$) = ([Height (in.) × Weight (lb.)]/3,131)$^{½}$ |

Adapted from Body Surface Area Calculator for medication doses at http://www.halls.md/body-surface-area/bsa.htm

no standardization of formulas at this time. The Mosteller formula is gaining support as a common standard because it is much simpler and can be easily calculated with a handheld calculator. A Web site that can be used to estimate BSA is http://www.halls.md/body-surface-area/bsa.htm. Table 27.1 lists formulas to determine BSA.

As stated previously, one of the main determinants of REE is fat-free mass (FFM) or lean body mass (LBM). Because of their greater FFM, athletes or individuals who are extremely fit have approximately a 5 % higher BMR than nonathletic individuals. The effect of age has on REE is highly correlated with FFM. Resting energy expenditure is highest during periods of rapid growth, chiefly during the first and second years of life, and peaks throughout adolescence and puberty [11]. As a child becomes older, the caloric requirement for growth is reduced to about 1 % of the total energy expended. Resting energy expenditure continues to decline with increasing age in adulthood. The loss of FFM with aging can be attenuated with exercise; however, exercise cannot completely negate the effects of age. There is approximately a 2–3 % decline in REE after early adulthood largely due to loss of FFM [11]. Some authors also account for the reduction in brain weight with age on REE or basal metabolism, since it is an extremely metabolically active organ. In fact, the brain is more metabolically active than muscle tissue during rest [12]. Henry [12] also states that "the fall in BMR with ageing may be less dramatic than previously perceived. Indeed, some subjects may show an increase in BMR with ageing."

Sex differences in metabolic rates are primarily contributed to differences in body size and composition. Women have approximately 5–10 % lower REE than men primarily due to differences in LBM [5].

Hormonal status also has an effect on the metabolic rate. The hormones associated with the sympathetic nervous system or those involved in the fight or flight response such as epinephrine and norepinephrine increase metabolic rate. Probably the hormones most closely aligned with REE are the thyroid hormones since these hormones are considered to be the permissive hormones and allow other hormones to exert their full effect. Also the metabolic rate of women fluctuates with the menstrual cycle. An average of 359 kcal/day difference in the BMR has been measured from 1 week before ovulation and just before the onset of menstruation. The average increase in energy expenditure is about 150 kcal/day during the second half of the menstrual cycle [13].

#### 27.3.4.2 Factors Affecting the Thermic Effect of Food

The TEF accounts for approximately 10 % of the TEE. The TEF varies with the composition of the diet and is greater after the consumption of and proteins than after carbohydrates and fat. Spicy foods enhance and prolong the effect of TEF. Caffeine and nicotine also stimulate the TEF [5].

#### 27.3.4.3 Factors Affecting the Energy Expended in Physical Activity and Exercise

Before we begin our discussion of energy during physical activity and exercise, we need to define and differentiate the terms physical activity and exercise. According to Caspersen, Powell, and Christenson [14]:

> Physical activity is defined as any bodily movement produced by skeletal muscles that results in energy expenditure. The energy expenditure can be measured in kilocalories. Physical activity in daily

life can be categorized into occupational, sports, conditioning, household, or other activities. Exercise is a subset of physical activity that is planned, structured, and repetitive and has as a final or an intermediate objective the improvement or maintenance of physical fitness.

In this chapter, when we speak of physical activity, we include the subset exercise. The energy expended in physical activity (EEPA) is the most variable component of TEE. To illustrate this point think of this, the basal oxygen ($O_2$) consumption rate of adults is approximately 250 mL/min while elite athletes, such as marathon runners, can sustain $O_2$ consumption rates of 5,000 mL/min [2]. So you can see quite easily that the scale of metabolic responses to exercise varies over a 20-fold range. The EEPA not only includes the energy cost of the movement but also includes energy during these activities such as shivering and maintaining postural control [2, 5].

Energy cost of physical activity is related to intensity, duration, skill level, and FFM. As the intensity of the physical activity and or duration of the activity increases, so does the energy expenditure. All else being equal, individuals with less skill in performing an activity will expend more energy in performing the motion. Also, individuals with greater FFM will expend more energy at the same intensity and or duration of the exercise. In order to estimate the energy cost associated with activity or exercise, we will refer to the Compendium of Physical Activity [7] that can be found in Appendix 1. The MET value for each activity is listed. These values can then be converted to kcal by using the formula 1 MET = 1 kcal $kg^{-1}$ $h^{-1}$. We also have a more generalized version of activity categories in Table 27.2 for the sake of simplicity.

**Table 27.2** MET values for physical activity levels

| Physical activity intensity | MET |
|---|---|
| *Light intensity activities* | <3 |
| Sleeping | 0.9 |
| Watching television | 1.0 |
| Writing, desk work, typing | 1.8 |
| Walking, 1.7 mph (2.7 km/h), level ground, strolling, very slow | 2.3 |
| Walking, 2.5 mph (4 km/h) | 2.9 |
| *Moderate intensity activities* | 3–6 |
| Bicycling, stationary, 50 W, very light effort | 3.0 |
| Walking 3.0 mph (4.8 km/h) | 3.3 |
| Calisthenics, home exercise, light or moderate effort, general | 3.5 |
| Walking 3.4 mph (5.5 km/h) | 3.6 |
| Bicycling, <10 mph (16 km/h), leisure, to work or for pleasure | 4.0 |
| Bicycling, stationary, 100 W, light effort | 5.5 |
| *Vigorous intensity activities* | >6 |
| Jogging, general | 7.0 |
| Calisthenics (e.g., push-ups, sit-ups, pull-ups, jumping jacks), heavy, vigorous effort | 8.0 |
| Running jogging, in place | 8.0 |
| Rope jumping | 10.0 |

From compendium of Physical Activity found at https://sites.google.com/site/compendiumofphysicalactivities/home. Project supported by University of Arizona and the National Cancer Institute

## 27.4 Contemporary Understanding of the Issues

### 27.4.1 Methods of Measuring Energy Expenditure

The Doubly Labeled Water Technique (DLW) is currently considered the most accurate technique for measuring TEE in free-living individuals. The Doubly Labeled Water Technique (DLW) for measuring TEE in free-living people uses two stable isotopes of water (deuterium [$^2H_2O$] and oxygen-18 [$H_2{}^{18}O$]); the difference in the turnover rates of the two isotopes measures the carbon dioxide production rate, from which total energy expenditure can be calculated [15]. The premise of the method is that the $O_2$ atoms in expired $CO_2$ have isotopically equilibrated atoms. Thus, after a loading dose of water labeled with 2H and 18O, the 2H is eliminated from the body as water, whereas the 18O is eliminated from the body as water and $CO_2$. The difference between the elimination rates is therefore proportional to $CO_2$ production and hence energy expenditure can be estimated using calculated $O_2$ from the equation of respiratory quotient ($RQ = VCO_2/VO_2$); RQ is estimated from respiratory quotient of diet or assumed to be 0.85 (Western diet).

The primary advantage of this technique is its accuracy (2–8 % precision) and that it provides a measure of energy expenditure that incorporates all the components of TEE [5, 15]. Also it can be used to measure free-living energy expenditure, while subjects engage in normal daily activities. It is for these reasons, that the DLW technique has been used for validation studies and to generate data to develop prediction equations [2]. Normally, estimates of EEPA using different techniques are validated against the DLW technique. However, the expense of the technique makes it impractical for routine use by clinicians.

Direct calorimetry is a method for measuring the amount of energy expended by monitoring the rate at which a person loses heat from the body using a structure called a whole-room calorimeter. Direct calorimetry provides a measure of energy expended in the form of heat but does not provide information on the type of fuel being oxidized.

Indirect calorimetry is a method of estimating energy expenditure by measuring oxygen consumption and carbon dioxide utilizing a respirator gas-exchange canopy or ventilation hood. The amount of heat produced by oxidation of a nutrient is proportional to the consumed $O_2$ and the $CO_2$ produced. The energy per liter of oxygen will be converted to kilocalories of heat produced and extrapolated to energy expenditure in 24 h. Data obtained from indirect calorimetry permit the calculation of the respiratory quotient (RQ), which is calculated as the ratio moles $CO_2$ expired/moles $O_2$ consumed. The volume of $O_2$ used and $CO_2$ produced and the amount of heat released in the oxidative process, depend on the type of nutrient being oxidized. This allows determine the proportion of each substrate being used, ranging normally from 1 (only carbohydrates) to 0.7. It is assumed that all the $O_2$ consumed is used to oxidize substrates, that all the $CO_2$ produced can be recovered, and that the proteins are not involved primarily in energy production.

Accelerometers detect body displacement electronically, using piezo-resistive or piezoelectric sensors, with varying degrees of sensitivity; the triaxial monitor uses three different planes to measure movement rather than a single vertical plane, as in the uniaxial monitor, and has been found to be more accurate than the uniaxial monitor [16]. Portable uniaxial accelerometer units have been widely used to detect physical activity, but these instruments are not sufficiently sensitive to quantify the physical activity of a given free-living subject, although they are valuable for comparing activity levels between groups of subjects [17–19].

Minute-by-minute heart rate monitors have been found to be valid in estimating habitual TEE in certain populations but not in individuals, at least in the absence of exercise [11]. Variance in other factors that also affect heart rate, such as emotion, also impact this relationship. According to Levine [19]:

> In humans, there is a significant relationship between heart rate and energy expenditure, at least in the absence of exercise. The conceptual limitation is that energy expenditure and heart rate are not linearly related for an individual in part because cardiac stroke volume changes with changing heart rate and even posture.

Many questionnaires have been developed to measure physical activity in adults. Currently, the most widely used are the different versions of the International Physical Activity Questionnaire (IPAQ). Van Poppel et al. [20] reviewed the validity, reliability and responsiveness of 85 PA (physical activity) questionnaires, including the IPAQ. In light of their results, these authors conclude that, no questionnaire or type of questionnaire for assessing PA was superior and therefore could not be strongly recommended above others. Furthermore, they observed that there is a clear lack of standardization of PA questionnaires, resulting in many variations not well described and validated. Thus, researchers should decide which questionnaire best fit their purposes considering the content of the questionnaire, the nature of the sample and the available validation studies [20, 21].

More recently, Bonn et al. [22] developed the Web-based questionnaire, Active-Q. The authors validate their questionnaire against DLW and conclude that Active-Q is a valid method for estimating total energy expenditure, and is also reproducible and user-friendly method. However, many methodological limitations make this conclusion perhaps too audacious.

### 27.4.2 Estimated Energy Expenditure Prediction Equations

The Harris–Benedict formula published in 1919 [23] is one of the most widely used formulas to determine RMR. Results of the accuracy of the prediction equation are mixed. Daly et al. [24] suggested that it overestimates RMR by 7–24 % in many contemporary populations. However, Hasson et al. [25] compared predicted RMR derived from commonly used prediction regression equations to measured RMR in a diverse group of individuals and found that the Harris–Benedict equation was the most likely to predict RMR to within 10 % of measured RMR. In addition, the Harris–Benedict equation accurately predicted RMR in both sexes, all body mass index (BMI) categories, individuals aged 30–60 years and all racial/ethnic groups. Table 27.3 includes the original Harris–Benedict prediction equation published in 1919 and a 1984 Harris–Benedict equations revised by Roza and Shizgal [26].

The 1985 predictive equations included in the Technical Report Series 724 [8] were developed from a meta-analysis of about 100 studies conducted over a long time period (1914–1980), including the subjects studied by Harris and Benedict. The current predictive equations published in 2004 can be found in the *FAO Food and Nutrition Technical Report Series* [1]. The full report can be downloaded free of charge at the World Health Organization Web site (seehttp://www.who.int/nutrition/publications/nutrientrequirements/9251052123/en/index.html). However, Henry et al. [27] found that the equations published in the *FAO Food and Nutrition Technical Report Series* overestimated REE.

Frankenfield et al. [28] examined the validity of RMR prediction equations applied to the general public and concluded that the Mifflin–St Jeor equation is the most likely to estimate RMR within 10 % of that measured: However, noteworthy limitations exist when it is generalized to certain age and ethnic groups. This equation is presented in Table 27.4 [29].

Hasson et al. [25] highlighted that one limitation of the Frankenfield et al. [28] systematic review of predictive equations for RMR was the absence of a direct comparison of these regression equations to a criterion measure. Results from Hasson's study [25] suggest the Mifflin–St Jeor equation is primarily useful in overweight/obese groups, whereas the equations in the *FAO Food and Nutrition Technical Report Series* 1 may be optimal in younger adults aged 18–49 years.

A considerable number of prediction equations have been developed since 1990. Most of the equations have not been adequately validated and the equations have a poor predictive value for individuals. The basis of several prediction methods is an estimation of REE to which is added a "stress" or "injury" factor [27]. The main advantage of these "newly" developed prediction

**Table 27.3** Harris–Benedict prediction equations

| | |
|---|---|
| *Step 1*: *calculating the BMR* | |
| The original Harris–Benedict equations published in 1918 and 1919 | |
| Men | BMR = 66.4730 + (13.7516 × weight in kg) + (5.0033 × height in cm) − (6.7550 × age in years) |
| Women | BMR = 655.0955 + (9.5634 × weight in kg) + (1.8496 × height in cm) − (4.6756 × age in years) |
| The Harris–Benedict equations revised by Roza and Shizgal in 1984 | |
| Men | BMR = 88.362 + (13.397 × weight in kg) + (4.799 × height in cm) − (5.677 × age in years) |
| Women | BMR = 447.593 + (9.247 × weight in kg) + (3.098 × height in cm) − (4.330 × age in years) |
| *Step 2*: *applying the Harris–Benedict Principle* | |
| The following table enables calculation of an individual's recommended daily calorie intake to maintain current weight | |
| Little to no exercise | Daily calories needed = BMR × 1.2 |
| Light exercise (1–3 days/week) | Daily calories needed = BMR × 1.375 |
| Moderate exercise (3–5 days/week) | Daily calories needed = BMR × 1.55 |
| Heavy exercise (6–7 days/week) | Daily calories needed = BMR × 1.725 |
| Very heavy exercise (twice per day, extra heavy workouts) | Daily calories needed = BMR × 1.9 |

From Wikipedia at http://en.wikipedia.org/wiki/Harris-Benedict_equation

**Table 27.4** An estimated energy expenditure prediction equation using the Mifflin–St Jeor equation to determine resting metabolic rate

| *Step 1*: *Estimate resting metabolic rate* (*RMR*) *using the Mifflin–St Jeor equation* | |
|---|---|
| RMR = 9.99 × weight (kg) + 6.25 × height (cm) − 4.92 × age (year) + 166 × sex (males, 1; females, 0) − 161. | |
| *Step 2*: *Determine additional caloric requirements based on level of activity* | |
| Physical activity level | Percentage above resting level |
| Bed rest | 10 |
| Quiet rest | 30 |
| Light activity | 40–60 |
| Moderate activity | 60–80 |
| Heavy activity | 100 |
| Additional caloric requirements = RMR × Percentage above resting level | |
| *Step 3*: *Determine predicted total energy expenditure* (*TEE*) | |
| TEE = RMR + Additional caloric requirements based on activity | |

Adapted from Physiology of Fitness (3rd ed.) (p. 359) by B. J. Sharkey, 1990, Champaign, IL: Human Kinetics

equations to estimate energy expenditure (EE) is that they are easy to use and inexpensive [30].

The Standing Committee on the Scientific Evaluation of Dietary Reference Intakes of the Food and Nutrition Board, Institute of Medicine, and the National Academies, in collaboration with Health Canada developed prediction equations to estimate energy requirements (EER) for people according to their life-stage group [2]. The EER incorporates age, weight, height, gender, and level of physical activity for individuals in various life-stages. The equations for girls and women published in the 2005 Dietary Reference Intakes for Energy Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids can be found in Table 27.5. Equations for both genders can be downloaded free of charge on line at The National Academies Press at http://www.nap.edu/catalog.php?record_id=10490.

## 27.5 Future Directions

Recommendations for energy expenditure used by exercise physiologists incorporate a thermic constant in exercise physiology. One of the most commonly used thermic constants of exercise physiology is the MET [6]. TEE can be estimated after quantifying daily common activities (sleeping, home activities, etc.) and multiplying by a specific MET-activity value (see an example in the case study at end of the chapter). MET-activity values can also be found in Table 27.2 and Appendix 1. You can also find the compendium and modified versions of the compendium at https://sites.google.com/site/compendiumofphysicalactivities/. A noteworthy comment about the compendium taken directly from their Web site is as follows:

> When using the Compendium to estimate the energy cost of activities, investigators should remind participants to recall only the time spent in movement. The Compendium was not developed to determine the precise energy cost of physical activity within individuals, but rather to provide a classification system that standardizes the MET intensities of physical activities used in survey research. The values in the Compendium do not estimate the energy cost of physical activity in individuals in ways that account for differences in body mass, adiposity, age, sex, efficiency of movement, geographic and environmental conditions in which the activities are performed. Thus, individual differences in energy expenditure for the same activity can be large and the true energy cost for an individual may or may not be close to the stated mean MET level as presented in the Compendium [7].

When estimating energy requirements using the compendium, individuals must take into account factors that affect the RMR as well as a concept called adaptive thermogenesis. In the information that ensues, these factors are discussed as well as a relatively new concept called the non-exercise activity thermogenesis. Formulas and examples of how to estimate energy using the compendium are also presented.

### 27.5.1 *Resting Metabolic Rate and Adaptive Thermogenesis*

RMR represents at least 60 % of TEE [31]. Measurements of RMR have been performed extensively on animals and humans using traditional calorimeters [32]. Several definitions of RMR have been reported in animals [4] and

**Table 27.5** Physical activity level index (PAL) and physical activity coefficient (PA) used to derive estimated energy requirements (EER) for women

| | Sedentary | Low active | Active | Very active |
|---|---|---|---|---|
| PAL | (1.0–1.39) | (1.4–1.59) | (1.6–1.89) | (1.9–2.5) |
| | Typical daily living activities (e.g., household tasks, walking to the bus) | Typical daily living activities PLUS 30–60 min of daily moderate activities (e.g., walking at 5–7 km/h) | Typical daily living activities PLUS at least 60 min of daily moderate activities | Typical daily living activities PLUS at least 60 min of daily moderate activities an additional 60 min of vigorous activity or 120 min of moderate activity |
| PA | PA (level 1) | PA (level 2) | PA (level 3) | PA (level 4) |
| Girls 3–18 year | 1.00 | 1.16 | 1.31 | 1.56 |
| Women 19 year+ | 1.00 | 1.12 | 1.27 | 1.45 |
| *Equations to estimate energy requirement using the PA* | | | | |
| **Infants and young children** | | | | |
| Estimated energy requirement (kcal/day) = Total energy expenditure + Energy deposition | | | | |
| 0–3 months | EER = (89 × weight [kg] − 100) + 175 | | | |
| 4–6 months | EER = (89 × weight [kg] − 100) + 56 | | | |
| 7–12 months | EER = (89 × weight [kg] − 100) + 22 | | | |
| 13–35 months | EER = (89 × weight [kg] − 100) + 20 | | | |
| **Children and adolescents 3–18 years** | | | | |
| Estimated energy requirement (kcal/day) = Total energy expenditure + Energy deposition | | | | |
| **Girls** | | | | |
| 3–8 years | | | | |
| EER = 135.3 − (30.8 × age [year]) + PA × [(10.0 × weight [kg]) + (934 × height [m])] + 20 | | | | |
| 9–18 years | | | | |
| EER = 135.3 − (30.8 × age [year]) + PA × [(10.0 × weight [kg]) + (934 × height [m])] + 25 | | | | |
| **Adults 19 years and older** | | | | |
| Estimated energy requirement (kcal/day) = Total energy expenditure | | | | |
| ***Women*** | | | | |
| EER = 354 − (6.91 × age [year]) + PA × [(9.36 × weight [kg]) + (726 × height [m])] | | | | |
| **Pregnancy** | | | | |
| Estimated Energy Requirement (kcal/day) = Nonpregnant EER + Pregnancy Energy Deposition | | | | |
| First trimester | EER = Nonpregnant EER + 0 | | | |
| Second trimester | EER = Nonpregnant EER + 340 | | | |
| Third trimester | EER = Nonpregnant EER + 452 | | | |
| ***Lactation*** | | | | |
| Estimated energy requirement (kcal/day) = Nonpregnant EER + Milk energy output − Weight loss | | | | |
| 0–6 months postpartum | EER = Nonpregnant EER + 500 − 170 | | | |
| 7–12 months postpartum | EER = Nonpregnant EER + 400 − 0 | | | |

*Note*: These equations provide an estimate of energy requirement. Relative body weight (i.e., loss, stable, gain) is the preferred indicator of energy adequacy

Adapted from A Report of the Panel on Macronutrients, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for energy carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (*macronutrients*). Washington DC: National Academy Press; 2005

Complete report can be viewed and downloaded at http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes/dri-reports

A Report of the Panel on Macronutrients, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for energy carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (**macronutrients**). Washington DC: National Academy Press; 2005

PAL = Physical activity level or physical activity index

PA = Physical activity coefficient

EER = Estimated energy requirement

**Table 27.6** Equations to estimate resting metabolic rate (RMR) in women using body composition variables as paradigm

| Author | Equation (kcal/day) |
|---|---|
| Schofield [34][a] | *<3 years*: RMR = 16.252 W + 1,023.2 H − 413.5 |
| | *3–10 years*: RMR = 16.969 W + 161.8 H + 371.2 |
| | *10–18 years*: RMR = 8.365 W + 465.6 H + 200.0 |
| | *18–30 years*: RMR = 13.623 W + 283.0 H + 98.2 |
| | *30–65 years*: RMR = 8.126 W + 1.43 H + 843.7 |
| | *>65 years*: RMR = 7.887 W + 458.2 H − 17.7 |
| Arciero et al. [36] | RMR = 13.7 FFM + 3.3 FM + 74 $VO_{2max}$ − 50 + 596 |
| Smith et al. [63] | RMR = 3.39 FFM + 0.45 $VO_{2max}$ + 77.41 (kJ/dia)[b] |
| Wang et al. [35] | RMR = 24.6 FFM + 175 |
| Bosy-Westphal et al. [64][c] | *%FM between >10* and *30*: RMR = 11.8 FFM + 14.4 FM + 629.2 |
| | *%FM between >30* and *40*: RMR = 5.5 FFM + 19.3 FM + 926.3 |
| | *%FM between >40* and *50*: RMR = 12.0 FFM + 10.4 FM + 886 |
| | *%FM >50*: RMR = 11.5 FFM + 7.1 FM + 1,097.2 |

*W* weight (kg), *H* height (m), *FFM* fat free mass (kg), *FM* fat mass (%), $VO_{2max}$ maximal oxygen uptake (L $min^{-1}$)
[a]A specific model for each person must be selected after classifying women inside of age grade
[b]To convert kJ to kcal, divide by 4.18
[c]A specific model for each person must be selected after classifying women inside of %FM grade

humans [33]. As discussed in this chapter equations to estimate RMR are in widespread use. Since the first models created by Harris and Benedict [23] several equations have been created using anthropometric variables [34], body composition models [35], or $VO_{2max}$ [36] as predictors of RMR. Nevertheless, FFM continue to be the most important predictor in large heterogeneous samples [37].

FFM is composed of skeletal muscle mass, bone, internal organs, and residual mass, and each one has specific metabolic rates [38]. The latter presents a high variability between subjects with different height, age, and weight body [39]. Indeed, one kg of FFM must have a different RMR between individuals with the same FFM but different height, age, or gender [40], and particularly in obese adults [39]. Moreover, FFM has not been reported to be the principal determinant of RMR in overweight in obese women [41]. In order to resolve this conundrum, new models, which estimate individual masses of internal organs and tissues, have been developed to apply specific-organ metabolic rates to predict RMR [37], whereas older equations, which used anthropometric and two-compartment body composition models, have shown poor validity in predicting RMR in several populations, mainly among overweight and obese people [28, 34, 39]. Since 1918, when Harris and Benedict proposed their classical equation to estimate RMR [23], new models have been developed to estimate RMR, some of them specific for women, ages, and ethnics. Although body composition-based equations should be the best approach, we propose several solutions for different assessment context (see Table 27.6)

#### 27.5.1.1 Adaptive Thermogenesis

Equations developed to estimate RMR have been validated from cross-sectional studies, so those must not always fit to apply in longitudinal interventions, where qualitative and quantitative changes of FFM may result. Alterations in all components of energy expenditure may occurs following weight loss programs with either energy restriction alone, exercise alone or a combination of both [42]. When these alterations are above or below the predictions we can say that an "adaptive thermogenesis" (AT) has occurred. The operational definition of adaptive thermogenesis is: "heat production in response to environmental temperature or diet, and serves the purpose of protecting the organism from cold exposure or regulating energy balance after changes in diet." [43] Adaptive thermogenesis has been considered

to be the result of adaptation to diet and temperature. Adaptive thermogenesis is thought to occur principally in human brown adipose tissue. Since studies have shown that exercise training can modify the energy efficiency of skeletal muscle mass [44], it may be possible that exercise training promotes adaptive thermogenesis in skeletal muscle. While brown adipose tissue is scarce in adult humans, we cannot completely forget the contribution of skeletal muscle to adaptive thermogenesis. Even, the influence of brown adipose tissue deposits have recently been effectively demonstrated in humans also [45]. So greater brown adipose tissue content is related to higher RMR [46], which may have important implications for energy expenditure regulation [47].

Although by definition AT is a concept principally related to RMR, AT could also affect other components of TDEE. Thus, the term "improved energy efficiency" (reduction of the ratio; kJ or kcal of work output/kJ or kcal of internal work) has been coined in the past to explain the phenomenon of reduced EE after physical activity or exercise interventions. However, changes in energy efficiency are conceptually different to the changes of adaptive thermogenesis [see (27.1)].

In this chapter, we only use the term "adaptive thermogenesis" in connection with RMR; even though reductions in walking EE after exercise training may be a form of adaptive thermogenesis. Considering previous paragraphs, AT can be result of increased or decreased EE. Herein, our interest is focused on suppressed adaptive thermogenesis. The importance of adaptive thermogenesis has generated some controversies in the field of the physiology of energy expenditure regulation. Some authors have postulated that alterations on RMR are explained by changes on FFM [42]; moreover, some have proposed that after exercise training RMR is conserved [48]. Since that the concept of AT is governed by reductions in RMR, which are not explained by changes on body composition [49], an AT must not be expected after exercise training, and so RMR will have not any impact on weight control [48]. On the other hand, small alterations in any component of TEE can lead to a substantial impact on daily energy balance [50], other researchers have demonstrated that AT has a clinically significant impact on TEE [47]. Furthermore, adaptive thermogenesis has been confirmed in a number of reports [51]. However, the assessment of AT is difficult and requires good experimental control, large sample size and high accuracy, as small differences can be clinically important in the long-term [50].

Traditionally, a reduction in RMR after weight loss has been associated with a parallel reduction in FFM. Nonetheless, several studies have shown that this adaptation is mostly dependent on FM [52] and distribution of FM and FFM [53], since the reductions in FFM-adjusted RMR present a close relationship either with reductions of FM and FFM after interventions with energy intake restriction [54]. Also, in several studies where exercise and diet were used, a reduction on RMR was confirmed even though FFM was conserved [55], therefore AT must trade on some component of FFM possibly skeletal muscle.

Several molecular mechanism have been suggested to explain an uncoupling between heat produced and synthesized ATP, which may be responsible for AT at skeletal muscle mass level. The proposed molecular mechanisms include: leakage of protons back across the mitochondrial inner membrane which is catalyzed by uncoupling proteins (UCPs [47]); decreased proton pumping by cytochrome oxidase by complex IV [53]; contribution of $Na^+$, $K^+$, and $Ca^{2+}$ ion leaks, and substrate cycles like protein turnover which consume ATP [43]. Considering the previous arguments the study of AT is turning into a new and exciting research area; however, the effect of exercise training has not been fully explored yet.

The estimation of AT requires the assessment of changes in RMR and body composition (FM and FFM) following interventions, and statistical procedures must be performed to create equations to estimate RMR of studied sample from data at baseline, where physiological conditions must be in steady-state. In this way, the changes in RMR can be predicted using simple linear regression models, which were created using body composition variables at baseline as independent variables (FM and FFM). AT can be calculated as proposed by Doucet et al. [56]:

$$AT = \left[EE_{measured} - EE_{estimated}\right]_{after\ intervention} - \left[EE_{measured} - EE_{estimated}\right]_{baseline\ vlues} \quad (27.1)$$

Where, AT, adaptive thermogenesis; EE was energy expenditure either during resting; $EE_{estimated}$ was calculated by stepwise simple linear regression, using FFM and/or FM.

AT has also been explained using the starvation paradigm [56]; however, some questions remain unanswered. The exclusive effects of exercise or the influence of specific protocols of training remains to be determined.

### 27.5.2 NEAT Definition

Another concept that the reader may come across when discussing TEE is the term non-exercise activity thermogenesis (NEAT). When used in the equations to estimate TEE, NEAT is a highly variable component of TEE, ranging from about 15 % in sedentary population to >50 % in highly active people [57]. NEAT is composed of spontaneous physical activity (SPA), which is included and lifestyle physical activities. So NEAT includes the energy expenditure of occupation, leisure, sitting, standing, walking, talking, toe tapping, shopping, household activities, etc. There is a close relationship between change in NEAT and fat mass gain. Since the EE of different activities which are included in NEAT can change after stimuli such as overfeeding or exercise, a theory whereby an improved energetic efficiency after weight loss interventions has been postulated [57], and several neuroendocrine mechanisms have been described to track this resistance to loss fat and weight [58, 59].

Although the concept of NEAT is interesting, its assessment requires measurement of TEE which only can be measured directly by the isotopic technique of doubly labeled water which is highly expensive to apply in large cohorts (see previous sections). Nonetheless, changes estimations of NEAT it would be performed using some devices as accelerometers or pedometers.

Physical activity recalls and pedometer can also be used to perform estimations of NEAT assuming the next paradigm:

- EEPA = ExEE + MVEE + NEAT.
- ExEE = EE from exercise training physical activities.
- MVEE = physical activities which are performed commonly all days, as a job or compulsory transportation, but with moderate/vigorous EE.

Now, we need to quantify total daily movement (using a well-calibrated pedometer) and how we used it. Additionally, a record of ExEE and MVEE hours must be carried out, also steps during both need to be registered. So an estimation of NEAT change can be obtained from the difference between total daily movement (steps) and ExEE + MVEE (steps). A similar approach could be done using accelerometers although more expertise is needed to manage the data.

On the other hand, NEAT can be calculated by a tight recall of daily activities to calculated TDEE, afterwards the EE of EEPA components can be obtained in order to estimate NEAT (see case study).

### 27.5.3 Estimating TEE Using the Compendium of PA

When estimating TEE using these steps, the reader can use the MET values from Table 27.2 or can refer to Appendix 1 for a more detailed list. The MET values listed are from the *Compendium of Physical Activities* which can be found in Appendix 1 or at https://sites.google.com/site/compendiumofphysicalactivities/.

#### 27.5.3.1 Estimating TEE: A Case Study

Alice is a 37-year-old woman, weight 64 kg, 12 % of fat mass (FM). She wants to know what is her TEE and PAL, in order to know if her TEE is enough to maintain the energy balance. Using a recall of one typical day. Calculate total daily energy expenditure (TDEE) of a woman who does not perform any exercise training. Follow the next steps of our example:

First step. Calculate RMR/24 h = kcal/h.

Second step. To count hours sleeping.

Third step. To count hours sitting. Check different activities.

**Table 27.7** Summary of daily activities from the case study

| Hours | Code | kcal |
|---|---|---|
| 24 | PA | 450 kcal/day |
| 23 | PA | |
| 22 | PA | |
| 21 | HA | 563 kcal/day |
| 20 | HA | |
| 19 | HA | |
| 18 | ST | 1,418 kcal/day |
| 17 | ST | |
| 16 | ST | |
| 15 | ST | |
| 14 | ST | |
| 13 | ST | |
| 12 | ST | |
| 11 | ST | |
| 10 | ST | |
| 9 | ST | |
| 8 | ST | |
| 7 | ST | |
| 6 | ST | |
| 5 | S | 328 kcal/day |
| 4 | S | |
| 3 | S | |
| 2 | S | |
| 1 | S | |

*S* sleeping, *ST* sitting, *HA* home activities, *PA* habitual physical activity

Fourth step. To count hours with house activities.
Fifth step. To count hours with physical activities.
*Note*: The sum of total Hours must be equal to 24 h (Table 27.7).

First step. To calculate RMR by hour (it will be useful in order to calculate EE of sleeping):
RMR (kcal/day) = 24.6 × FFM + 175.
FFM = BW − (%FM × BW).
FFM = 75 − (0.12 × 75) = 75 − (9) = 64 kg.
RMR = 24.6 × (64 kg) + 175 = 1,749 kcal/day.
RMR (kcal/h) = 1,749/24 = 72.9 kcal/h.

Second step. EE of sleeping:
1. Hours sleeping = 5 h.
2. EE of sleeping = 0.9 × RMR (kcal/h) or 0.9 METs.
3. EE of sleeping = 0.9 × 72.9 kcal/h = 65.6 kcal/h.

Total EE of sleeping = 65.6 × 5 = 328 kcal/day.

Third step. EE of sitting hours:
1. Hours to eat = 0.5 breakfast + 1 lunch + 0.5 dinner.
2. Hours working = 8 h.
3. Hours watching TV or something like that = 3 h.

Total = 13 h/day.

Use the compendium of PA METs in Appendix 1 to calculate EE:
1. *13030* self care = eating, sitting = *1.5 METs*.
2. *07022* inactivity quiet/light lying = sitting quietly, fidgeting, general, fidgeting hands = *1.5 METs* (or 11770 = 1.3).
3. *07020* inactivity quiet/light lying = sitting and watching television = *1.3 METs*

Calculations:

$$General\ Equation: EE = Hours \times METs\ (kcal/kg/hr) \times BW\ (body\ weight)$$

1. EE to eat = 2 h × 1.5 METs = 3 kcal/kg × 75 kg = 225 kcal/day.
2. EE to work = 8 h × 1.5 METs = 12 kcal/kg × 75 kg = 900 kcal/day.
3. EE during sedentary leisure = 3 h × 1.3 METs = 3.9 kcal/kg × 75 kg = 293 kcal/day.

Total daily EE of sitting hours = 1,418 kcal/day.

Fourth step. EE of home activities:
1. Cleaning = 1 h.
2. Cooking = 1 h.
3. Self-care = 1 h.

Total = 3 h/day.

Use the compendium of PA METs in Appendix 1 to calculate EE:
1. *05026 3.5* home activities = multiple household tasks all at once, moderate effort.
2. *05050 2.0* home activities = cooking or food preparation-standing or sitting or in general (not broken into stand/walk components), manual appliances, light effort.
3. *13040 2.0* self-care = grooming, washing hands, shaving, brushing teeth/showering, toweling off, standing.

Calculations:

1. EE to clean home = 1 h × 3.5 METs = 3.5 kcal/kg × 75 kg = 263 kcal/day.
2. EE to cook = 1 h × 2.0 METs = 2 kcal/kg × 75 kg = 150 kcal/day.
3. EE during sedentary leisure = 1 h × 2.0 METs = 3.9 kcal/kg × 75 kg = 150 kcal/day.

Total Daily EE of Home activities = 563 kcal/day.

Fifth step. EE of other physical, leisure, or transportation activities:

1. Reading = 2 h.
2. Walking = 0.5 h.
3. Walking for transportation = 0.5 h.

Total = 3 h/day.

Use the compendium of PA METs in Appendix 1 to calculate EE:

1. *11580 1.5* occupation = sitting tasks, light effort (e.g., office work, chemistry lab work, computer work, light assembly repair, watch repair, reading, desk work).
2. *17161 2.5* walking = walking from house to car or bus, from car or bus to go places, from car or bus to and from the worksite.
3. *16060 3.5* transportation = walking for transportation, 2.8–3.2 mph, level, moderate pace, firm surface.

Calculations:

1. EE during sedentary leisure = 2.0 h × 1.5 METs = 3.0 kcal/kg × 75 kg = 225 kcal/day.
2. EE during public transportation = 0.5 h × 3.5 METs = 1.75 kcal/kg × 75 kg = 131 kcal/day.
3. Walking EE to pick up public transportation or from = 0.5 h × 2.5 METs = 1.25 kcal/kg × 75 kg = 94 kcal/day.

Total Daily EE of PA or transportation = 450 kcal/day.

Sixth step. TEE and PAL Calculations:

Final results

$$TEE = 2759 kcal / day$$
$$PAL = 2759\left(kcal / day\right) / 1749\left(kcal / day\right) = 1.5$$

As suggested in the literature her PAL is equivalent with a sedentary lifestyle, moreover in order to prevent herself from weight gain, a minimum PAL of 1.70 has been reported (Table 27.8) [60].

**Table 27.8** Table of physical activity levels

| Classification | PAL range |
|---|---|
| Sedentary or light active lifestyle | 1.40–1.69 |
| Active or moderately active lifestyle | 1.70–1.99 |
| Highly active lifestyle | 2.00–2.40 |

Adapted from FAO/WHO/UNU 2001. Human energy requirements. Report of a Joint FAO/WHO/UNU Expert Consultation Rome, 17–24 October 2001

### 27.5.4 Estimating NEAT

### 27.5.5 NEAT Estimation

Components of PAEE:

- EEPA = ExEE + MVEE + NEAT
- NEAT = EEPA − (ExEE + MVEE)

Where:

- ExEE = 0 kcal/day (she does not perform any exercise training).
- MVEE = 0 kcal/day (she is not involved at moderate or vigorous physical activity, which is mandatory).
- NEAT = 1,418 kcal/day (sitting activities) + 563 kcal/day (home activities) ± 450 kcal/day (other activities) = 2,431 kcal/day.

Since MET units include RMR we need to subtract the latter factor in order to obtain the final net EEPA. All together NEAT physical activities lasted 13 h, and RMR was 72.9 kcal/h. So we need to multiply 13h × 72.9 kcal/h in order to calculate kcal that were expended for resting during NEAT activities, which were 948 kcal. So final net NEAT was 1,483 kcal during a day.

Final NEAT Results 2,431 kcal/day − 948 kcal/day = 1,483 kcal/day

## 27.6 Concluding Remarks

The equations presented in this chapter should only be used as a guide to promote optimal energy balance; the individual should be monitored closely to adjust caloric intake based on target goals and changes in body mass. As in all prediction equations, standard errors are inherent [24, 28, 61]. Most of the equations have been developed to maintain current body weight for the par-

ticipant's current activity level; these equations have not been developed to promote weight loss.

The equations discussed in this chapter should be used only as a guide in maintaining energy balance. Even though numerous energy prediction equations to promote energy balance are widely cited in the literature, there are limitations in the use of these equations. Prediction errors are inherent when using any estimated equations. Further validation studies of predictive equations are needed to minimize prediction error in certain age and ethnic groups. Older adults and US residing ethnic minorities have been underrepresented both in the development of predictive equations and in validation studies [28].

Another methodological problem in measuring energy balance, in particular energy intake, is the phenomena of subjects reporting lower energy intake than physiologically required, noted as underreporting. Under reporting of energy intake is expressed as a ratio of reported energy intake to estimated BMR [61]. Underreporting is especially problematic in the obese, but also occurs in the relatively lean population also [62].

A clinical decision of whether an accurate metabolic rate by measurement is required to provide nutritional care and counseling should be made on a case-by-case basis. If the target goals are not being met, the client should be monitored closely utilizing any dietary intake records, energy expenditure logs, and physiological measurements of body mass and or weight change. Indirect calorimetry may be an important tool when, in the judgment of the clinician, the predictive methods fail an individual in a clinically relevant way [15].

## Appendix 1: 2011 Compendium of Physical Activities

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 01003 | 14.0 | Bicycling | Bicycling, mountain, uphill, vigorous |
| 01004 | 16.0 | Bicycling | Bicycling, mountain, competitive, racing |
| *01008* | *8.5* | Bicycling | Bicycling, BMX |
| *01009* | *8.5* | Bicycling | Bicycling, mountain, general |
| 01010 | 4.0 | Bicycling | Bicycling, <10 mph, leisure, to work or for pleasure (Taylor Code 115) |
| 01011 | 6.8 | Bicycling | Bicycling, to/from work, self selected pace |
| 01013 | 5.8 | Bicycling | Bicycling, on dirt or farm road, moderate pace |
| 01015 | 7.5 | Bicycling | Bicycling, general |
| 01018 | 3.5 | Bicycling | Bicycling, leisure, 5.5 mph |
| 01019 | 5.8 | Bicycling | Bicycling, leisure, 9.4 mph |
| 01020 | 6.8 | Bicycling | Bicycling, 10–11.9 mph, leisure, slow, light effort |
| 01030 | 8.0 | Bicycling | Bicycling, 12–13.9 mph, leisure, moderate effort |
| 01040 | 10.0 | Bicycling | Bicycling, 14–15.9 mph, racing or leisure, fast, vigorous effort |
| *01050* | *12.0* | Bicycling | Bicycling, 16–19 mph, racing/not drafting or >19 mph drafting, very fast, racing general |
| 01060 | 15.8 | Bicycling | Bicycling, >20 mph, racing, not drafting |
| 01065 | 8.5 | Bicycling | Bicycling, 12 mph, seated, hands on brake hoods or bar drops, 80 rpm |
| 01066 | 9.0 | Bicycling | Bicycling, 12 mph, standing, hands on brake hoods, 60 rpm |
| *01070* | *5.0* | Bicycling | Unicycling |
| 02001 | 2.3 | Conditioning exercise | Activity promoting video game (e.g., Wii Fit), light effort (e.g., balance, yoga) |
| 02003 | 3.8 | Conditioning exercise | Activity promoting video game (e.g., Wii Fit), moderate effort (e.g., aerobic, resistance) |
| 02005 | 7.2 | Conditioning exercise | Activity promoting video/arcade game (e.g., Exergaming, Dance Dance Revolution), vigorous effort |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 02008 | 5.0 | Conditioning exercise | Army-type obstacle course exercise, boot camp training program |
| *02010* | *7.0* | Conditioning exercise | Bicycling, stationary, general |
| 02011 | 3.5 | Conditioning exercise | Bicycling, stationary, 30–50 W, very light to light effort |
| 02012 | 6.8 | Conditioning exercise | Bicycling, stationary, 90–100 W, moderate to vigorous effort |
| 02013 | 8.8 | Conditioning exercise | Bicycling, stationary, 101–160 W, vigorous effort |
| 02014 | 11.0 | Conditioning exercise | Bicycling, stationary, 161–200 W, vigorous effort |
| 02015 | 14.0 | Conditioning exercise | Bicycling, stationary, 201–270 W, very vigorous effort |
| 02017 | 4.8 | Conditioning exercise | Bicycling, stationary, 51–89 W, light-to-moderate effort |
| 02019 | 8.5 | Conditioning exercise | Bicycling, stationary, RPM/Spin bike class |
| *02020* | *8.0* | Conditioning exercise | Calisthenics (e.g., push-ups, sit-ups, pull-ups, jumping jacks), vigorous effort |
| 02022 | 3.8 | Conditioning exercise | Calisthenics (e.g., push-ups, sit-ups, pull-ups, lunges), moderate effort |
| 02024 | 2.8 | Conditioning exercise | Calisthenics (e.g., sit-ups, abdominal crunches), light effort |
| 02030 | 3.5 | Conditioning exercise | Calisthenics, light or moderate effort, general (e.g., back exercises), going up and down from floor (Taylor Code 150) |
| 02035 | 4.3 | Conditioning exercise | Circuit training, moderate effort |
| 02040 | 8.0 | Conditioning exercise | Circuit training, including kettle bells, some aerobic movement with minimal rest, general, vigorous intensity |
| 02045 | 3.5 | Conditioning exercise | Curves TM exercise routines in women |
| 02048 | 5.0 | Conditioning exercise | Elliptical trainer, moderate effort |
| 02050 | 6.0 | Conditioning exercise | Resistance training (weightlifting, free weight, nautilus or universal), power lifting or body building, vigorous effort (Taylor Code 210) |
| 02052 | 5.0 | Conditioning exercise | Resistance (weight) training, squats, slow or explosive effort |
| 02054 | 3.5 | Conditioning exercise | Resistance (weight) training, multiple exercises, 8–15 repetitions at varied resistance |
| 02060 | 5.5 | Conditioning exercise | Health club exercise, general (Taylor Code 160) |
| 02061 | 5.0 | Conditioning exercise | Health club exercise classes, general, gym/weight training combined in one visit |
| 02062 | 7.8 | Conditioning exercise | Health club exercise, conditioning classes |
| 02064 | 3.8 | Conditioning exercise | Home exercise, general |
| *02065* | *9.0* | Conditioning exercise | Stair-treadmill ergometer, general |
| 02068 | 12.3 | Conditioning exercise | Rope skipping, general |
| *02070* | *6.0* | Conditioning exercise | Rowing, stationary ergometer, general, vigorous effort |
| 02071 | 4.8 | Conditioning exercise | Rowing, stationary, general, moderate effort |
| *02072* | *7.0* | Conditioning exercise | Rowing, stationary, 100 W, moderate effort |
| *02073* | *8.5* | Conditioning exercise | Rowing, stationary, 150 W, vigorous effort |
| *02074* | *12.0* | Conditioning exercise | Rowing, stationary, 200 W, very vigorous effort |
| 02080 | 6.8 | Conditioning exercise | Ski machine, general |
| 02085 | 11.0 | Conditioning exercise | Slide board exercise, general |
| *02090* | *6.0* | Conditioning exercise | Slimnastics, jazzercise |
| 02101 | 2.3 | Conditioning exercise | Stretching, mild |
| 02105 | 3.0 | Conditioning exercise | Pilates, general |
| 02110 | 6.8 | Conditioning exercise | Teaching exercise class (e.g., aerobic, water) |
| 02112 | 2.8 | Conditioning exercise | Therapeutic exercise ball, Fit ball exercise |
| 02115 | 2.8 | Conditioning exercise | Upper body exercise, arm ergometer |
| 02117 | 4.3 | Conditioning exercise | Upper body exercise, Stationary bicycle—Air dyne (arms only) 40 rpm, moderate |
| 02120 | 5.3 | Conditioning exercise | Water aerobics, water calisthenics, water exercise |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| *02135* | *1.3* | Conditioning exercise | Whirlpool, sitting |
| *02140* | *2.3* | Conditioning exercise | Video exercise workouts, TV conditioning programs (e.g., yoga, stretching), light effort |
| *02143* | *4.0* | Conditioning exercise | Video exercise workouts, TV conditioning programs (e.g., cardio-resistance), moderate effort |
| 02146 | 6.0 | Conditioning exercise | Video exercise workouts, TV conditioning programs (e.g., cardio-resistance), vigorous effort |
| 02150 | 2.5 | Conditioning exercise | Yoga, Hatha |
| *02160* | *4.0* | Conditioning exercise | Yoga, Power |
| 02170 | 2.0 | Conditioning exercise | Yoga, Nadi sodhana |
| 02180 | 3.3 | Conditioning exercise | Yoga, Surya Namaskar |
| 02200 | 5.3 | Conditioning exercise | Native New Zealander physical activities (e.g., Haka Powhiri, Moteatea, Waiata Tira, Whakawatea), general, moderate effort |
| 02205 | 6.8 | Conditioning exercise | Native New Zealander physical activities (e.g., Haka, Taiahab), general, vigorous effort |
| 03010 | 5.0 | Dancing | Ballet, modern, or jazz, general, rehearsal or class |
| 03012 | 6.8 | Dancing | Ballet, modern, or jazz, performance, vigorous effort |
| 03014 | 4.8 | Dancing | Tap |
| 03015 | 7.3 | Dancing | Aerobic, general |
| 03016 | 7.5 | Dancing | Aerobic, step, with 6–8 in. step |
| 03017 | 9.5 | Dancing | Aerobic, step, with 10–12 in. step |
| 03018 | 5.5 | Dancing | Aerobic, step, with 4-in. step |
| 03019 | 8.5 | Dancing | Bench step class, general |
| *03020* | *5.0* | Dancing | Aerobic, low impact |
| 03021 | 7.3 | Dancing | Aerobic, high impact |
| 03022 | 10.0 | Dancing | Aerobic dance wearing 10–15 lb weights |
| *03025* | *4.5* | Dancing | Ethnic or cultural dancing (e.g., Greek, Middle Eastern, hula, salsa, merengue, bomba y plena, flamenco, belly, and swing) |
| 03030 | 5.5 | Dancing | Ballroom, fast (Taylor Code 125) |
| 03031 | 7.8 | Dancing | General dancing (e.g., disco, folk, Irish step dancing, line dancing, polka, contra, country) |
| 03038 | 11.3 | Dancing | Ballroom dancing, competitive, general |
| *03040* | *3.0* | Dancing | Ballroom, slow (e.g., waltz, foxtrot, slow dancing, samba, tango, nineteenth century dance, mambo, cha-cha) |
| 03050 | 5.5 | Dancing | Anishinaabe Jingle Dancing |
| 03060 | 3.5 | Dancing | Caribbean dance (Abakua, Beguine, Bellair, Bongo, Brukin's, Caribbean Quadrills, Dinki Mini, Gere, Gumbay, Ibo, Jonkonnu, Kumina, Oreisha, Jambu) |
| 04001 | 3.5 | Fishing and hunting | Fishing, general |
| 04005 | 4.5 | Fishing and hunting | Fishing, crab fishing |
| 04007 | 4.0 | Fishing and hunting | Fishing, catching fish with hands |
| 04010 | 4.3 | Fishing and hunting | Fishing related, digging worms, with shovel |
| *04020* | *4.0* | Fishing and hunting | Fishing from river bank and walking |
| *04030* | *2.0* | Fishing and hunting | Fishing from boat or canoe, sitting |
| *04040* | *3.5* | Fishing and hunting | Fishing from river bank, standing (Taylor Code 660) |
| *04050* | *6.0* | Fishing and hunting | Fishing in stream, in waders (Taylor Code 670) |
| *04060* | *2.0* | Fishing and hunting | Fishing, ice, sitting |
| 04061 | 1.8 | Fishing and hunting | Fishing, jog or line, standing, general |
| *04062* | *3.5* | Fishing and hunting | Fishing, dip net, setting net and retrieving fish, general |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 04063 | 3.8 | Fishing and hunting | Fishing, set net, setting net and retrieving fish, general |
| *04064* | *3.0* | Fishing and hunting | Fishing, fishing wheel, setting net and retrieving fish, general |
| 04065 | 2.3 | Fishing and hunting | Fishing with a spear, standing |
| *04070* | *2.5* | Fishing and hunting | Hunting, bow and arrow, or crossbow |
| 04080 | 6.0 | Fishing and hunting | Hunting, deer, elk, large game (Taylor Code 170) |
| 04081 | 11.3 | Fishing and hunting | Hunting large game, dragging carcass |
| *04083* | *4.0* | Fishing and hunting | Hunting large marine animals |
| *04085* | *2.5* | Fishing and hunting | Hunting large game, from a hunting stand, limited walking |
| *04086* | *2.0* | Fishing and hunting | Hunting large game from a car, plane, or boat |
| *04090* | *2.5* | Fishing and hunting | Hunting, duck, wading |
| 04095 | 3.0 | Fishing and hunting | Hunting, flying fox, squirrel |
| *04100* | *5.0* | Fishing and hunting | Hunting, general |
| 04110 | 6.0 | Fishing and hunting | Hunting, pheasants or grouse (Taylor Code 680) |
| 04115 | 3.3 | Fishing and hunting | Hunting, birds |
| 04120 | 5.0 | Fishing and hunting | Hunting, rabbit, squirrel, prairie chick, raccoon, small game (Taylor Code 690) |
| 04123 | 3.3 | Fishing and hunting | Hunting, pigs, wild |
| *04124* | *2.0* | Fishing and hunting | Trapping game, general |
| 04125 | 9.5 | Fishing and hunting | Hunting, hiking with hunting gear |
| *04130* | *2.5* | Fishing and hunting | Pistol shooting or trap shooting, standing |
| 04140 | 2.3 | Fishing and hunting | Rifle exercises, shooting, lying down |
| 04145 | 2.5 | Fishing and hunting | Rifle exercises, shooting, kneeling or standing |
| 05010 | 3.3 | Home activities | Cleaning, sweeping carpet or floors, general |
| 05011 | 2.3 | Home activities | Cleaning, sweeping, slow, light effort |
| 05012 | 3.8 | Home activities | Cleaning, sweeping, slow, moderate effort |
| *05020* | *3.5* | Home activities | Cleaning, heavy or major (e.g., wash car, wash windows, clean garage), moderate effort |
| 05021 | 3.5 | Home activities | Cleaning, mopping, standing, moderate effort |
| 05022 | 3.2 | Home activities | Cleaning windows, washing windows, general |
| 05023 | 2.5 | Home activities | Mopping, standing, light effort |
| 05024 | 4.5 | Home activities | Polishing floors, standing, walking slowly, using electric polishing machine |
| 05025 | 2.8 | Home activities | Multiple household tasks all at once, light effort |
| *05026* | *3.5* | Home activities | Multiple household tasks all at once, moderate effort |
| 05027 | 4.3 | Home activities | Multiple household tasks all at once, vigorous effort |
| 05030 | 3.3 | Home activities | Cleaning, house or cabin, general, moderate effort |
| 05032 | 2.3 | Home activities | Dusting or polishing furniture, general |
| 05035 | 3.3 | Home activities | Kitchen activity, general (e.g., cooking, washing dishes, cleaning up), moderate effort |
| 05040 | 2.5 | Home activities | Cleaning, general (straightening up, changing linen, carrying out trash), light effort |
| 05041 | 1.8 | Home activities | Wash dishes, standing or in general (not broken into stand/walk components) |
| *05042* | *2.5* | Home activities | Wash dishes, clearing dishes from table, walking, light effort |
| 05043 | 3.3 | Home activities | Vacuuming, general, moderate effort |
| *05044* | *3.0* | Home activities | Butchering animals, small |
| *05045* | *6.0* | Home activities | Butchering animal, large, vigorous effort |
| *05046* | *2.3* | Home activities | Cutting and smoking fish, drying fish or meat |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| *05048* | *4.0* | Home activities | Tanning hides, general |
| 05049 | 3.5 | Home activities | Cooking or food preparation, moderate effort |
| 05050 | 2.0 | Home activities | Cooking or food preparation—standing or sitting or in general (not broken into stand/walk components), manual appliances, light effort |
| *05051* | *2.5* | Home activities | Serving food, setting table, implied walking or standing |
| *05052* | *2.5* | Home activities | Cooking or food preparation, walking |
| *05053* | *2.5* | Home activities | Feeding household animals |
| *05055* | *2.5* | Home activities | Putting away groceries (e.g., carrying groceries, shopping without a grocery cart), carrying packages |
| *05056* | *7.5* | Home activities | Carrying groceries upstairs |
| *05057* | *3.0* | Home activities | Cooking Indian bread on an outside stove |
| 05060 | 2.3 | Home activities | Food shopping with or without a grocery cart, standing or walking |
| *05065* | *2.3* | Home activities | Non-food shopping, with or without a cart, standing or walking |
| 05070 | 1.8 | Home activities | Ironing |
| 05080 | 1.3 | Home activities | Knitting, sewing, light effort, wrapping presents, sitting |
| 05082 | 2.8 | Home activities | Sewing with a machine |
| 05090 | 2.0 | Home activities | Laundry, fold or hang clothes, put clothes in washer or dryer, packing suitcase, washing clothes by hand, implied standing, light effort |
| 05092 | 4.0 | Home activities | Laundry, hanging wash, washing clothes by hand, moderate effort |
| *05095* | *2.3* | Home activities | Laundry, putting away clothes, gathering clothes to pack, putting away laundry, implied walking |
| 05100 | 3.3 | Home activities | Making bed, changing linens |
| 05110 | 5.0 | Home activities | Maple syruping/sugar bushing (including carrying buckets, carrying wood) |
| 05120 | 5.8 | Home activities | Moving furniture, household items, carrying boxes |
| 05121 | 5.0 | Home activities | Moving, lifting light loads |
| 05125 | 4.8 | Home activities | Organizing room |
| 05130 | 3.5 | Home activities | Scrubbing floors, on hands and knees, scrubbing bathroom, bathtub, moderate effort |
| 05131 | 2.0 | Home activities | Scrubbing floors, on hands and knees, scrubbing bathroom, bathtub, light effort |
| 05132 | 6.5 | Home activities | Scrubbing floors, on hands and knees, scrubbing bathroom, bathtub, vigorous effort |
| *05140* | *4.0* | Home activities | Sweeping garage, side walk or outside of house |
| *05146* | *3.5* | Home activities | Standing, packing/unpacking boxes, occasional lifting of light weight household items, loading or unloading items in car, moderate effort |
| *05147* | *3.0* | Home activities | Implied walking, putting away household items, moderate effort |
| *05148* | *2.5* | Home activities | Watering plants |
| *05149* | *2.5* | Home activities | Building a fire inside |
| *05150* | *9.0* | Home activities | Moving household items upstairs, carrying boxes or furniture |
| *05160* | *2.0* | Home activities | Standing, light effort tasks (pump gas, change light bulb, etc.) |
| 05165 | 3.5 | Home activities | Walking, moderate effort tasks, non-cleaning (readying to leave, shut/lock doors, close windows, etc.) |
| 05170 | 2.2 | Home activities | Sitting, playing with child(ren), light effort, only active periods |
| *05171* | *2.8* | Home activities | Standing, playing with child(ren) light effort, only active periods |
| 05175 | 3.5 | Home activities | Walking/running, playing with child(ren), moderate effort, only active periods |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 05180 | 5.8 | Home activities | Walking/running, playing with child(ren), vigorous effort, only active periods |
| *05181* | *3.0* | Home activities | Walking and carrying small child, child weighing 15 lb or more |
| 05182 | 2.3 | Home activities | Walking and carrying small child, child weighing less than 15 lb |
| 05183 | 2.0 | Home activities | Standing, holding child |
| 05184 | 2.5 | Home activities | Child care, infant, general |
| 05185 | 2.0 | Home activities | Child care, sitting/kneeling (e.g., dressing, bathing, grooming, feeding, occasional lifting of child), light effort, general |
| *05186* | *3.0* | Home activities | Child care, standing (e.g., dressing, bathing, grooming, feeding, occasional lifting of child), moderate effort |
| *05188* | *1.5* | Home activities | Reclining with baby |
| 05189 | 2.0 | Home activities | Breastfeeding, sitting or reclining |
| *05190* | *2.5* | Home activities | Sit, playing with animals, light effort, only active periods |
| *05191* | *2.8* | Home activities | Stand, playing with animals, light effort, only active periods |
| 05192 | 3.0 | Home activities | Walk/run, playing with animals, general, light effort, only active periods |
| *05193* | *4.0* | Home activities | Walk/run, playing with animals, moderate effort, only active periods |
| *05194* | *5.0* | Home activities | Walk/run, playing with animals, vigorous effort, only active periods |
| *05195* | *3.5* | Home activities | Standing, bathing dog |
| *05197* | *2.3* | Home activities | Animal care, household animals, general |
| *05200* | *4.0* | Home activities | Elder care, disabled adult, bathing, dressing, moving into and out of bed, only active periods |
| *05205* | *2.3* | Home activities | Elder care, disabled adult, feeding, combing hair, light effort, only active periods |
| *06010* | *3.0* | Home repair | Airplane repair |
| *06020* | *4.0* | Home repair | Automobile body work |
| 06030 | 3.3 | Home repair | Automobile repair, light or moderate effort |
| 06040 | 3.0 | Home repair | Carpentry, general, workshop (Taylor Code 620) |
| 06050 | 6.0 | Home repair | Carpentry, outside house, installing rain gutters (Taylor Code 640), carpentry, outside house, building a fence |
| 06052 | 3.8 | Home repair | Carpentry, outside house, building a fence |
| 06060 | 3.3 | Home repair | Carpentry, finishing or refinishing cabinets or furniture |
| 06070 | 6.0 | Home repair | Carpentry, sawing hardwood |
| 06072 | 4.0 | Home repair | Carpentry, home remodeling tasks, moderate effort |
| 06074 | 2.3 | Home repair | Carpentry, home remodeling tasks, light effort |
| *06080* | *5.0* | Home repair | Caulking, chinking log cabin |
| *06090* | *4.5* | Home repair | Caulking, except log cabin |
| *06100* | *5.0* | Home repair | Cleaning gutters |
| *06110* | *5.0* | Home repair | Excavating garage |
| *06120* | *5.0* | Home repair | Hanging storm windows |
| *06122* | *5.0* | Home repair | Hanging sheetrock inside house |
| 06124 | 3.0 | Home repair | Hammering nails |
| *06126* | *2.5* | Home repair | Home repair, general, light effort |
| *06127* | *4.5* | Home repair | Home repair, general, moderate effort |
| *06128* | *6.0* | Home repair | Home repair, general, vigorous effort |
| *06130* | *4.5* | Home repair | Laying or removing carpet |
| 06140 | 3.8 | Home repair | Laying tile or linoleum, repairing appliances |
| *06144* | *3.0* | Home repair | Repairing appliances |
| 06150 | 5.0 | Home repair | Painting, outside home (Taylor Code 650) |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 06160 | 3.3 | Home repair | Painting inside house, wallpapering, scraping paint |
| 06165 | 4.5 | Home repair | Painting (Taylor Code 630) |
| *06167* | *3.0* | Home repair | Plumbing, general |
| *06170* | *3.0* | Home repair | Put on and removal of tarp—sailboat |
| *06180* | *6.0* | Home repair | Roofing |
| *06190* | *4.5* | Home repair | Sanding floors with a power sander |
| *06200* | *4.5* | Home repair | Scraping and painting sailboat or powerboat |
| 06205 | 2.0 | Home repair | Sharpening tools |
| *06210* | *5.0* | Home repair | Spreading dirt with a shovel |
| *06220* | *4.5* | Home repair | Washing and waxing hull of sailboat or airplane |
| 06225 | 2.0 | Home repair | Washing and waxing car |
| *06230* | *4.5* | Home repair | Washing fence, painting fence, moderate effort |
| 06240 | 3.3 | Home repair | Wiring, tapping-splicing |
| *07010* | *1.0* | Inactivity quiet/light | Lying quietly and watching television |
| 07011 | 1.3 | Inactivity quiet/light | Lying quietly, doing nothing, lying in bed awake, listening to music (not talking or reading) |
| 07020 | 1.3 | Inactivity quiet/light | Sitting quietly and watching television |
| 07021 | 1.3 | Inactivity quiet/light | Sitting quietly, general |
| 07022 | 1.5 | Inactivity quiet/light | Sitting quietly, fidgeting, general, fidgeting hands |
| 07023 | 1.8 | Inactivity quiet/light | Sitting, fidgeting feet |
| *07024* | *1.3* | Inactivity quiet/light | Sitting, smoking |
| 07025 | 1.5 | Inactivity quiet/light | Sitting, listening to music (not talking or reading) or watching a movie in a theater |
| 07026 | 1.3 | Inactivity quiet/light | Sitting at a desk, resting head in hands |
| 07030 | 0.95 | Inactivity quiet/light | Sleeping |
| 07040 | 1.3 | Inactivity quiet/light | Standing quietly, standing in a line |
| 07041 | 1.8 | Inactivity quiet/light | Standing, fidgeting |
| *07050* | *1.3* | Inactivity quiet/light | Reclining, writing |
| *07060* | *1.3* | Inactivity quiet/light | Reclining, talking or talking on phone |
| 07070 | 1.3 | Inactivity quiet/light | Reclining, reading |
| *07075* | *1.0* | Inactivity quiet/light | Meditating |
| 08009 | 3.3 | Lawn and garden | Carrying, loading or stacking wood, loading/unloading or carrying lumber, light-to-moderate effort |
| 08010 | 5.5 | Lawn and garden | Carrying, loading or stacking wood, loading/unloading or carrying lumber |
| 08019 | 4.5 | Lawn and garden | Chopping wood, splitting logs, moderate effort |
| 08020 | 6.3 | Lawn and garden | Chopping wood, splitting logs, vigorous effort |
| 08025 | 3.5 | Lawn and garden | Clearing light brush, thinning garden, moderate effort |
| 08030 | 6.3 | Lawn and garden | Clearing brush/land, undergrowth, or ground, hauling branches, wheelbarrow chores, vigorous effort |
| 08040 | 5.0 | Lawn and garden | Digging sandbox, shoveling sand |
| 08045 | 3.5 | Lawn and garden | Digging, spading, filling garden, composting, light-to-moderate effort |
| 08050 | 5.0 | Lawn and garden | Digging, spading, filling garden, compositing (Taylor Code 590) |
| 08052 | 7.8 | Lawn and garden | Digging, spading, filling garden, composting, vigorous effort |
| 08055 | 2.8 | Lawn and garden | Driving tractor |
| 08057 | 8.3 | Lawn and garden | Felling trees, large size |
| 08058 | 5.3 | Lawn and garden | Felling trees, small-medium size |
| 08060 | 5.8 | Lawn and garden | Gardening with heavy power tools, tilling a garden, chain saw |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 08065 | 2.3 | Lawn and garden | Gardening, using containers, older adults >60 years |
| 08070 | 4.0 | Lawn and garden | Irrigation channels, opening and closing ports |
| 08080 | 6.3 | Lawn and garden | Laying crushed rock |
| *08090* | *5.0* | Lawn and garden | Laying sod |
| 08095 | 5.5 | Lawn and garden | Mowing lawn, general |
| 08100 | 2.5 | Lawn and garden | Mowing lawn, riding mower (Taylor Code 550) |
| 08110 | 6.0 | Lawn and garden | Mowing lawn, walk, hand mower (Taylor Code 570) |
| 08120 | 5.0 | Lawn and garden | Mowing lawn, walk, power mower, moderate or vigorous effort |
| 08125 | 4.5 | Lawn and garden | Mowing lawn, power mower, light or moderate effort (Taylor Code 590) |
| 08130 | 2.5 | Lawn and garden | Operating snow blower, walking |
| 08135 | 2.0 | Lawn and garden | Planting, potting, transplanting seedlings or plants, light effort |
| 08140 | 4.3 | Lawn and garden | Planting seedlings, shrub, stooping, moderate effort |
| 08145 | 4.3 | Lawn and garden | Planting crops or garden, stooping, moderate effort |
| *08150* | *4.5* | Lawn and garden | Planting trees |
| 08160 | 3.8 | Lawn and garden | Raking lawn or leaves, moderate effort |
| 08165 | 4.0 | Lawn and garden | Raking lawn (Taylor Code 600) |
| *08170* | *4.0* | Lawn and garden | Raking roof with snow rake |
| *08180* | *3.0* | Lawn and garden | Riding snow blower |
| *08190* | *4.0* | Lawn and garden | Sacking grass, leaves |
| 08192 | 5.5 | Lawn and garden | Shoveling dirt or mud |
| 08195 | 5.3 | Lawn and garden | Shoveling snow, by hand, moderate effort |
| 08200 | 6.0 | Lawn and garden | Shoveling snow, by hand (Taylor Code 610) |
| 08202 | 7.5 | Lawn and garden | Shoveling snow, by hand, vigorous effort |
| 08210 | 4.0 | Lawn and garden | Trimming shrubs or trees, manual cutter |
| 08215 | 3.5 | Lawn and garden | Trimming shrubs or trees, power cutter, using leaf blower, edge, moderate effort |
| 08220 | 3.0 | Lawn and garden | Walking, applying fertilizer or seeding a lawn, push applicator |
| *08230* | *1.5* | Lawn and garden | Watering lawn or garden, standing or walking |
| 08239 | 3.5 | Lawn and garden | Weeding, cultivating garden, light-to-moderate effort |
| 08240 | 4.5 | Lawn and garden | Weeding, cultivating garden (Taylor Code 580) |
| 08241 | 5.0 | Lawn and garden | Weeding, cultivating garden, using a hoe, moderate-to-vigorous effort |
| 08245 | 3.8 | Lawn and garden | Gardening, general, moderate effort |
| 08246 | 3.5 | Lawn and garden | Picking fruit off trees, picking fruits/vegetables, moderate effort |
| 08248 | 4.5 | Lawn and garden | Picking fruit off trees, gleaning fruits, picking fruits/vegetables, climbing ladder to pick fruit, vigorous effort |
| 08250 | 3.3 | Lawn and garden | Implied walking/standing—picking up yard, light, picking flowers or vegetables |
| *08251* | *3.0* | Lawn and garden | Walking, gathering gardening tools |
| 08255 | 5.5 | Lawn and garden | Wheelbarrow, pushing garden cart or wheelbarrow |
| 08260 | 3.0 | Lawn and garden | Yard work, general, light effort |
| 08261 | 4.0 | Lawn and garden | Yard work, general, moderate effort |
| 08262 | 6.0 | Lawn and garden | Yard work, general, vigorous effort |
| *09000* | *1.5* | Miscellaneous | Board game playing, sitting |
| *09005* | *2.5* | Miscellaneous | Casino gambling, standing |
| 09010 | 1.5 | Miscellaneous | Card playing, sitting |
| 09013 | 1.5 | Miscellaneous | Chess game, sitting |
| 09015 | 1.5 | Miscellaneous | Copying documents, standing |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 09020 | 1.8 | Miscellaneous | Drawing, writing, painting, standing |
| 09025 | 1.0 | Miscellaneous | Laughing, sitting |
| 09030 | 1.3 | Miscellaneous | Sitting, reading, book, newspaper, etc. |
| 09040 | 1.3 | Miscellaneous | Sitting, writing, deskwork, typing |
| 09045 | 1.0 | Miscellaneous | Sitting, playing traditional video game, computer game |
| *09050* | *1.8* | Miscellaneous | Standing, talking in person, on the phone, computer, or text messaging, light effort |
| *09055* | *1.5* | Miscellaneous | Sitting, talking in person, on the phone, computer, or text messaging, light effort |
| 09060 | 1.3 | Miscellaneous | Sitting, studying, general, including reading and/or writing, light effort |
| 09065 | 1.8 | Miscellaneous | Sitting, in class, general, including note-taking or class discussion |
| *09070* | *1.8* | Miscellaneous | Standing, reading |
| 09071 | 2.5 | Miscellaneous | Standing, miscellaneous |
| 09075 | 1.8 | Miscellaneous | Sitting, arts and crafts, carving wood, weaving, spinning wool, light effort |
| *09080* | *3.0* | Miscellaneous | Sitting, arts and crafts, carving wood, weaving, spinning wool, moderate effort |
| 09085 | 2.5 | Miscellaneous | Standing, arts and crafts, sand painting, carving, weaving, light effort |
| *09090* | *3.3* | Miscellaneous | Standing, arts and crafts, sand painting, carving, weaving, moderate effort |
| *09095* | *3.5* | Miscellaneous | Standing, arts and crafts, sand painting, carving, weaving, vigorous effort |
| 09100 | 1.8 | Miscellaneous | Retreat/family reunion activities involving sitting, relaxing, talking, eating |
| 09101 | 3.0 | Miscellaneous | Retreat/family reunion activities involving playing games with children |
| *09105* | *2.0* | Miscellaneous | Touring/traveling/vacation involving riding in a vehicle |
| *09106* | *3.5* | Miscellaneous | Touring/traveling/vacation involving walking |
| *09110* | *2.5* | Miscellaneous | Camping involving standing, walking, sitting, light-to-moderate effort |
| *09115* | *1.5* | Miscellaneous | Sitting at a sporting event, spectator |
| 10010 | 1.8 | Music playing | Accordion, sitting |
| 10020 | 2.3 | Music playing | Cello, sitting |
| 10030 | 2.3 | Music playing | Conducting orchestra, standing |
| 10035 | 2.5 | Music playing | Double bass, standing |
| 10040 | 3.8 | Music playing | Drums, sitting |
| 10045 | 3.0 | Music playing | Drumming (e.g., bongo, conga, bembe), moderate, sitting |
| 10050 | 2.0 | music playing | Flute, sitting |
| 10060 | 1.8 | Music playing | Horn, standing |
| 10070 | 2.3 | Music playing | Piano, sitting |
| 10074 | 2.0 | Music playing | Playing musical instruments, general |
| 10077 | 2.0 | Music playing | Organ, sitting |
| 10080 | 3.5 | Music playing | Trombone, standing |
| 10090 | 1.8 | Music playing | Trumpet, standing |
| 10100 | 2.5 | Music playing | Violin, sitting |
| 10110 | 1.8 | Music playing | Woodwind, sitting |
| *10120* | *2.0* | Music playing | Guitar, classical, folk, sitting |
| *10125* | *3.0* | Music playing | Guitar, rock and roll band, standing |
| *10130* | *4.0* | Music playing | Marching band, baton twirling, walking, moderate pace, general |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 10131 | 5.5 | Music playing | Marching band, playing an instrument, walking, brisk pace, general |
| *10135* | *3.5* | Music playing | Marching band, drum major, walking |
| 11003 | 2.3 | Occupation | Active workstation, treadmill desk, walking |
| 11006 | 3.0 | Occupation | Airline flight attendant |
| *11010* | *4.0* | Occupation | Bakery, general, moderate effort |
| 11015 | 2.0 | Occupation | Bakery, light effort |
| 11020 | 2.3 | Occupation | Bookbinding |
| *11030* | *6.0* | Occupation | Building road, driving heavy machinery |
| *11035* | *2.0* | Occupation | Building road, directing traffic, standing |
| 11038 | 2.5 | Occupation | Carpentry, general, light effort |
| 11040 | 4.3 | Occupation | Carpentry, general, moderate effort |
| 11042 | 7.0 | Occupation | Carpentry, general, heavy or vigorous effort |
| *11050* | *8.0* | Occupation | Carrying heavy loads (e.g., bricks, tools) |
| *11060* | *8.0* | Occupation | Carrying moderate loads up stairs, moving boxes 25–49 lb |
| 11070 | 4.0 | Occupation | Chambermaid, hotel housekeeper, making bed, cleaning bathroom, pushing cart |
| 11080 | 5.3 | Occupation | Coalmining, drilling coal, rock |
| 11090 | 5.0 | Occupation | Coalmining, erecting supports |
| 11100 | 5.5 | Occupation | Coalmining, general |
| 11110 | 6.3 | Occupation | Coalmining, shoveling coal |
| 11115 | 2.5 | Occupation | Cook, chef |
| 11120 | 4.0 | Occupation | Construction, outside, remodeling, new structures (e.g., roof repair, miscellaneous) |
| 11125 | 2.3 | Occupation | Custodial work, light effort (e.g., cleaning sink and toilet, dusting, vacuuming, light cleaning) |
| 11126 | 3.8 | Occupation | Custodial work, moderate effort (e.g., electric buffer, feathering arena floors, mopping, taking out trash, vacuuming) |
| 11128 | 2.0 | Occupation | Driving delivery truck, taxi, shuttle bus, school bus |
| 11130 | 3.3 | Occupation | Electrical work (e.g., hookup wire, tapping-splicing) |
| 11135 | 1.8 | Occupation | Engineer (e.g., mechanical or electrical) |
| 11145 | 7.8 | Occupation | Farming, vigorous effort (e.g., baling hay, cleaning barn) |
| 11146 | 4.8 | Occupation | Farming, moderate effort (e.g., feeding animals, chasing cattle by walking and/or horseback, spreading manure, harvesting crops) |
| 11147 | 2.0 | Occupation | Farming, light effort (e.g., cleaning animal sheds, preparing animal feed) |
| 11170 | 2.8 | Occupation | Farming, driving tasks (e.g., driving tractor or harvester) |
| 11180 | 3.5 | Occupation | Farming, feeding small animals |
| 11190 | 4.3 | Occupation | Farming, feeding cattle, horses |
| 11191 | 4.3 | Occupation | Farming, hauling water for animals, general hauling water, farming, general hauling water |
| 11192 | 4.5 | Occupation | Farming, taking care of animals (e.g., grooming, brushing, shearing sheep, assisting with birthing, medical care, branding), general |
| 11195 | 3.8 | Occupation | Farming, rice, planting, grain milling activities |
| 11210 | 3.5 | Occupation | Farming, milking by hand, cleaning pails, moderate effort |
| 11220 | 1.3 | Occupation | Farming, milking by machine, light effort |
| 11240 | 8.0 | Occupation | Fire fighter, general |
| 11244 | 6.8 | Occupation | Fire fighter, rescue victim, automobile accident, using pike pole |
| 11245 | 8.0 | Occupation | Fire fighter, raising and climbing ladder with full gear, simulated fire suppression |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 11246 | 9.0 | Occupation | Fire fighter, hauling hoses on ground, carrying/hoisting equipment, breaking down walls etc., wearing full gear |
| *11247* | *3.5* | Occupation | Fishing, commercial, light effort |
| *11248* | *5.0* | Occupation | Fishing, commercial, moderate effort |
| *11249* | *7.0* | Occupation | Fishing, commercial, vigorous effort |
| 11250 | 17.5 | Occupation | Forestry, ax chopping, very fast, 1.25 kg ax, 51 blows/min, extremely vigorous effort |
| 11260 | 5.0 | Occupation | Forestry, ax chopping, slow, 1.25 kg ax, 19 blows/min, moderate effort |
| 11262 | 8.0 | Occupation | Forestry, ax chopping, fast, 1.25 kg ax, 35 blows/min, vigorous effort |
| 11264 | 4.5 | Occupation | Forestry, moderate effort (e.g., sawing wood with power saw, weeding, hoeing) |
| 11266 | 8.0 | Occupation | Forestry, vigorous effort (e.g., barking, felling, or trimming trees, carrying or stacking logs, planting seeds, sawing lumber by hand) |
| 11370 | 4.5 | Occupation | Furriery |
| 11375 | 4.0 | Occupation | Garbage collector, walking, dumping bins into truck |
| 11378 | 1.8 | Occupation | Hairstylist (e.g., plaiting hair, manicure, make-up artist) |
| 11380 | 7.3 | Occupation | Horse grooming, including feeding, cleaning stalls, bathing, brushing, clipping, lunging, and exercising horses |
| *11381* | *4.3* | Occupation | Horse, feeding, watering, cleaning stalls, implied walking and lifting loads |
| 11390 | 7.3 | Occupation | Horse racing, galloping |
| 11400 | 5.8 | Occupation | Horse racing, trotting |
| 11410 | 3.8 | Occupation | Horse racing, walking |
| 11413 | 3.0 | Occupation | Kitchen maid |
| 11415 | 4.0 | Occupation | Lawn keeper, yard work, general |
| 11418 | 3.3 | Occupation | Laundry worker |
| 11420 | 3.0 | Occupation | Locksmith |
| 11430 | 3.0 | Occupation | Machine tooling (e.g., machining, working sheet metal, machine fitter, operating lathe, welding) light-to-moderate effort |
| 11450 | 5.0 | Occupation | Machine tooling, operating punch press, moderate effort |
| 11472 | 1.8 | Occupation | Manager, property |
| *11475* | *2.8* | Occupation | Manual or unskilled labor, general, light effort |
| *11476* | *4.5* | Occupation | Manual or unskilled labor, general, moderate effort |
| *11477* | *6.5* | Occupation | Manual or unskilled labor, general, vigorous effort |
| 11480 | 4.3 | Occupation | Masonry, concrete, moderate effort |
| 11482 | 2.5 | Occupation | Masonry, concrete, light effort |
| *11485* | *4.0* | Occupation | Massage therapist, standing |
| *11490* | *7.5* | Occupation | Moving, carrying or pushing heavy objects, 75 lb or more, only active time (e.g., desks, moving van work) |
| *11495* | *12.0* | Occupation | Skin diving or SCUBA diving as a frogman, Navy Seal |
| *11500* | *2.5* | Occupation | Operating heavy duty equipment, automated, not driving |
| 11510 | 4.5 | Occupation | Orange grove work, picking fruit |
| 11514 | 3.3 | Occupation | Painting, house, furniture, moderate effort |
| *11516* | *3.0* | Occupation | Plumbing activities |
| 11520 | 2.0 | Occupation | Printing, paper industry worker, standing |
| *11525* | *2.5* | Occupation | Police, directing traffic, standing |
| 11526 | 2.5 | Occupation | Police, driving a squad car, sitting |
| 11527 | 1.3 | Occupation | Police, riding in a squad car, sitting |
| *11528* | *4.0* | Occupation | Police, making an arrest, standing |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 11529 | 2.3 | Occupation | Postal carrier, walking to deliver mail |
| 11530 | 2.0 | Occupation | Shoe repair, general |
| 11540 | 7.8 | Occupation | Shoveling, digging ditches |
| 11550 | 8.8 | Occupation | Shoveling, more than 16 lb/min, deep digging, vigorous effort |
| 11560 | 5.0 | Occupation | Shoveling, less than 10 lb/min, moderate effort |
| 11570 | 6.5 | Occupation | Shoveling, 10–15 lb/min, vigorous effort |
| 11580 | 1.5 | Occupation | Sitting tasks, light effort (e.g., office work, chemistry lab work, computer work, light assembly repair, watch repair, reading, deskwork) |
| 11585 | 1.5 | Occupation | Sitting meetings, light effort, general, and/or with talking involved (e.g., eating at a business meeting) |
| *11590* | *2.5* | Occupation | Sitting tasks, moderate effort (e.g., pushing heavy levers, riding mower/forklift, crane operation) |
| *11593* | *2.8* | Occupation | Sitting, teaching stretching or yoga, or light effort exercise class |
| 11600 | 3.0 | Occupation | Standing tasks, light effort (e.g., bartending, store clerk, assembling, filing, duplicating, librarian, putting up a Christmas tree, standing and talking at work, changing clothes when teaching physical education, standing) |
| *11610* | *3.0* | Occupation | Standing, light/moderate effort (e.g., assemble/repair heavy parts, welding, stocking parts, auto repair, standing, packing boxes, nursing patient care) |
| 11615 | 4.5 | Occupation | Standing, moderate effort, lifting items continuously, 10–20 lb, with limited walking or resting |
| 11620 | 3.5 | Occupation | Standing, moderate effort, intermittent lifting 50 lb, hitch/twisting ropes |
| 11630 | 4.5 | Occupation | Standing, moderate/heavy tasks (e.g., lifting more than 50 lb, masonry, painting, paper hanging) |
| 11708 | 5.3 | Occupation | Steel mill, moderate effort (e.g., fettling, forging, tipping molds) |
| 11710 | 8.3 | Occupation | Steel mill, vigorous effort (e.g., hand rolling, merchant mill rolling, removing slag, tending furnace) |
| 11720 | 2.3 | Occupation | Tailoring, cutting fabric |
| 11730 | 2.5 | Occupation | Tailoring, general |
| 11740 | 1.8 | Occupation | Tailoring, hand sewing |
| 11750 | 2.5 | Occupation | Tailoring, machine sewing |
| 11760 | 3.5 | Occupation | Tailoring, pressing |
| 11763 | 2.0 | Occupation | Tailoring, weaving, light effort (e.g., finishing operations, washing, dyeing, inspecting cloth, counting yards, paperwork) |
| 11765 | 4.0 | Occupation | Tailoring, weaving, moderate effort (e.g., spinning and weaving operations, delivering boxes of yam to spinners, loading of warp bean, pin winding, cone winding, warping, cloth cutting) |
| *11766* | *6.5* | Occupation | Truck driving, loading and unloading truck, tying down load, standing, walking and carrying heavy loads |
| 11767 | 2.0 | Occupation | Truck, driving delivery truck, taxi, shuttle bus, school bus |
| 11770 | 1.3 | Occupation | Typing, electric, manual or computer |
| 11780 | 6.3 | Occupation | Using heavy power tools such as pneumatic tools (e.g., jackhammers, drills) |
| 11790 | 8.0 | Occupation | Using heavy tools (not power) such as shovel, pick, tunnel bar, spade |
| 11791 | 2.0 | Occupation | Walking on job, less than 2.0 mph, very slow speed, in office or lab area |
| 11792 | 3.5 | Occupation | Walking on job, 3.0 mph, in office, moderate speed, not carrying anything |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 11793 | 4.3 | Occupation | Walking on job, 3.5 mph, in office, brisk speed, not carrying anything |
| 11795 | 3.5 | Occupation | Walking on job, 2.5 mph, slow speed and carrying light objects less than 25 lb |
| *11796* | *3.0* | Occupation | Walking, gathering things at work, ready to leave |
| 11797 | 3.8 | Occupation | Walking, 2.5 mph, slows peed, carrying heavy objects more than 25 lb |
| 11800 | 4.5 | Occupation | Walking, 3.0 mph, moderately and carrying light objects less than 25 lb |
| 11805 | 3.5 | Occupation | Walking, pushing a wheelchair |
| *11810* | *4.8* | Occupation | Walking, 3.5 mph, briskly and carrying objects less than 25 lb |
| *11820* | *5.0* | Occupation | Walking or walk downstairs or standing, carrying objects about 25 to 49 lb |
| *11830* | *6.5* | Occupation | Walking or walk downstairs or standing, carrying objects about 50–74 lb |
| *11840* | *7.5* | Occupation | Walking or walk downstairs or standing, carrying objects about 75–99 lb |
| *11850* | *8.5* | Occupation | Walking or walk downstairs or standing, carrying objects about 100 lb or more |
| *11870* | *3.0* | Occupation | Working in scene shop, theater actor, backstage employee |
| 12010 | 6.0 | Running | Jog/walk combination (jogging component of less than 10 min) (Taylor Code 180) |
| *12020* | *7.0* | Running | Jogging, general |
| *12025* | *8.0* | Running | Jogging, in place |
| *12027* | *4.5* | Running | Jogging, on a mini-tramp |
| 12029 | 6.0 | Running | Running, 4 mph (13 min/mile) |
| 12030 | 8.3 | Running | Running, 5 mph (12 min/mile) |
| *12040* | *9.0* | Running | Running, 5.2 mph (11.5 min/mile) |
| 12050 | 9.8 | Running | Running, 6 mph (10 min/mile) |
| 12060 | 10.5 | Running | Running, 6.7 mph (9 min/mile) |
| 12070 | 11.0 | Running | Running, 7 mph (8.5 min/mile) |
| 12080 | 11.5 | Running | Running, 7.5 mph (8 min/mile) |
| 12090 | 11.8 | Running | Running, 8 mph (7.5 min/mile) |
| *12100* | *12.3* | Running | Running, 8.6 mph (7 min/mile) |
| 12110 | 12.8 | Running | Running, 9 mph (6.5 min/mile) |
| 12120 | 14.5 | Running | Running, 10 mph (6 min/mile) |
| 12130 | 16.0 | Running | Running, 11 mph (5.5 min/mile) |
| 12132 | 19.0 | Running | Running, 12 mph (5 min/mile) |
| 12134 | 19.8 | Running | Running, 13 mph (4.6 min/mile) |
| 12135 | 23.0 | Running | Running, 14 mph (4.3 min/mile) |
| 12140 | 9.0 | Running | Running, cross country |
| 12150 | 8.0 | Running | Running (Taylor code 200) |
| *12170* | *15.0* | Running | Running, stairs, up |
| *12180* | *10.0* | Running | Running, on a track, team practice |
| *12190* | *8.0* | Running | Running, training, pushing a wheelchair or baby carrier |
| 12200 | 13.3 | Running | Running, marathon |
| 13000 | 2.3 | Self care | Getting ready for bed, general, standing |
| 13009 | 1.8 | Self care | Sitting on toilet, eliminating while standing or squatting |
| *13010* | *1.5* | Self care | Bathing, sitting |
| 13020 | 2.5 | Self care | Dressing, undressing, standing or sitting |
| 13030 | 1.5 | Self care | Eating, sitting |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| *13035* | *2.0* | Self care | Talking and eating or eating only, standing |
| *13036* | *1.5* | Self care | Taking medication, sitting or standing |
| 13040 | 2.0 | Self care | Grooming, washing hands, shaving, brushing teeth, putting on make-up, sitting or standing |
| *13045* | *2.5* | Self care | Hairstyling, standing |
| *13046* | *1.3* | Self care | having Hair or nails done by someone else, sitting |
| *13050* | *2.0* | Self care | Showering, toweling off, standing |
| 14010 | 2.8 | Sexual activity | Active, vigorous effort |
| 14020 | 1.8 | Sexual activity | General, moderate effort |
| 14030 | 1.3 | Sexual activity | Passive, light effort, kissing, hugging |
| *15000* | *5.5* | Sports | Alaska Native Games, Eskimo Olympics, general |
| 15010 | 4.3 | Sports | Archery, non-hunting |
| 15020 | 7.0 | Sports | Badminton, competitive (Taylor Code 450) |
| 15030 | 5.5 | Sports | Badminton, social singles and doubles, general |
| 15040 | 8.0 | Sports | Basketball, game (Taylor Code 490) |
| 15050 | 6.0 | Sports | Basketball, non-game, general (Taylor Code 480) |
| 15055 | 6.5 | Sports | Basketball, general |
| 15060 | 7.0 | Sports | Basketball, officiating (Taylor Code 500) |
| *15070* | *4.5* | Sports | Basketball, shooting baskets |
| 15072 | 9.3 | Sports | Basketball, drills, practice |
| 15075 | 7.8 | Sports | Basketball, wheelchair |
| 15080 | 2.5 | Sports | Billiards |
| 15090 | 3.0 | Sports | Bowling (Taylor Code 390) |
| 15092 | 3.8 | Sports | Bowling, indoor, bowling alley |
| 15100 | 12.8 | Sports | Boxing, in ring, general |
| 15110 | 5.5 | Sports | Boxing, punching bag |
| 15120 | 7.8 | Sports | Boxing, sparring |
| *15130* | *7.0* | Sports | Broomball |
| 15135 | 5.8 | Sports | Children's games, adults playing (e.g., hopscotch, 4-square, dodge ball, playground apparatus, t-ball, tetherball, marbles, arcade games), moderate effort |
| *15138* | *6.0* | Sports | Cheerleading, gymnastic moves, competitive |
| *15140* | *4.0* | Sports | Coaching, football, soccer, basketball, baseball, swimming, etc. |
| 15142 | 8.0 | Sports | Coaching, actively playing sport with players |
| 15150 | 4.8 | Sports | Cricket, batting, bowling, fielding |
| 15160 | 3.3 | Sports | Croquet |
| *15170* | *4.0* | Sports | Curling |
| *15180* | *2.5* | Sports | Darts, wall or lawn |
| *15190* | *6.0* | Sports | Drag racing, pushing or driving a car |
| 15192 | 8.5 | Sports | Auto racing, open wheel |
| *15200* | *6.0* | Sports | Fencing |
| 15210 | 8.0 | Sports | Football, competitive |
| 15230 | 8.0 | Sports | Football, touch, flag, general (Taylor Code 510) |
| *15232* | *4.0* | Sports | Football, touch, flag, light effort |
| *15235* | *2.5* | Sports | Football or baseball, playing catch |
| *15240* | *3.0* | Sports | Frisbee playing, general |
| *15250* | *8.0* | Sports | Frisbee, ultimate |
| 15255 | 4.8 | Sports | Golf, general |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 15265 | 4.3 | Sports | Golf, walking, carrying clubs |
| *15270* | *3.0* | Sports | Golf, miniature, driving range |
| 15285 | 5.3 | Sports | Golf, walking, pulling clubs |
| 15290 | 3.5 | Sports | Golf, using power cart (Taylor Code 070) |
| 15300 | 3.8 | Sports | Gymnastics, general |
| *15310* | *4.0* | Sports | Hacky sack |
| 15320 | 12.0 | Sports | Handball, general (Taylor Code 520) |
| *15330* | *8.0* | Sports | Handball, team |
| 15335 | 4.0 | Sports | High ropes course, multiple elements |
| *15340* | *3.5* | Sports | Hang gliding |
| 15350 | 7.8 | Sports | Hockey, field |
| *15360* | *8.0* | Sports | Hockey, ice, general |
| *15362* | *10.0* | Sports | Hockey, ice, competitive |
| 15370 | 5.5 | Sports | Horseback riding, general |
| *15375* | *4.3* | Sports | Horse chores, feeding, watering, cleaning stalls, implied walking and lifting loads |
| 15380 | 4.5 | Sports | Saddling, cleaning, grooming, harnessing and unharnessing horse |
| 15390 | 5.8 | Sports | Horseback riding, trotting |
| 15395 | 7.3 | Sports | Horseback riding, canter or gallop |
| 15400 | 3.8 | Sports | Horseback riding, walking |
| 15402 | 9.0 | Sports | Horseback riding, jumping |
| 15408 | 1.8 | Sports | Horse cart, driving, standing or sitting |
| *15410* | *3.0* | Sports | Horseshoe pitching, quoits |
| *15420* | *12.0* | Sports | Jai alai |
| 15425 | 5.3 | Sports | Martial arts, different types, slower pace, novice performers, practice |
| 15430 | 10.3 | Sports | Martial arts, different types, moderate pace (e.g., judo, jujitsu, karate, kickboxing, taekwondo, Tae Bo, Muay Thai boxing) |
| *15440* | *4.0* | Sports | Juggling |
| *15450* | *7.0* | Sports | Kickball |
| *15460* | *8.0* | Sports | Lacrosse |
| 15465 | 3.3 | Sports | Lawn bowling, bocce ball, outdoor |
| *15470* | *4.0* | Sports | Moto-cross, off-road motor sports, all-terrain vehicle, general |
| *15480* | *9.0* | Sports | Orienteering |
| *15490* | *10.0* | Sports | Paddleball, competitive |
| 15500 | 6.0 | Sports | Paddleball, casual, general (Taylor Code 460) |
| *15510* | *8.0* | Sports | Polo, on horseback |
| *15520* | *10.0* | Sports | Racquetball, competitive |
| 15530 | 7.0 | Sports | Racquetball, general (Taylor Code 470) |
| 15533 | 8.0 | Sports | Rock or mountain climbing (Taylor Code 470)(*Formerly code = 17120*) |
| 15535 | 7.5 | Sports | Rock climbing, ascending rock, high difficulty |
| 15537 | 5.8 | Sports | Rock climbing, ascending or traversing rock, low-to-moderate difficulty |
| *15540* | *5.0* | Sports | Rock climbing, rappelling |
| *15542* | *4.0* | Sports | Rodeo sports, general, light effort |
| *15544* | *5.5* | Sports | Rodeo sports, general, moderate effort |
| *15546* | *7.0* | Sports | Rodeo sports, general, vigorous effort |
| 15550 | 12.3 | Sports | Rope jumping, fast pace, 120–160 skips/min |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 15551 | 11.8 | Sports | Rope jumping, moderate pace, 100–120 skips/min, general, 2 foot skip, plain bounce |
| 15552 | 8.8 | Sports | Rope jumping, slow pace, <100 skips/min, 2 foot skip, rhythm bounce |
| 15560 | 8.3 | Sports | Rugby, union, team, competitive |
| 15562 | 6.3 | Sports | Rugby, touch, non-competitive |
| *15570* | *3.0* | Sports | Shuffleboard |
| *15580* | *5.0* | Sports | Skateboarding, general, moderate effort |
| *15582* | *6.0* | Sports | Skateboarding, competitive, vigorous effort |
| 15590 | 7.0 | Sports | Skating, roller (Taylor Code 360) |
| 15591 | 7.5 | Sports | Rollerblading, in-line skating, 14.4 km/h (9.0 mph), recreational pace |
| 15592 | 9.8 | Sports | Rollerblading, in-line skating, 17.7 km/h (11.0 mph), moderate pace, exercise training |
| 15593 | 12.3 | Sports | Rollerblading, in-line skating, 21.0–21.7 km/h (13.0–13.6 mph), fast pace, exercise training |
| 15594 | 14.0 | Sports | Rollerblading, in-line skating, 24.0 km/h (15.0 mph), maximal effort |
| *15600* | *3.5* | Sports | Skydiving, base jumping, bungee jumping |
| 15605 | 10.0 | Sports | Soccer, competitive |
| 15610 | 7.0 | Sports | Soccer, casual, general (Taylor Code 540) |
| 15620 | 5.0 | Sports | Softball or baseball, fast or slow pitch, general (Taylor Code 440) |
| 15625 | 4.0 | Sports | Softball, practice |
| *15630* | *4.0* | Sports | Softball, officiating |
| *15640* | *6.0* | Sports | Softball, pitching |
| 15645 | 3.3 | Sports | Sports spectator, very excited, emotional, physically moving |
| 15650 | 12.0 | Sports | Squash (Taylor Code 530) |
| 15652 | 7.3 | Sports | Squash, general |
| 15660 | 4.0 | Sports | Table tennis, ping pong (Taylor Code 410) |
| 15670 | 3.0 | Sports | Tai chi, qigong, general |
| 15672 | 1.5 | Sports | Tai chi, qigong, sitting, light effort |
| 15675 | 7.3 | Sports | Tennis, general |
| 15680 | 6.0 | Sports | Tennis, doubles (Taylor Code 430) |
| 15685 | 4.5 | Sports | Tennis, doubles |
| 15690 | 8.0 | Sports | Tennis, singles (Taylor Code 420) |
| *15695* | *5.0* | Sports | Tennis, hitting balls, non-game play, moderate effort |
| *15700* | *3.5* | Sports | Trampoline, recreational |
| *15702* | *4.5* | Sports | Trampoline, competitive |
| 15710 | 4.0 | Sports | Volleyball (Taylor Code 400) |
| 15711 | 6.0 | Sports | Volleyball, competitive, in gymnasium |
| 15720 | 3.0 | Sports | Volleyball, non-competitive, 6–9 member team, general |
| *15725* | *8.0* | Sports | Volleyball, beach, in sand |
| *15730* | *6.0* | Sports | Wrestling (one match = 5 min) |
| *15731* | *7.0* | Sports | Wallyball, general |
| *15732* | *4.0* | Sports | Track and field (e.g., shot, discus, hammer throw) |
| *15733* | *6.0* | Sports | Track and field (e.g., high jump, long jump, triple jump, javelin, pole vault) |
| *15734* | *10.0* | Sports | Track and field (e.g., steeplechase, hurdles) |
| 16010 | 2.5 | Transportation | Automobile or light truck (nota semi) driving |
| 16015 | 1.3 | Transportation | Riding in a car or truck |
| 16016 | 1.3 | Transportation | Riding in a bus or train |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 16020 | 1.8 | Transportation | Flying airplane or helicopter |
| 16030 | 3.5 | Transportation | Motor scooter, motorcycle |
| 16035 | 6.3 | Transportation | Pulling rickshaw |
| *16040* | *6.0* | Transportation | Pushing plane in and out of hangar |
| 16050 | 2.5 | Transportation | Truck, semi, tractor, >1 ton, or bus, driving |
| 16060 | 3.5 | Transportation | Walking for transportation, 2.8–3.2 mph, level, moderate pace, firm surface |
| 17010 | 7.0 | Walking | Backpacking (Taylor Code 050) |
| 17012 | 7.8 | Walking | Backpacking, hiking or organized walking with a daypack |
| 17020 | 5.0 | Walking | Carrying 15 lb load (e.g., suitcase), level ground or downstairs |
| 17021 | 2.3 | Walking | Carrying 15 lb child, slow walking |
| *17025* | *8.3* | Walking | Carrying load upstairs, general |
| *17026* | *5.0* | Walking | Carrying 1–15 lb load, upstairs |
| *17027* | *6.0* | Walking | Carrying 16–24 lb load, upstairs |
| *17028* | *8.0* | Walking | Carrying 25–49 lb load, upstairs |
| *17029* | *10.0* | Walking | Carrying 50–74 lb load, upstairs |
| *17030* | *12.0* | Walking | Carrying >74 lb load, upstairs |
| *17031* | *3.5* | Walking | Loading/unloading a car, implied walking |
| 17033 | 6.3 | Walking | Climbing hills, no load |
| 17035 | 6.5 | Walking | Climbing hills with 0–9 lb load |
| 17040 | 7.3 | Walking | Climbing hills with 10–20 lb load |
| 17050 | 8.3 | Walking | Climbing hills with 21–42 lb load |
| *17060* | *9.0* | Walking | Climbing hills with 42+ lb load |
| 17070 | 3.5 | Walking | Descending stairs |
| 17080 | 6.0 | Walking | Hiking, cross country (Taylor Code 040) |
| 17082 | 5.3 | Walking | Hiking or walking at a normal pace through fields and hillsides |
| *17085* | *2.5* | Walking | Bird watching, slow walk |
| 17088 | 4.5 | Walking | Marching, moderate speed, military, no pack |
| 17090 | 8.0 | Walking | Marching rapidly, military, no pack |
| 17100 | 4.0 | Walking | Pushing or pulling stroller with child or walking with children, 2.5–3.1 mph |
| 17105 | 3.8 | Walking | Pushing a wheelchair, non-occupational |
| *17110* | *6.5* | Walking | Race walking |
| 17130 | 8.0 | Walking | Stair climbing, using or climbing up ladder (Taylor Code 030) |
| 17133 | 4.0 | Walking | Stair climbing, slow pace |
| 17134 | 8.8 | Walking | Stair climbing, fast pace |
| 17140 | 5.0 | Walking | Using crutches |
| *17150* | *2.0* | Walking | Walking, household |
| 17151 | 2.0 | Walking | Walking, less than 2.0 mph, level, strolling, very slow |
| 17152 | 2.8 | Walking | Walking, 2.0 mph, level, slow pace, firm surface |
| 17160 | 3.5 | Walking | Walking for pleasure (Taylor Code 010) |
| *17161* | *2.5* | Walking | Walking from house to car or bus, from car or bus to go places, from car or bus to and from the worksite |
| *17162* | *2.5* | Walking | Walking to neighbor's house or family's house for social reasons |
| *17165* | *3.0* | Walking | Walking the dog |
| 17170 | 3.0 | Walking | Walking, 2.5 mph, level, firm surface |
| 17180 | 3.3 | Walking | Walking, 2.5 mph, downhill |
| 17190 | 3.5 | Walking | Walking, 2.8–3.2 mph, level, moderate pace, firm surface |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 17200 | 4.3 | Walking | Walking, 3.5 mph, level, brisk, firm surface, walking for exercise |
| 17210 | 5.3 | Walking | Walking, 2.9–3.5 mph, uphill, 1–5 % grade |
| 17211 | 8.0 | Walking | Walking, 2.9–3.5 mph, uphill, 6–15 % grade |
| 17220 | 5.0 | Walking | Walking, 4.0 mph, level, firm surface, very brisk pace |
| 17230 | 7.0 | Walking | Walking, 4.5 mph, level, firm surface, very, very brisk |
| 17231 | 8.3 | Walking | Walking, 5.0 mph, level, firm surface |
| 17235 | 9.8 | Walking | Walking, 5.0 mph, uphill, 3 % grade |
| 17250 | 3.5 | Walking | Walking, for pleasure, work break |
| 17260 | 4.8 | Walking | Walking, grass track |
| 17262 | 4.5 | Walking | Walking, normal pace, plowed field or sand |
| 17270 | 4.0 | Walking | Walking, to work or class (Taylor Code 015) |
| *17280* | *2.5* | Walking | Walking, to and from an outhouse |
| 17302 | 4.8 | Walking | Walking, for exercise, 3.5–4 mph, with ski poles, Nordic walking, level, moderate pace |
| 17305 | 9.5 | Walking | Walking, for exercise, 5.0 mph, with ski poles, Nordic walking, level, fast pace |
| *17310* | *6.8* | Walking | Walking, for exercise, with ski poles, Nordic walking, uphill |
| 17320 | 6.0 | Walking | Walking, backwards, 3.5 mph, level |
| 17325 | 8.0 | Walking | Walking, backwards, 3.5 mph, uphill, 5 % grade |
| *18010* | *2.5* | Water activities | Boating, power, driving |
| *18012* | *1.3* | Water activities | Boating, power, passenger, light |
| 18020 | 4.0 | Water activities | Canoeing, on camping trip (Taylor Code 270) |
| 18025 | 3.3 | Water activities | Canoeing, harvesting wild rice, knocking rice off the stalks |
| *18030* | *7.0* | Water activities | Canoeing, portaging |
| 18040 | 2.8 | Water activities | Canoeing, rowing, 2.0–3.9 mph, light effort |
| 18050 | 5.8 | Water activities | Canoeing, rowing, 4.0–5.9 mph, moderate effort |
| 18060 | 12.5 | Water activities | Canoeing, rowing, kayaking, competition, >6 mph, vigorous effort |
| 18070 | 3.5 | Water activities | Canoeing, rowing, for pleasure, general (Taylor Code 250) |
| 18080 | 12.0 | Water activities | Canoeing, rowing, in competition, or crew or sculling (Taylor Code 260) |
| *18090* | *3.0* | Water activities | Diving, springboard or platform |
| 18100 | 5.0 | Water activities | Kayaking, moderate effort |
| 18110 | 4.0 | Water activities | Paddleboat |
| 18120 | 3.0 | Water activities | Sailing, boat and board sailing, windsurfing, ice sailing, general (Taylor Code 235) |
| 18130 | 4.5 | Water activities | Sailing, in competition |
| 18140 | 3.3 | Water activities | Sailing, Sunfish/Laser/Hobby Cat, Keelboats, ocean sailing, yachting, leisure |
| 18150 | 6.0 | Water activities | Skiing, water or wakeboarding (Taylor Code 220) |
| 18160 | 7.0 | Water activities | Jet skiing, driving, in water |
| 18180 | 15.8 | Water activities | Skin diving, fast |
| 18190 | 11.8 | Water activities | Skin diving, moderate |
| 18200 | 7.0 | Water activities | Skin diving, scuba diving, general (Taylor Code 310) |
| 18210 | 5.0 | Water activities | Snorkeling (Taylor Code 310) |
| *18220* | *3.0* | Water activities | Surfing, body or board, general |
| *18222* | *5.0* | Water activities | Surfing, body or board, competitive |
| *18225* | *6.0* | Water activities | Paddle boarding, standing |
| 18230 | 9.8 | Water activities | Swimming laps, freestyle, fast, vigorous effort |
| 18240 | 5.8 | Water activities | Swimming laps, freestyle, front crawl, slow, light or moderate effort |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 18250 | 9.5 | Water activities | Swimming, backstroke, general, training or competition |
| 18255 | 4.8 | Water activities | Swimming, backstroke, recreational |
| 18260 | 10.3 | Water activities | Swimming, breaststroke, general, training or competition |
| 18265 | 5.3 | Water activities | Swimming, breaststroke, recreational |
| 18270 | 13.8 | Water activities | Swimming, butterfly, general |
| 18280 | 10.0 | Water activities | Swimming, crawl, fast speed, ~75 yards/min, vigorous effort |
| 18290 | 8.3 | Water activities | Swimming, crawl, medium speed, ~50 yards/min, vigorous effort |
| 18300 | 6.0 | Water activities | Swimming, lake, ocean, river (Taylor Codes 280, 295) |
| *18310* | *6.0* | Water activities | Swimming, leisurely, not lap swimming, general |
| 18320 | 7.0 | Water activities | Swimming, sidestroke, general |
| *18330* | *8.0* | Water activities | Swimming, synchronized |
| 18340 | 9.8 | Water activities | Swimming, treading water, fast, vigorous effort |
| 18350 | 3.5 | Water activities | Swimming, treading water, moderate effort, general |
| *18352* | *2.3* | Water activities | Tubing, floating on a river, general |
| 18355 | 5.5 | Water activities | Water aerobics, water calisthenics |
| *18360* | *10.0* | Water activities | Water polo |
| *18365* | *3.0* | Water activities | Water volleyball |
| 18366 | 9.8 | Water activities | Water jogging |
| 18367 | 2.5 | Water activities | Water walking, light effort, slow pace |
| 18368 | 4.5 | Water activities | Water walking, moderate effort, moderate pace |
| 18369 | 6.8 | Water activities | Water walking, vigorous effort, brisk pace |
| *18370* | *5.0* | Water activities | Whitewater rafting, kayaking, or canoeing |
| 18380 | 5.0 | Water activities | Windsurfing, not pumping for speed |
| 18385 | 11.0 | Water activities | Windsurfing or kite surfing, crossing trial |
| 18390 | 13.5 | Water activities | Windsurfing, competition, pumping for speed |
| *19005* | *7.5* | Winter activities | Dog sledding, mushing |
| *19006* | *2.5* | Winter activities | Dog sledding, passenger |
| *19010* | *6.0* | Winter activities | Moving ice house, set up/drill holes |
| *19011* | *2.0* | Winter activities | Ice fishing, sitting |
| 19018 | 14.0 | Winter activities | Skating, ice dancing |
| *19020* | *5.5* | Winter activities | Skating, ice, 9 mph or less |
| 19030 | 7.0 | Winter activities | Skating, ice, general (Taylor Code 360) |
| *19040* | *9.0* | Winter activities | Skating, ice, rapidly, more than 9 mph, not competitive |
| 19050 | 13.3 | Winter activities | Skating, speed, competitive |
| *19060* | *7.0* | Winter activities | Ski jumping, climb up carrying skis |
| *19075* | *7.0* | Winter activities | Skiing, general |
| 19080 | 6.8 | Winter activities | Skiing, cross country, 2.5 mph, slow or light effort, ski walking |
| 19090 | 9.0 | Winter activities | Skiing, cross country, 4.0–4.9 mph, moderate speed and effort, general |
| 19100 | 12.5 | Winter activities | Skiing, cross country, 5.0–7.9 mph, brisk speed, vigorous effort |
| 19110 | 15.0 | Winter activities | Skiing, cross country, >8.0 mph, elite skier, racing |
| 19130 | 15.5 | Winter activities | Skiing, cross country, hard snow, uphill, maximum, snow mountaineering |
| 19135 | 13.3 | Winter activities | Skiing, cross-country, skating |
| 19140 | 13.5 | Winter activities | Skiing, cross-country, biathlon, skating technique |
| 19150 | 4.3 | Winter activities | Skiing, downhill, alpine or snowboarding, light effort, active time only |
| 19160 | 5.3 | Winter activities | Skiing, downhill, alpine or snowboarding, moderate effort, general, active time only |
| *19170* | *8.0* | Winter activities | Skiing, downhill, vigorous effort, racing |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 19175 | 12.5 | Winter activities | Skiing, roller, elite racers |
| 19180 | 7.0 | Winter activities | Sledding, tobogganing, bobsledding, luge (Taylor Code 370) |
| 19190 | 5.3 | Winter activities | Snowshoeing, moderate effort |
| 19192 | 10.0 | Winter activities | Snowshoeing, vigorous effort |
| *19200* | *3.5* | Winter activities | Snowmobiling, driving, moderate |
| *19202* | *2.0* | Winter activities | Snowmobiling, passenger |
| 19252 | 5.3 | Winter activities | Snow shoveling, by hand, moderate effort |
| 19254 | 7.5 | Winter activities | Snow shoveling, by hand, vigorous effort |
| 19260 | 2.5 | Winter activities | Snow blower, walking and pushing |
| 20000 | 1.3 | Religious activities | Sitting in church, in service, attending a ceremony, sitting quietly |
| 20001 | 2.0 | Religious activities | Sitting, playing an instrument at church |
| 20005 | 1.8 | Religious activities | Sitting in church, talking or singing, attending a ceremony, sitting, active participation |
| 20010 | 1.3 | Religious activities | Sitting, reading religious materials at home |
| 20015 | 1.3 | Religious activities | Standing quietly in church, attending a ceremony |
| *20020* | *2.0* | Religious activities | Standing, singing in church, attending a ceremony, standing, active participation |
| *20025* | *1.3* | Religious activities | Kneeling in church or at home, praying |
| 20030 | 1.8 | Religious activities | Standing, talking in church |
| 20035 | 2.0 | Religious activities | Walking in church |
| 20036 | 2.0 | Religious activities | Walking, less than 2.0 mph, very slow |
| 20037 | 3.5 | Religious activities | Walking, 3.0 mph, moderate speed, not carrying anything |
| 20038 | 4.3 | Religious activities | Walking, 3.5 mph, brisk speed, not carrying anything |
| *20039* | *2.0* | Religious activities | Walk–stand combination for religious purposes, usher |
| *20040* | *5.0* | Religious activities | Praise with dance or run, spiritual dancing in church |
| 20045 | 2.5 | Religious activities | Serving food at church |
| 20046 | 2.0 | Religious activities | Preparing food at church |
| 20047 | 3.3 | Religious activities | Washing dishes, cleaning kitchen at church |
| 20050 | 1.5 | Religious activities | Eating at church |
| 20055 | 2.0 | Religious activities | Eating/talking at church or standing eating, American Indian Feast days |
| 20060 | 3.3 | Religious activities | Cleaning church |
| 20061 | 4.0 | Religious activities | General yard work at church |
| 20065 | 3.5 | Religious activities | Standing, moderate effort (e.g., lifting heavy objects, assembling at fast rate) |
| 20095 | 4.5 | Religious activities | Standing, moderate-to-heavy effort, manual labor, lifting ≥50 lb, heavy maintenance |
| 20100 | 1.3 | Religious activities | Typing, electric, manual, or computer |
| 21000 | 1.5 | Volunteer activities | Sitting, meeting, general, and/or with talking involved |
| 21005 | 1.5 | Volunteer activities | Sitting, light office work, in general |
| 21010 | 2.5 | Volunteer activities | Sitting, moderate work |
| 21015 | 2.3 | Volunteer activities | Standing, light work (filing, talking, assembling) |
| 21016 | 2.0 | Volunteer activities | Sitting, child care, only active periods |
| 21017 | 3.0 | Volunteer activities | Standing, child care, only active periods |
| 21018 | 3.5 | Volunteer activities | Walk/run play with children, moderate, only active periods |
| 21019 | 5.8 | Volunteer activities | Walk/run play with children, vigorous, only active periods |
| 21020 | 3.0 | Volunteer activities | Standing, light/moderate work (e.g., pack boxes, assemble/repair, setup chairs/furniture) |

(continued)

**Appendix 1** (continued)

| Code | METs | Major heading | Specific activities |
|---|---|---|---|
| 21025 | 3.5 | Volunteer activities | Standing, moderate (lifting 50 lb., assembling at fast rate) |
| 21030 | 4.5 | Volunteer activities | Standing, moderate/heavy work |
| 21035 | 1.3 | Volunteer activities | Typing, electric, manual, or computer |
| 21040 | 2.0 | Volunteer activities | Walking, less than 2.0 mph, very slow |
| 21045 | 3.5 | Volunteer activities | Walking, 3.0 mph, moderate speed, not carrying anything |
| 21050 | 4.3 | Volunteer activities | Walking, 3.5 mph, brisk speed, not carrying anything |
| 21055 | 3.5 | Volunteer activities | Walking, 2.5 mph slowly and carrying objects less than 25 lb |
| 21060 | 4.5 | Volunteer activities | Walking, 3.0 mph moderately and carrying objects less than 25 lb, pushing something |
| 21065 | 4.8 | Volunteer activities | Walking, 3.5 mph, briskly and carrying objects less than 25 lb |
| 21070 | 3.0 | Volunteer activities | Walk–stand combination, for volunteer purposes |

Italicized codes and METs are estimated values

## References


1. FAO/WHO/UNU. Human energy requirements. Report of a joint FAO/WHO/UNU expert consultation. Rome: FAO; 2004.
2. (U.S.) IoM. Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for energy carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. Washington, DC: National Academies Press; 2002.
3. Garby L, Kurzer MS, Lammert O, Nielsen E. Energy expenditure during sleep in men and women: evaporative and sensible heat losses. Hum Nutr Clin Nutr. 1987;41(3):225–33.
4. Rolfe DF, Brown GC. Cellular energy utilization and molecular origin of standard metabolic rate in mammals. Physiol Rev. 1997;77(3):731–58.
5. Fray CD, Johnson RK. Energy. In: Mahan LK, Escott-Stump S, editors. Krause's food, nutrition, & diet therapy. Philadelphia, PA: Saunders; 2004. p. 21–36.
6. Ainsworth BE, Haskell WL, Herrmann SD, et al. 2011 Compendium of Physical Activities: a second update of codes and MET values. Med Sci Sports Exerc. 2011;43(8):1575–81.
7. Ainsworth B, Haskell W, Herrmann S, etal. The compendium of physical activities tracking guide. 2013. https://sites.google.com/site/compendiumofphysicalactivities/. Accessed 24 May, 2013.
8. FAO/WHO/UNU. Energy and protein requirements. Report of a joint FAO/WHO/UNU expert consultation. Geneva: WHO; 1985.
9. Poehlman ET. Regulation of energy expenditure in aging humans. J Am Geriatr Soc. 1993;41(5):552–9.
10. Bogardus C, Lillioja S, Ravussin E, et al. Familial dependence of the resting metabolic rate. N Engl J Med. 1986;315(2):96–100.
11. Torun B, Davies PS, Livingstone MB, Paolisso M, Sackett R, Spurr GB. Energy requirements and dietary energy recommendations for children and adolescents 1 to 18 years old. Eur J Clin Nutr. 1996;50 Suppl 1:S37–80. discussion S80–31.
12. Henry CJ. Mechanisms of changes in basal metabolism during ageing. Eur J Clin Nutr. 2000;54 Suppl 3:S77–91.
13. Webb GD, Poehlman ET, Tonino RP. Dissociation of changes in metabolic rate and blood pressure with erythrocyte Na-K pump activity in older men after endurance training. J Gerontol. 1993;48(2):M47–52.
14. Caspersen CJ, Powell KE, Christenson GM. Physical activity, exercise, and physical fitness: definitions and distinctions for health-related research. Public Health Rep. 1985;100(2):126–31.
15. Schoeller DA. Measurement of energy expenditure in free-living humans by using doubly labeled water. J Nutr. 1988;118(11):1278–89.
16. Bouten CV, Verboeket-van de Venne WP, Westerterp KR, Verduin M, Janssen JD. Daily physical activity assessment: comparison between movement registration and doubly labeled water. J Appl Physiol. 1996;81(2):1019–26.
17. Plasqui G, Westerterp KR. Physical activity assessment with accelerometers: an evaluation against doubly labeled water. Obesity (Silver Spring). 2007;15(10):2371–9.
18. Leenders NY, Sherman WM, Nagaraja HN. Energy expenditure estimated by accelerometry and doubly labeled water: do they agree? Med Sci Sports Exerc. 2006;38(12):2165–72.
19. Levine JA. Measurement of energy expenditure. Public Health Nutr. 2005;8(7A):1123–32.
20. van Poppel MN, Chinapaw MJ, Mokkink LB, van Mechelen W, Terwee CB. Physical activity questionnaires for adults: a systematic review of measurement properties. Sports Med. 2010;40(7):565–600.
21. Craig CL, Marshall AL, Sjostrom M, et al. International physical activity questionnaire: 12-country reliability and validity. Med Sci Sports Exerc. 2003;35(8):1381–95.

22. Bonn SE, Surkan PJ, Lagerros YT, Balter K. Feasibility of a novel web-based physical activity questionnaire for young children. Pediatr Rep. 2012;4(4):e37.
23. Harris JA, Benedict FG. A biometric study of human basal metabolism. Proc Natl Acad Sci U S A. 1918; 4(12):370–3.
24. Daly JM, Heymsfield SB, Head CA, et al. Human energy requirements: overestimation by widely used prediction equation. Am J Clin Nutr. 1985;42(6):1170–4.
25. Hasson RE, Howe CA, Jones BL, Freedson PS. Accuracy of four resting metabolic rate prediction equations: effects of sex, body mass index, age, and race/ethnicity. J Sci Med Sport. 2011;14(4):344–51.
26. Roza AM, Shizgal HM. The Harris Benedict equation reevaluated: resting energy requirements and the body cell mass. Am J Clin Nutr. 1984;40(1):168–82.
27. Henry CJ. Basal metabolic rate studies in humans: measurement and development of new equations. Public Health Nutr. 2005;8(7A):1133–52.
28. Frankenfield D, Roth-Yousey L, Compher C. Comparison of predictive equations for resting metabolic rate in healthy nonobese and obese adults: a systematic review. J Am Diet Assoc. 2005; 105(5):775–89.
29. Mifflin MD, St Jeor ST, Hill LA, Scott BJ, Daugherty SA, Koh YO. A new predictive equation for resting energy expenditure in healthy individuals. Am J Clin Nutr. 1990;51(2):241–7.
30. Weekes C. Controversies in the determination of energy requirements. Proc Nutri Soc. 2007;66: 367–77.
31. Ravussin E, Lillioja S, Knowler WC, et al. Reduced rate of energy expenditure as a risk factor for body-weight gain. N Engl J Med. 1988;318(8):467–72.
32. da Rocha EE, Alves VG, da Fonseca RB. Indirect calorimetry: methodology, instruments and clinical application. Curr Opin Clin Nutr Metab Care. 2006; 9(3):247–56.
33. Ferrannini E. The theoretical bases of indirect calorimetry: a review. Metabolism. 1988;37(3):287–301.
34. Schofield WN. Predicting basal metabolic rate, new standards and review of previous work. Hum Nutr Clin Nutr. 1985;39 Suppl 1:5–41.
35. Wang Z, Heshka S, Gallagher D, Boozer CN, Kotler DP, Heymsfield SB. Resting energy expenditure-fat-free mass relationship: new insights provided by body composition modeling. Am J Physiol Endocrinol Metab. 2000;279(3):E539–45.
36. Arciero PJ, Goran MI, Poehlman ET. Resting metabolic rate is lower in women than in men. J Appl Physiol. 1993;75(6):2514–20.
37. Wang Z, Heshka S, Zhang K, Boozer CN, Heymsfield SB. Resting energy expenditure: systematic organization and critique of prediction methods. Obes Res. 2001;9(5):331–6.
38. Muller MJ, Bosy-Westphal A, Kutzner D, Heller M. Metabolically active components of fat-free mass and resting energy expenditure in humans: recent lessons from imaging technologies. Obes Rev. 2002;3(2): 113–22.
39. Bosy-Westphal A, Reinecke U, Schlorke T, et al. Effect of organ and tissue masses on resting energy expenditure in underweight, normal weight and obese adults. Int J Obes Relat Metab Disord. 2004; 28(1):72–9.
40. Heymsfield SB, Gallagher D, Kotler DP, Wang Z, Allison DB, Heshka S. Body-size dependence of resting energy expenditure can be attributed to nonenergetic homogeneity of fat-free mass. Am J Physiol Endocrinol Metab. 2002;282(1):E132–8.
41. Foster GD, Wadden TA, Mullen JL, et al. Resting energy expenditure, body composition, and excess weight in the obese. Metabolism. 1988;37(5):467–72.
42. Stiegler P, Cunliffe A. The role of diet and exercise for the maintenance of fat-free mass and resting metabolic rate during weight loss. Sports Med. 2006;36(3):239–62.
43. Lowell BB, Spiegelman BM. Towards a molecular understanding of adaptive thermogenesis. Nature. 2000;404(6778):652–60.
44. Bangsbo J, Krustrup P, Gonzalez-Alonso J, Saltin B. ATP production and efficiency of human skeletal muscle during intense exercise: effect of previous exercise. Am J Physiol Endocrinol Metab. 2001;280(6): E956–64.
45. van Marken Lichtenbelt WD, Vanhommerig JW, Smulders NM, et al. Cold-activated brown adipose tissue in healthy men. N Engl J Med. 2009;360(15):1500–8.
46. Seale P, Bjork B, Yang W, et al. PRDM16 controls a brown fat/skeletal muscle switch. Nature. 2008;454(7207):961–7.
47. Wijers SL, Saris WH, van Marken Lichtenbelt WD. Recent advances in adaptive thermogenesis: potential implications for the treatment of obesity. Obes Rev. 2009;10(2):218–26.
48. Hunter GR, Byrne NM, Sirikul B, et al. Resistance training conserves fat-free mass and resting energy expenditure following weight loss. Obesity (Silver Spring). 2008;16(5):1045–51.
49. Leibel RL, Rosenbaum M, Hirsch J. Changes in energy expenditure resulting from altered body weight. N Engl J Med. 1995;332(10):621–8.
50. Schoeller DA. The energy balance equation: looking back and looking forward are two very different views. Nutr Rev. 2009;67(5):249–54.
51. Major GC, Doucet E, Trayhurn P, Astrup A, Tremblay A. Clinical significance of adaptive thermogenesis. Int J Obes (Lond). 2007;31(2):204–12.
52. Heshka S, Yang MU, Wang J, Burt P, Pi-Sunyer FX. Weight loss and change in resting metabolic rate. Am J Clin Nutr. 1990;52(6):981–6.
53. Byrne NM, Weinsier RL, Hunter GR, et al. Influence of distribution of lean body mass on resting metabolic rate after weight loss and weight regain: comparison of responses in white and black women. Am J Clin Nutr. 2003;77(6):1368–73.
54. Dulloo AG. Suppressed thermogenesis as a cause for resistance to slimming and obesity rebound: adaptation or illusion? Int J Obes (Lond). 2007; 31(2):201–3.

55. Speakman JR, Selman C. Physical activity and resting metabolic rate. Proc Nutr Soc. 2003;62(3):621–34.
56. Doucet E, St-Pierre S, Almeras N, Despres JP, Bouchard C, Tremblay A. Evidence for the existence of adaptive thermogenesis during weight loss. Br J Nutr. 2001;85(6):715–23.
57. Levine JA. Nonexercise activity thermogenesis (NEAT): environment and biology. Am J Physiol Endocrinol Metab. 2004;286(5):E675–85.
58. Levine JA, Kotz CM. NEAT-non-exercise activity thermogenesis-egocentric & geocentric environmental factors vs. biological regulation. Acta Physiol Scand. 2005;184(4):309–18.
59. Kotz CM, Teske JA, Billington CJ. Neuroregulation of nonexercise activity thermogenesis and obesity resistance. Am J Physiol Regul Integr Comp Physiol. 2008;294(3):R699–710.
60. Saris WH, Blair SN, van Baak MA, et al. How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement. Obes Rev. 2003;4(2):101–14.
61. Goldberg GR, Black AE, Jebb SA, et al. Critical evaluation of energy intake data using fundamental principles of energy physiology: 1. Derivation of cut-off limits to identify under-recording. Eur J Clin Nutr. 1991;45(12):569–81.
62. Okubo H, Sasaki S. Underreporting of energy intake among Japanese women aged 18-20 years and its association with reported nutrient and food group intakes. Public Health Nutr. 2004;7(7):911–7.
63. Smith DA, Dollman J, Withers RT, et al. Relationship between maximum aerobic power and resting metabolic rate in young adult women. J Appl Physiol. 1997;82:156–63.
64. Bosy-Westphal A, et al. Grade of adiposity affects the impact of fat mass on resting energy expenditure in women. Br J Nutr. 2009;101:474–7.

# Nutritional Guidelines for Active Children

28

Shelby D. Kloiber and Eduardo Iglesias-Gutiérrez

**Abstract**

Nutrition is a large component of participating in a healthy lifestyle. Understanding the guidelines of proper nutrition set forth by the United States Department of Agriculture (USDA) can help a person maintain adequate nutrition. Dietary recommendations have been set for adults as well as children. These recommendations, when met, can be beneficial to one's health. Stemming away from dietary guidelines can result in nutrition-related chronic diseases including but not limited to obesity, diabetes, cardiovascular disease, and some forms of cancer. Appropriate nutrition is vital for proper growth and development in children and adolescents. Creating a lifestyle with physical activity can also have many benefits. According to the Dietary Guidelines for Americans, the dietary reference intake offers recommendations of macronutrients, minerals, and vitamins to consume on a daily basis. Recently the USDA has changed the icon to a serving plate that shows through visual cues how much of each food group you should consume. The MyPlate icon shows the five major food groups that are the building blocks of a healthy diet. Fruits, vegetables, grains, protein foods, and dairy are the food groups individuals should choose from, and the choosemyplate.gov website explains each group and shows healthy choices and the recommendations or serving sizes within each group. Teaching children the importance of eating proper nutrition can lead to a healthy lifestyle. It is suggested that our daily meal consists of half of our plate full of fruits and vegetables, along with lean meats, whole grains, and fat-free and/or low-fat dairy products. Limiting our intake of saturated fats, trans-fatty acids, and simple sugars is important as well.

S.D. Kloiber, M.Sc. (✉)
Department of Health, Exercise, and Sport Sciences,
Texas Tech University, Lubbock, TX, USA
e-mail: shelby.kloiber@ttu.edu

E. Iglesias-Gutiérrez, B.Sc., Ph.D., M.Sc.
Department of Funcational Biology (Physiology),
University of Oviedo, C/Julian Claveria s/n,
Oviedo, Asturias, Spain
e-mail: iglesiaseduardo@uniovi.es

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_28, 

**Keywords**

Child • Adolescent • Nutrition • Physical activity • Recommendations • Myplate

## 28.1 Learning Objectives

After completion of this chapter, you should understand the following:

1. Dietary Guidelines for Americans
2. MyPlate Food Guidance System: main food groups and foods found in each of these groups
3. Child recommendations for daily calorie intake
4. The role and source of energy of macronutrients: carbohydrates, proteins, and fats
5. Importance to incorporate micronutrients: vitamins, minerals, fiber, and water into a child's diet

## 28.2 Introduction

Nutrition is a large component of participating in a healthy lifestyle. Understanding the guidelines of proper nutrition set forth by the United States Department of Agriculture (USDA) can help a person attain adequate nutrition. Dietary recommendations have been set for adults as well as children. These recommendations, when met, have been demonstrated to be beneficial to one's health. Not following these dietary guidelines can result in nutrition-related chronic diseases (NRCDs). NRCDs include but are not limited to obesity, diabetes, cardiovascular disease, and some forms of cancer [1]. Appropriate nutrition is vital for proper growth and development in children and adolescents. Improper nutrition may lead to permanent and irreversible impairments in brain development, behavior changes and delayed psychomotor development, altered immune function, decreased levels of physical activity, decreased social interaction and curiosity, and decreased cognitive functioning [2]. Portion sizes of processed and ready-to-eat foods and also the portions of junk food have increased over the years. This is one of the contributing factors leading to the increase in overweight and obese children and adolescents. An increase in energy intake above the recommended amount and/or no physical activity can lead to changes in body composition. Although it is difficult to determine appropriate portion sizes for children, data suggests that reducing portion sizes may be an effective strategy for decreasing energy intake [3].

Another large component within a healthy lifestyle is physical activity. An increase in moderate physical activity is an important goal for reducing health problems [4]. Along with physiological and psychological benefits, engaging in physical activity at a young age can lead to continued activity into adulthood. For those children who participate, competitive sports and school-based physical education are good predictors of later physical activity. Creating a lifestyle with physical activity can have many benefits, and the recommendations for adults and children change to reflect the ever-changing culture and progressive research. Recommendations for children (aged 5–16) include 60 min of at least moderate-intensity physical activity per day, including vigorous-intensity aerobic activities that can improve bone density and muscle strength [4].

## 28.3 Research Findings

### 28.3.1 Dietary Guidelines for Americans

According to the US Department of Agriculture and the US Department of Health and Human Services (USHHS), the 2010 Dietary Guidelines for Americans is based on the most up-to-date scientific evidence. The guidelines provide advice on choosing a healthy eating pattern and are intended to be used as educational material for policymakers, for the general public, and for specific audiences such as children [5, 6]. Approximately 32 % of children and adolescents ages 2–19 years are overweight or obese. The

prevalence of chronic diseases once found in adults are now being seen in younger ages [7]. The Dietary Guidelines for Americans, which focuses on health promotion and disease risk reduction, is reviewed and updated every 5 years. Every updated edition goes through three vital steps to ensure accurate information. Stage one brings about a scientific approach where scientists and researchers conduct analysis on new scientific information on health and fitness. The second stage targets policymakers, nutrition educators, health-care providers, etc. The final stage prepares information to be presented to the general public [4].

According to the Dietary Guidelines for Americans, the dietary reference intake offers recommendations of macronutrients, minerals, and vitamins to consume on a daily basis. Dietary reference intake (DRI) offers recommendations with considerations of life stage, gender, and activity level. An example of recommended macronutrient proportions by age are seen in Appendix 1.

### 28.3.2 Macronutrients

The three macronutrients involved in a healthy diet include carbohydrates, proteins, and fats. Carbohydrates are the primary energy source for our body and are crucial for red blood cells and neurons. Carbohydrates contain 4 cal/1 g and include sugars, starches, and most of the compounds considered as fiber. The main food sources of carbohydrates include grains, beans and peas, vegetables, and fruits. For all children above ages 1 year old, it is recommended that 45–65 % of their daily calories come from carbohydrates. Those foods that contain functional fiber should be consumed as they have been found to have beneficial physiological effects in humans. Whole grains may reduce the risk for heart disease, reduce constipation, and help maintain with weight management. Over-consumption of those carbohydrates that are made up of simple sugars and have little nutritional value can lead to weight gain and should be avoided for those engaging in a healthy diet [6].

The second of the three macronutrients is protein. One gram of protein yields 4 cal of energy and is the major functional and structural component of all cells within the body. Protein, which is not a primary energy source, is composed of amino acids which are necessary for growth and repair of our body's tissue. There are 21 amino acids that can form proteins in our body, eight of which (nine for young children) humans cannot synthesize to meet the needs of our body. It is important when choosing proteins that you consume those of high quality to acquire all of the necessary amino acids. High-quality proteins are those that provide all the amino acids that our body needs in the correct amount. They can be found mainly in animal foods like eggs and meat of fish, but also when combining other foods, like cereals and legumes or dairy products and cereals.

Recommended daily consumption of protein for children ages 1–3 years old is 5–20 % of total daily calories. Children ages 4–18 years old can increase their protein consumption to 10–30 % of their daily caloric intake. A variety of proteins is important for a healthy diet. Examples of foods with a high protein content include lean meats and poultry, seafood, beans and peas, soy products, nuts and seeds [6, 8].

Fats, the third of the macronutrients, are categorized into monounsaturated fatty acids, polyunsaturated fatty acids, saturated fatty acids, and trans-fatty acids. One gram of fat yields 9 cal of energy and should make up 30–40 % of the daily calories consumed for children ages 1–3 years old. Children 4–18 years of age should consume 25–35 % of their total daily calories as fat. Although fats should be consumed in moderation, it is an important nutrient needed in an overall healthy diet. Fat provides structure to the body's tissue, nerves, and cell membranes. During exercise, depending on its duration and intensity, your body also utilizes fat as energy source [6, 9]. MUFAs can be found in foods such as nuts, olive oil, and some pork derivatives. PUFAs can be chemically classified into several groups. From a nutritional point of view, the two main subgroups are omega-6 and omega-3 fatty acids. Both omega-6 and omega-3 fatty acids cannot be synthesized in the body and can be

found in foods such as liquid vegetable oils, flaxseed, and some fish and shellfish. It is recommended to replace saturated fatty acids with MUFAs and PUFAs. Saturated fats are found in palm and coconut oil and in animal products including meat and milk and also in fish. In fact, most fish containing high PUFA (much higher than meat) have even higher SFA content, similar to meat. A diet high in saturated fat can increase blood cholesterol level. As we said before, increased LDL due to a diet high in SFA and cholesterol and an elevated sedentarism may increase risk factors for certain diseases such as cardiovascular disease. It is recommended to consume less than 10 % of saturated fats in your daily fat intake and less than 300 mg of cholesterol per day. Trans-fatty acids, a form of unsaturated fatty acids, can be found in shortening, commercially prepared baked foods, fried foods, and snack foods. A diet high in trans-fatty acids may lead to an increase risk for heart disease. Most Americans consume too much saturated and trans-fatty acids and not enough unsaturated fatty acids [6].

### 28.3.3 Micronutrients

Vitamins do not provide energy, but are essential to at least one vital process within the human body. Vitamins can be divided into two groups: fat soluble and water soluble. Fat-soluble vitamins can be stored in the body and are known as vitamin A, D, E, and K. Food sources for fat-soluble vitamins include dairy products, dark green leafy vegetables, yellow vegetables, whole grains, legumes, fortified milk, and egg yolks. Eating fruits and vegetables with a variety of color can increase the availability of different vitamins. Water-soluble vitamins, including B vitamins and vitamin C, are involved in energy metabolism and maintenance of bone, cartilage, and connective tissue. Food sources that contain water-soluble vitamins include but are not limited to eggs, meat, poultry, milk products, beans, nuts, cereals, and vegetables. Vitamin recommendations for children are found in Appendix 1 [6].

Minerals are an important part of our daily diet. Some minerals are required to support human biochemical processes by serving structural and functional roles as well as electrolytes. Appropriate levels of these minerals have shown to be required to maintain proper health. Recommendations for daily mineral consumption for children are listed in Appendix 1.

Examples of food sources rich in minerals include dairy products, green leafy vegetables, salmon, sardines, and spinach [6]. In this sense it should be taken into account that the bioavailability of some minerals in some of this foods is extremely low, which make them poor sources of minerals, although their content is high. This happens, for example, with calcium and spinach.

Including fiber into a diet can aid in digestion and other beneficial physiological functions. Dietary fiber cannot be digested so it passes through the small intestine into the colon where it helps maintain regularity and bowel health. Insoluble fiber does not dissolve in water so it helps move material through the colon. This is helpful for those children who suffer from constipation. Diets high in insoluble fiber may decrease the risk of diabetes and can be found in foods such as whole grains, nuts, wheat bran, and vegetables. Soluble fiber absorbs water and can help soften stool. Foods rich in soluble fiber include oats, apples, flaxseed, and legumes. Appendix 1 shows the daily recommendation for fiber consumption in children [6].

Water is approximately 50–70 % of an adult's body weight, and as high as 79 % of children's weight, and essential for life. Water helps regulate body temperature, lubrication, and transportation within the body. Dehydration can lead to heat injuries including cramps, heat exhaustion, and/or heat stroke. Hydration can be met with adequate fluid intake as well as fluid found in some foods. On average consuming 64 ounces per day is necessary for your body's needs and will increase as your physical activity increases. There is not specific requirement level for water intake because it varies for each individual; however, the combination of thirst and drinking beverages with meals can provide your body with the fluid levels needed [6].

### 28.3.4 Nutritional Responsibility

As a parent or caregiver, it is important to provide a foundation of healthy eating patterns and regular physical activity for children. The lifestyle children engage in can influence their lifestyle as an adult. Studies show those who engage in an unhealthy lifestyle are more at risk for becoming obese as an adult [2] and suffer from many other metabolic diseases, like type 2 diabetes. Strategies that can create a healthy lifestyle include but are not limited to ensuring schools and childcare facilities are following Dietary Guidelines for all meals and snacks, providing the public with physical activity and nutrition education, encourage physical activity within the school system, reducing children's time in front of the television and/or computer, and develop supportive marketing tactics on children's food and beverages [10, 11]. In the early 1970s, prevalence of obesity in children was very low. In 2008, the prevalence of obesity in children ages 6–11 years rose to 20 %. Obesity levels rose to 18 % for those American's ages 12–19. Reversing America's obesity epidemic is important when considering the levels of overweight and obese children is higher than ever before. Health risks and diseases associated with obesity that were once diagnosed within the adult population are now being seen in the child and adolescent population [6, 7].

## 28.4 Contemporary Understanding of the Issue

For almost 20 years, the United States Department of Agriculture (USDA) used the food pyramid to represent a healthy diet [12]. MyPlate recently replaced the MyPyramid, which the USDA found was too complex as a nutritional guide for American families [13]. Nutritionists, dietitians, economists, and policy experts at the USDA designed the MyPlate to help individuals understand portion sizes with a familiar image, a plate [14]. This design reminds the National Food Guide (commonly called "The Basic Seven") published back in the 1940s, as well as food guides from countries all over the world, like Mexico, Portugal, Great Britain, or Spain, that have been using the same idea for several decades.

The MyPlate icon shows the five major food groups, fruits, vegetables, grains, protein foods, and dairy, that are the building blocks of a healthy diet. The information given to consumers about healthy eating choices is based on recommendations for Americans 2 years of age and older. Policy experts at the USDA wanted to not only provide nutrition information but to actively change the American eating behavior. The website allows individuals to gain knowledge of healthy eating recommendations and habits to live by as well as interactive games for children to learn what types of foods to eat and those they should avoid [15]. Thus, the MyPlate website, www.choosemyplate.gov, is a very interactive website that allows children to click on one of the five food groups to learn the best choices to build a healthy plate (USDA) [10], explains each group, and shows healthy choices and the recommendations or serving sizes within each group [10].

The purpose of this new icon is to encourage people to eat smaller portions and to fill at least half of your plate with fruits and vegetables.

Focusing on fruit consumptions is very important; any fruit or 100 % fruit juice counts as part of the fruit group. The fruit food gallery on the USDA website shows common fruits consumed and the amount of fruit needed every day. The daily fruit recommendation can be found on Table 28.1 for children. The amounts are appropriate for children who get less than 30 min per day of moderate physical activity. Those who are more physically active may be able to consume more while staying within calorie needs [16].

The vegetable group includes any vegetable or 100 % vegetable juice and is separated into five subgroups since this is a very diverse group considering the macronutrient content of the foods included. The MyPlate's vegetable food gallery lists the groups as dark green vegetables, beans and peas, starchy vegetables, red and orange vegetables, and others. Recommended amounts of vegetables vary with age, sex, and level of physical activity. The vegetable recommendations for children for subgroups are listed as weekly

consumption amounts (Table 28.2). Note that although you do not need to eat vegetables from each subgroup every day, it is important to eat from each subgroup on a weekly basis [17].

It is recommended to build your meal with fruits and vegetables filling up half your plate. It is important for children to consume a variety of fruits and vegetables that will provide the nutrients needed for proper health and maintenance of their body while growing and to reduce the risk for some chronic diseases. MyPlate not only offers food choices with recommended serving sizes but health benefits and nutrients found in fruit and vegetables as well. A diet including recommended amounts of fruit and vegetables may reduce the risk of certain hypokinetic diseases including heart attacks, stroke, and certain types of cancer, type 2 diabetes, high blood pressure, kidney stones, bone loss, and obesity [4, 10].

Including grains into your diet is also important in reducing your risk for certain diseases. Certain grains may reduce the risk for heart disease, reduce constipation, help maintain with weight management, and when fortified with folate, can prevent neural tube defects in pregnant women. However, indiscriminate folate fortification is a very controversial topic. Excessive folate intake is not inert and could have serious consequences, especially for those populations with a misbalanced diet or with a deficient intake of other metabolically related vitamins. Grains are separated into two groups: whole grain and refined grain. Whole grains contain the entire grain kernel and include whole-wheat flour, bulgur, oatmeal, whole cornmeal, and brown rice. Although refined grains are milled to give the product a finer texture and longer shelf life, the process removes important dietary fiber, iron, and many B vitamins [18]. Examples of refined grains include white flour, degermed cornmeal, white bread, and white rice. A list of commonly eaten whole and refined grains can be found on the MyPlate website [18].

Most American adults and children consume enough grains, but few are whole grains. It is recommended that at least half of the grains in your diet are whole grains. Table 28.3 lists is the daily recommended amount of grains and the daily minimum amount of whole grains for children. Daily recommendations for grain are given in ounces. Examples of one ounce of grain include one slice of bread and 1/2 cup cooked pasta or cooked cereal. A list of common eaten foods and their equivalent to one ounce can be found on the MyPlate website [18].

A healthy diet includes foods from the protein group. Any food made from meat, poultry, seafood, beans and peas, eggs, soy products, nuts, and seeds are all examples of the types of foods

**Table 28.1** Daily fruit recommendations

| Age | Serving size |
|---|---|
| Children 2–3 years old | 1 cup |
| Children 4–8 years old | 1–1½ cups |
| Girls 9–18 years old | 1½ cups |
| Boys 9–13 years old | 1½ cups |
| Boys 14–18 years old | 2 cups |

Referenced by [4]

**Table 28.2** Weekly vegetable recommendations

| Age | Dark greens | Red and orange | Beans and peas | Starchy | Other |
|---|---|---|---|---|---|
| Children 2–3 years old | ½ cup | 2½ cups | ½ cups | 2 cups | 1½ cups |
| Children 4–8 years old | 1 cup | 3 cups | ½ cup | 3½ cups | 2½ cups |
| Girls 9–13 years old | 1½ cup | 4 cups | 1 cup | 4 cups | 3½ cups |
| Girls 14–18 years old | 1½ cup | 5½ cups | 1½ cup | 5 cups | 4 cups |
| Boys 9–13 years old | 1½ cup | 5½ cups | 1½ cups | 5 cup | 4 cups |
| Boys 14–18 years old | 2 cups | 6 cups | 2 cups | 6 cups | 5 cups |

Referenced by [4]

**Table 28.3** Daily grain recommendations

| Age | Daily recommendations (ounce) | Daily minimum amount of whole grains (ounce) |
|---|---|---|
| Children 2–3 years old | 3 | 1½ |
| Children 4–8 years old | 5 | 2½ |
| Girls 9–13 years old | 5 | 3 |
| Girls 14–18 years old | 6 | 3 |
| Boys 9–13 years old | 6 | 3 |
| Boys 18–18 years old | 8 | 4 |

Referenced by [4]

**Table 28.4** Daily protein recommendations

| Age | Daily amount of protein (ounce) |
|---|---|
| Children 2–3 years old | 2 |
| Children 4–8 years old | 4 |
| Girls 9–13 years old | 5 |
| Girls 14–18 years old | 5 |
| Boys 9–13 years old | 5 |
| Boys 14–18 years old | 6½ |

Referenced by [4]

that should be consumed to meet the recommendations of the protein group. It is controversial including beans and peas and nuts in this group, since most of them have a high carbohydrate and even fat content, sometimes higher than their protein intake. Choosing a variety of foods with protein is important as well as those foods that are lean or low in fat. Certain seafood rich in omega-3 fatty acids is also important to include into a proper diet. Processed meats such as deli meats should be limited due to its high sodium content. Protein requirements are different depending on age, gender, and physical activity level. Table 28.4 contains the daily recommendations of protein consumption for children. Protein recommendations are given in ounces. Examples of one ounce of protein include one egg, one can of tuna, or approximately twelve almonds [8].

The last food group included in the MyPlate is the dairy group. Most dairy that is consumed

**Table 28.5** Daily dairy recommendations

| Age | Daily amount of dairy |
|---|---|
| Children 2–3 years old | 2 cups |
| Children 4–8 years old | 2½ cups |
| Girls 9–13 years old | 3 cups |
| Girls 14–18 years old | 3 cups |
| Boys 9–13 years old | 3 cups |
| Boys 14–18 years old | 3 cups |

Referenced by [4]

should be fat-free or low in fat. Commonly eaten dairy products are listed on the MyPlate website which includes milk, milk-based desserts, calcium-fortified soymilk, cheese, and yogurt. Age is a large determinant on the amount of dairy recommended. The daily recommended amount of dairy to consume (Table 28.5) [19]. A diet that includes dairy can provide many health benefits. Dairy products can improve bone health, especially during childhood and adolescence, when bone mass is being built. Dairy products can also reduce the risk of certain diseases such as cardiovascular disease and type 2 diabetes and may lower blood pressure [8].

Although oil is not one of the five major food groups, it is important to state that although they can provide essential nutrients, oil should be consumed in moderation. A low intake of saturated fatty acids (SFA) and an adequate intake of unsaturated fatty acids (mono- and polyunsaturated, MUFA, and PUFA) have been largely associated with a healthier lipid profile (triglycerides, total cholesterol, LDL, HDL) and with a lower risk of cardiovascular disease. Most seed oils (sunflower, flax, corn, rapeseed, etc.) and fruit oils (olive oil) have a high content of unsaturated fatty acids. Most oils contain around 120 calories per tablespoon, and it is frequently recommended that they should be limited in a healthy diet [9]. However, it should also be taken into account that the paradigm of a healthy diet, as it is the Mediterranean diet, was originally a high-fat diet, including almost 40 % of calories from fat, especially MUFA and PUFA, based on the use of olive oil for cooking and avoiding other sources of SFA like butter. This means that more important than the amount of fat intake is the type of fatty acids included in our diet.

Along with the MyPlate guidelines, there are also helpful tips for children. On the website you can find resources for children such as the ten tips for making healthy foods more fun for children and ten tips to decrease added sugars. Being a healthy role model is very important in teaching children how to live a healthy lifestyle. Leading by example, eating at the dinner table together, shopping for healthy foods together, and participating in physical activity together are a few behaviors that can model a healthy lifestyle [20]. It is important to include foods from all of these food groups, but it is also just as important to not overeat. Daily caloric recommendations vary depending on age, gender, and activity level. A chart estimating the caloric daily needs for children based on their activity level can be viewed in Appendix 2 [6, 21].

## 28.5 Future Directions

The relationship between diet, physical activity, and health in the short and long term has been extensively analyzed. However, due to the growing prevalence of diet-related chronic and degenerative diseases, further investigation is needed, especially on these directions:

- Analyze the biological and psychosocial factors that determine the adherence (or not) to the dietary guidelines and to the recommendations on exercise in children.
- The influence of gaining knowledge of which foods to eat for proper nutrition on the prevalence of diseases like obesity and type 2 diabetes or on risk factors for these diseases in children and adults.
- In this sense, determine the understanding and effectiveness of the new dietary guide MyPlate in promoting healthy eating choices in children.
- The role of parents, caregivers, school systems, and policymakers on promoting healthy eating and exercise habits needs also to be analyzed, together with the influence in decreasing the obesity and diabetes epidemies.

## 28.6 Concluding Remarks

Teaching children the importance of eating proper nutrition can lead to a healthy lifestyle. It is suggested that our daily meal consists of half of our plate full of fruits and vegetables, along with lean meats, whole grains, and fat-free and/or low-fat dairy products. Limiting our intake of saturated fats, *trans*-fatty acids, and simple sugars is important as well. During childhood, following these guidelines is vital for proper growth and development. Parents and caregivers can lead by example and eat well-balanced meals along with engaging in regular physical activity [22].

## Appendix 1: Nutritional Goals for Age–Gender Groups, Based on Dietary Reference Intakes and Dietary Guidelines

| Nutrient (units) | Source of goal[a] | Child 1–3 | Female 4–8 | Male 4–8 | Female 9–13 | Male 9–13 | Female 14–18 | Male 14–18 | Female 19–30 | Male 19–30 | Female 31–50 | Male 31–50 | Female 51+ | Male 51+ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Macronutrients | | | | | | | | | | | | | | |
| Protein (g) | RDA[b] | 13 | 19 | 19 | 34 | 34 | 46 | 52 | 46 | 56 | 46 | 56 | 46 | 56 |
| (% of calories) | AMDR[c] | 5–20 | 10–30 | 10–30 | 10–30 | 10–30 | 10–30 | 10–30 | 10–35 | 10–35 | 10–35 | 10–35 | 10–35 | 10–35 |
| Carbohydrate (g) | RDA | 130 | 130 | 130 | 130 | 130 | 130 | 130 | 130 | 130 | 130 | 130 | 130 | 130 |
| (% of calories) | AMDR | 45–65 | 45–65 | 45–65 | 45–65 | 45–65 | 45–65 | 45–65 | 45–65 | 45–65 | 45–65 | 45–65 | 45–65 | 45–65 |
| Total fiber (g) | IOM[d] | 14 | 17 | 20 | 22 | 25 | 25 | 31 | 28 | 34 | 25 | 31 | 22 | 28 |
| Total fat (% of calories) | AMDR | 30–40 | 25–35 | 25–35 | 25–35 | 25–35 | 25–35 | 25–35 | 20–35 | 20–35 | 20–35 | 20–35 | 20–35 | 20–35 |
| Saturated fat (% of calories) | DG[e] | <10 % | <10 % | <10 % | <10 % | <10 % | <10 % | <10 % | <10 % | <10 % | <10 % | <10 % | <10 % | <10 % |
| Linoleic acid (g) | AI[f] | 7 | 10 | 10 | 10 | 12 | 11 | 16 | 12 | 17 | 12 | 17 | 11 | 14 |
| (% of calories) | AMDR | 5–10 | 5–10 | 5–10 | 5–10 | 5–10 | 5–10 | 5–10 | 5–10 | 5–10 | 5–10 | 5–10 | 5–10 | 5–10 |
| Alpha-linolenic acid (g) | AI | 0.7 | 0.9 | 0.9 | 1.0 | 1.2 | 1.1 | 1.6 | 1.1 | 1.6 | 1.1 | 1.6 | 1.1 | 1.6 |
| (% of calories) | AMDR | 0.6–1.2 | 0.6–1.2 | 0.6–1.2 | 0.6–1.2 | 0.6–1.2 | 0.6–1.2 | 0.6–1.2 | 0.6–1.2 | 0.6–1.2 | 0.6–1.2 | 0.6–1.2 | 0.6–1.2 | 0.6–1.2 |
| Cholesterol (mg) | DG | <300 | <300 | <300 | <300 | <300 | <300 | <300 | <300 | <300 | <300 | <300 | <300 | <300 |
| Minerals | | | | | | | | | | | | | | |
| Calcium (mg) | RDA | 700 | 1,000 | 1,000 | 1,300 | 1,300 | 1,300 | 1,300 | 1,000 | 1,000 | 1,000 | 1,000 | 1,200 | 1,200 |
| Iron (mg) | RDA | 7 | 10 | 10 | 8 | 8 | 15 | 11 | 18 | 8 | 18 | 8 | 8 | 8 |
| Magnesium (mg) | RDA | 80 | 130 | 130 | 240 | 240 | 360 | 410 | 310 | 400 | 320 | 420 | 320 | 420 |
| Phosphorus (mg) | RDA | 460 | 500 | 500 | 1,250 | 1,250 | 1,250 | 1,250 | 700 | 700 | 700 | 700 | 700 | 700 |
| Potassium (mg) | AI | 3,000 | 3,800 | 3,800 | 4,500 | 4,500 | 4,700 | 4,700 | 4,700 | 4,700 | 4,700 | 4,700 | 4,700 | 4,700 |
| Sodium (mg) | UL[g] | <1,500 | <1,900 | <1,900 | <2,200 | <2,200 | <2,300 | <2,300 | <2,300 | <2,300 | <2,300 | <2,300 | <2,300 | <2,300 |
| Zinc (mg) | RDA | 3 | 5 | 5 | 8 | 8 | 9 | 11 | 8 | 11 | 8 | 11 | 8 | 11 |
| Copper (mcg) | RDA | 340 | 440 | 440 | 700 | 700 | 890 | 890 | 900 | 900 | 900 | 900 | 900 | 900 |
| Selenium (mcg) | RDA | 20 | 30 | 30 | 40 | 40 | 55 | 55 | 55 | 55 | 55 | 55 | 55 | 55 |

(continued)

(continued)

| Nutrient (units) | Source of goal[a] | Child 1–3 | Female 4–8 | Male 4–8 | Female 9–13 | Male 9–13 | Female 14–18 | Male 14–18 | Female 19–30 | Male 19–30 | Female 31–50 | Male 31–50 | Female 51+ | Male 51+ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Vitamins | | | | | | | | | | | | | | |
| Vitamin A (mcg RAE) | RDA | 300 | 400 | 400 | 600 | 600 | 700 | 900 | 700 | 900 | 700 | 900 | 700 | 900 |
| Vitamin D[h] (mcg) | RDA | 15 | 15 | 15 | 15 | 15 | 15 | 15 | 15 | 15 | 15 | 15 | 15 | 15 |
| Vitamin E (mg AT) | RDA | 6 | 7 | 7 | 11 | 11 | 15 | 15 | 15 | 15 | 15 | 15 | 15 | 15 |
| Vitamin C (mg) | RDA | 15 | 25 | 25 | 45 | 45 | 65 | 75 | 75 | 90 | 75 | 90 | 75 | 90 |
| Thiamin (mg) | RDA | 0.5 | 0.6 | 0.6 | 0.9 | 0.9 | 1.0 | 1.2 | 1.1 | 1.2 | 1.1 | 1.2 | 1.1 | 1.2 |
| Riboflavin (mg) | RDA | 0.5 | 0.6 | 0.6 | 0.9 | 0.9 | 1.0 | 1.3 | 1.1 | 1.3 | 1.1 | 1.3 | 1.1 | 1.3 |
| Niacin (mg) | RDA | 6 | 8 | 8 | 12 | 12 | 14 | 16 | 14 | 16 | 14 | 16 | 14 | 16 |
| Folate (mcg) | RDA | 150 | 200 | 200 | 300 | 300 | 400 | 400 | 400 | 400 | 400 | 400 | 400 | 400 |
| Vitamin B6 (mg) | RDA | 0.5 | 0.6 | 0.6 | 1.0 | 1.0 | 1.2 | 1.3 | 1.3 | 1.3 | 1.3 | 1.3 | 1.5 | 1.7 |
| Vitamin B12 (mcg) | RDA | 0.9 | 1.2 | 1.2 | 1.8 | 1.8 | 2.4 | 2.4 | 2.4 | 2.4 | 2.4 | 2.4 | 2.4 | 2.4 |
| Choline (mg) | AI | 200 | 250 | 250 | 375 | 375 | 400 | 550 | 425 | 550 | 425 | 550 | 425 | 550 |
| Vitamin K (mcg) | AI | 30 | 55 | 55 | 60 | 60 | 75 | 75 | 90 | 120 | 90 | 120 | 90 | 120 |

*Sources*: Britten P, Marcoe K, Yamini S, Davis C. Development of food intake patterns for the MyPyramid Food Guidance System. J Nutr Educ Behav 2006;38(6 Suppl):S78–S92. IOM. Dietary Reference Intakes: The essential guide to nutrient requirements. Washington (DC): The National Academies Press; 2006. IOM. Dietary Reference Intakes for Calcium and Vitamin D. Washington (DC): The National Academies Press; 2010. http://www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/PolicyDoc/PolicyDoc.pdf

*AT* Alpha-tocopherol, *DFE* Dietary folate equivalents, *RAE* Retinol activity equivalents

[a]Dietary guidelines recommendations are used when no quantitative dietary reference intake value is available; apply to ages 2 years and older

[b]Recommended dietary allowance, IOM

[c]Acceptable macronutrient distribution range, IOM

[d]14 g per 1,000 cal, IOM

[e]Dietary guidelines recommendation

[f]Adequate intake, IOM

[g]Upper limit, IOM

[h]1 mcg of vitamin D is equivalent to 40 IU

## Appendix 2: Estimated Calorie Needs Per Day by Age, Gender, and Physical Activity Level (Detailed)

**Appendix 2** Estimated amounts of calories[a] needed to maintain calorie balance for various gender and age groups at three different levels of physical activity. The estimates are rounded to the nearest 200 cal. An individual's calorie needs may be higher or lower than these averages

| Gender/activity level[b] | Male/ sedentary | Male/moderately active | Male/active | Female[c]/ sedentary | Female[c]/ moderately active | Female[c]/active |
|---|---|---|---|---|---|---|
| Age (years) | | | | | | |
| 2 | 1,000 | 1,000 | 1,000 | 1,000 | 1,000 | 1,000 |
| 3 | 1,200 | 1,400 | 1,400 | 1,000 | 1,200 | 1,400 |
| 4 | 1,200 | 1,400 | 1,600 | 1,200 | 1,400 | 1,400 |
| 5 | 1,200 | 1,400 | 1,600 | 1,200 | 1,400 | 1,600 |
| 6 | 1,400 | 1,600 | 1,800 | 1,200 | 1,400 | 1,600 |
| 7 | 1,400 | 1,600 | 1,800 | 1,200 | 1,600 | 1,800 |
| 8 | 1,400 | 1,600 | 2,000 | 1,400 | 1,600 | 1,800 |
| 9 | 1,600 | 1,800 | 2,000 | 1,400 | 1,600 | 1,800 |
| 10 | 1,600 | 1,800 | 2,200 | 1,400 | 1,800 | 2,000 |
| 11 | 1,800 | 2,000 | 2,200 | 1,600 | 1,800 | 2,000 |
| 12 | 1,800 | 2,200 | 2,400 | 1,600 | 2,000 | 2,200 |
| 13 | 2,000 | 2,200 | 2,600 | 1,600 | 2,000 | 2,200 |
| 14 | 2,000 | 2,400 | 2,800 | 1,800 | 2,000 | 2,400 |
| 15 | 2,200 | 2,600 | 3,000 | 1,800 | 2,000 | 2,400 |
| 16 | 2,400 | 2,800 | 3,200 | 1,800 | 2,000 | 2,400 |
| 17 | 2,400 | 2,800 | 3,200 | 1,800 | 2,000 | 2,400 |
| 18 | 2,400 | 2,800 | 3,200 | 1,800 | 2,000 | 2,400 |
| 19–20 | 2,600 | 2,800 | 3,000 | 2,000 | 2,200 | 2,400 |
| 21–25 | 2,400 | 2,800 | 3,000 | 2,000 | 2,200 | 2,400 |
| 26–30 | 2,400 | 2,600 | 3,000 | 1,800 | 2,000 | 2,400 |
| 31–35 | 2,400 | 2,600 | 3,000 | 1,800 | 2,000 | 2,200 |
| 36–40 | 2,400 | 2,600 | 2,800 | 1,800 | 2,000 | 2,200 |
| 41–45 | 2,200 | 2,600 | 2,800 | 1,800 | 2,000 | 2,200 |
| 46–50 | 2,200 | 2,400 | 2,800 | 1,800 | 2,000 | 2,200 |
| 51–55 | 2,200 | 2,400 | 2,800 | 1,600 | 1,800 | 2,200 |
| 56–60 | 2,200 | 2,400 | 2,600 | 1,600 | 1,800 | 2,200 |
| 61–65 | 2,000 | 2,400 | 2,600 | 1,600 | 1,800 | 2,000 |
| 66–70 | 2,000 | 2,200 | 2,600 | 1,600 | 1,800 | 2,000 |
| 71–75 | 2,000 | 2,200 | 2,600 | 1,600 | 1,800 | 2,000 |
| 76+ | 2,000 | 2,200 | 2,400 | 1,600 | 1,800 | 2,000 |

*Source*: http://www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/PolicyDoc/PolicyDoc.pdf

[a]Based on Estimated Energy Requirements (EER) equations, using reference heights (average) and reference weights (healthy) for each age–gender group. For children and adolescents, reference height and weight vary. For adults, the reference man is 5 ft 10 in. tall and weighs 154 pounds. The reference woman is 5 ft 4 in. tall and weighs 126 pounds. EER equations are from the Institute of Medicine. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. Washington (DC): The National Academies Press; 2002

[b]Sedentary means a lifestyle that includes only the light physical activity associated with typical day-to-day life. Moderately active means a lifestyle that includes physical activity equivalent to walking about 1.5–3 miles per day at 3–4 miles per hour, in addition to the light physical activity associated with typical day-to-day life. Active means a lifestyle that includes physical activity equivalent to walking more than 3 miles per day at 3–4 miles per hour, in addition to the light physical activity associated with typical day-to-day life

[c]Estimates for females do not include women who are pregnant or breastfeeding. Source: Britten P, Marcoe K, Yamini S, Davis C. Development of food intake patterns for the MyPyramid Food Guidance System. J Nutr Educ Behav 2006;38(6 Suppl):S78–92

## References


1. Centers for Disease Control and Prevention. Overweight and Obesity. Health Consequences. www.cdc.gove/obesity/causes/health.html. Accessed 29 Apr 2012
2. U.S. Department of Agriculture. Center for Nutrition Policy and Promotion. Washington, DC. About us. www.cnpp.usda.gov/AboutUs.htm. Accessed 29 Apr 2012
3. National Food Service Management Institute: Building the Future Through Child Nutrition. Nutrition and Cognitive Development. www.nfsmi.org/documentlibraryfiles/PDF/20080612091850.pdf. Accessed 29 Apr 2012
4. U.S. Department of Agriculture. ChooseMyPlate.gov Website. Washington, DC. What foods are included in the dairy group? www.choosemyplate.gov/food-groups/dairy.html. Accessed 29 Apr 2012
5. U.S. Department of Agriculture. ChooseMyPlate.gov Website. Washington, DC. Health and nutrition information for children over five. www.choosemyplate.gov/children-over-five.html. Accessed 29 Apr 2012
6. U.S. Department of Agriculture. Center for Nutrition Policy and Promotion. Washington, DC. Improving the nutrition and well-being of Americans. www.cnpp.usda.gov. Accessed 29 Apr 2012
7. Centers for Disease Control and Prevention. Overweight and Obesity. Division of Nutrition and Physical Activity. www.cdc.gov/nccdphp/dnpa/obesity/ Accessed 29 Apr 2012
8. U.S. Department of Agriculture. ChooseMyPlate.gov Website. Washington, DC. What foods are in the grains group? www.choosemyplate.gov/food-groups/grains.html. Accessed 29 Apr 2012
9. U.S. Department of Agriculture. ChooseMyPlate.gov Website. Washington, DC. Weight management: decrease portion sizes. www.choosemyplate.gov/weight-management-calories/weight-managment/better-choices/decrease-portions.html. Accessed 29 Apr 2012
10. United States Department of Health and Human Services joint Web site. Available at http://www.health.gov/dietaryguidelines/dga2005/document/. Accessed 29 Apr 2012
11. U.S. Department of Agriculture. ChooseMyPlate.gov Website. Washington, DC. Why is it important to consume oils? www.choosemyplate.gov/food-groups/oils-why.html. Accessed 29 Apr 2012
12. Dietary Guidelines for Americans; 2005. United States Department of Agriculture
13. Park, M. Plate icon to guide Americans to healthier eating; 2011. From CNN health. http://www.cnn.com/2011/HEALTH/06/02/usda.new.food.plate/index.html. Accessed 29 Apr 2012
14. U.S. Department of Agriculture. Center for Nutrition Policy and Promotion. Washington, DC. Estimated calorie needs per day by age, gender, and physical activity level. www.cnpp.usda.gov/Publications/USDAFoodPatters/EstimatedCalorieNeedsPerDayTable.html. Accessed 29 Apr 2012
15. U.S. Department of Agriculture. ChooseMyPlate.gov Website. Washington, DC. FAQs. www.choosemyplate.gov/faqs.html. Accessed 29 Apr 2012
16. U.S. Department of Agriculture. ChooseMyPlate.gov Website. Washington, DC. My Plate. www.choosemyplate.gov/food-groups/. Accessed 29 Apr 2012
17. U.S. Department of Agriculture. ChooseMyPlate.gov Website. Washington, DC. What foods are in the fruit group? www.choosemyplate.gov/food-groups/fruits.html. Accessed 29 Apr 2012
18. U.S. Department of Agriculture. ChooseMyPlate.gov Website. Washington, DC. What foods are in the vegetable group? www.choosemyplate.gov/food-groups/vegetables.html. Accessed 29 Apr 2012
19. U.S. Department of Agriculture. ChooseMyPlate.gov Website. Washington, DC. What foods are in the protein foods group? www.choosemyplate.gov/food-groups/protein-foods.html. Accessed 29 Apr 2012
20. U.S. Department of Agriculture. ChooseMyPlate.gov Website. Washington, DC. How much physical activity is needed? www.choosemyplate.gov/physical-activity/amount.html. Accessed 29 Apr 2012
21. U.S. Department of Agriculture. Center for Nutrition Policy and Promotion. Washington. DC. Dietary guidelines for Americans, 2010. www.cnpp.usda.gov/DGAs2010-PolicyDocument.htm. Accessed Apr 2012
22. U.S. Department of Agriculture. ChooseMyPlate.gov Website. Washington, DC. Be a healthy role model for children. www.choosemyplate.gov/food-groups/downloads/TenTips/DGTipsheet12BeAHealthyRoleModel.pdf. Accessed 29 Apr 2012

# Nutritional Guidelines and Energy Needs for the Female Athlete: Preventing Low Energy Availability and Functional Amenorrhea Through Diet

29

Jacalyn J. Robert-McComb
and Ángela García González

**Abstract**

The prevalence of secondary amenorrhea, in athletes, varies widely with sport, age, training volume, and body weight. Studies over different athlete populations report rates of menstrual disorders ranging from 1 to 69 % and of 70 % for subclinical ovarian disturbances in athletes, while, on the other side, only about 2–15 % of sedentary youth women have menstrual irregularities. Studies showed that normal menstrual cycling is altered if there is a restriction in energy availability, that is, the amount of dietary energy remaining for other body functions after exercise training. When energy availability is too low, physiological mechanisms reduce the amount of energy used for cellular maintenance, thermoregulation, growth, and reproduction. This compensation tends to restore energy balance and promote survival but impairs general health. Studies consistently show that female athletes are not consuming enough energy to support their activity levels, and low energy and nutrient intake places these athletes at a greater risk for nutrition-related disorders such as amenorrhea, osteoporosis, iron-deficiency anemia, and eating disorders. This is not just a problem for those athletes that practice sports with a thigh weight control but for all women practicing high-level sports. So, individual nutritional assessment and dietetic advice are pillars for maintaining health and good performance. Nutritional educational projects must be carefully designed by

J.J. Robert-McComb, Ph.D., FACSM, ACSM P.D. (✉)
Department of Health, Exercise,
and Sport Sciences, Texas Tech University,
3204 Main Street, Lubbock TX 79409, USA
e-mail: jacalyn.mccomb@ttu.edu

Á.G. González, M.D., Ph.D.
Pharmaceutical and Food Sciences, University San
Pablo-CEU, Urb. Montepríncipe; Ctra. Boadilla del
Monte km 5.3, Madrid 28668, Spain
e-mail: angargon@fusp.ceu.es

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_29, © Springer Science+Business Media New York 2014

a multi-professional team and should involve not only the sportive women but also coaches and family to be successful in facilitating athletes the right skills for adopting healthy eating habits. A joint position statement by the American College of Sports Medicine, the American Dietetic Association, and the Dietitians of Canada states that a diet substantially different from that recommended in the Dietary Guidelines for Americans is not needed for athletes, once enough calories are ingested to meet their energy needs. It is recommended that an athlete's diet should consist of nutrient-dense food and beverages within and among the basic good groups while choosing foods that limit the intake of saturated and trans fats, cholesterol, added sugars, salt, and alcohol.

**Keywords**
Functional hypothalamic amenorrhea • Female athlete triad • Energy availability • Secondary amenorrhea • Dietary guidelines for Americans

After completing this chapter, you should have an understanding of the following:

- The term female athlete triad (Triad) and functional hypothalamic amenorrhea (FHA)
- The energy availability hypothesis and FHA
- Energy and nutritional intake estimations
- Nutritional guidelines for the female athlete
- The importance of hydration before, during, and after exercise

## 29.1 Introduction

Studies have repeatedly shown that endurance female athletes do not take in enough calories to meet the exercise challenges they have imposed on their body. Energy deficiency (either intentional or unintentional) may emerge through extreme exercise energy expenditure (EEE) alone, if it is not accompanied by a commensurate increase in energy intake. Even moderate dietary restriction and moderate EEE may result in energy deficiency. In this chapter, we review the research that has focused on the female athlete and a triad of disorders related to insufficient energy intake. Specifically we focus on energy availability and functional amenorrhea. We also discuss the dietary needs of the female athlete to prevent the cascade of disorders.

## 29.2 Research Findings

Both dietary restriction and exercise diminish the availability of utilizable fuels for body functions. During extended periods of deficient metabolic fuel, the body sustains functions necessary for life by diverting scarce metabolic fuels to essential cellular maintenance: The less critical functions not necessary for individual survival, such as reproductive function, are compromised [1]. A longitudinal pattern of disorders in the young athletic female population has been observed. Noteworthy is that the observed disorders may not be considered clinical or even subclinical to be harmful to the long-term health of the athlete. These behaviors have been observed in recreational athletes as well as elite athletes [2, 3]. There seemed to be a relationship among the disorders that had an effect on reproductive function in the short term and possible osteoporosis later in life. It was thought that one disorder led to another disorder. This pattern of disorders had a domino effect and has been referred to as a cascade of disorders.

In 1992, the term female athlete triad (Triad) was coined to describe three distinct but frequently interrelated disorders found in the female athletic population. These components were disordered eating, amenorrhea, and osteoporosis.

In 2007, the American College of Sports Medicine redefined the Triad in terms of the physiological mechanisms by which low energy availability caused functional hypothalamic menstrual disorders and low bone mineral density [4].

It is now understood that low energy availability can occur with or without disordered eating and that other kinds of menstrual disorders are excluded from the Triad. The menstrual disorder referred to in the Triad is known as functional amenorrhea. For the female athlete, the term FHA is used because it is a functional problem, not an anatomical one (i.e., altered hormonal patterns, rather than an anatomical problem), and it is reversible [5, 6].

Although the definitions of amenorrhea are somewhat arbitrary, amenorrhea can be described as primary or secondary. Primary amenorrhea (delayed menarche) is the absence of menstruation by age 15 in a girl with secondary sex characteristics [7]. Secondary amenorrhea is the absence of three or more consecutive menstrual cycles or a period of 3 months without menses after menarche or after cycles have been established [7].

The prevalence of secondary amenorrhea, in athletes, varies widely with sport, age, training volume, and body weight. Studies over different athlete populations report rates of menstrual disorders ranging from 1 to 69 % and of 70 % for subclinical ovarian disturbances. On the other side, studies show that only about 2–15 % of sedentary youth women have menstrual irregularities [8–14].

Warren (1980) was the first to suggest that menstrual disorders in female athletes are caused by an energy drain [15]. Winterer et al. (1985) hypothesized that failure to provide sufficient metabolic fuels to meet the energy requirements of the brain causes an alteration in brain function that disrupts the gonadotropin-releasing hormone (GnRH) pulse mechanism [16]. Reproductive function critically depends on the pulsatile release of GnRH from GnRH neurons in the arcuate nucleus of the hypothalamus and on the consequent pulsatile release of luteinizing hormone (LH) from the pituitary [17]. Figure 29.1 depicts the biochemical axis for menses to occur. This axis is known as the hypothalamus-pituitary-ovarian axis.

In the athletic female, *energy drain* can occur either by not taking in enough calories to meet the metabolic needs of the body or by overexercising and not compensating for the energy cost of the exercise by taking in additional calories. A series of well-controlled studies by Loucks at Ohio University demonstrated that normal menstrual cycling was altered if there is a restriction in energy availability [1, 17–20]. Energy availability (EA) was defined by Loucks as dietary energy intake (DEI) minus EEE. Perhaps it could be paraphrased by stating that EA is the amount of dietary energy remaining after exercise training for other functions of the body such as cellular maintenance, thermoregulation, growth, and reproduction. When EA is too low, physiological mechanisms reduce the amount of energy used for cellular maintenance, thermoregulation, growth, and reproduction. This compensation tends to maintain energy balance (by slowing down metabolism, etc. and not providing energy for noncritical functions such as reproduction) and promote survival but impairs general health [8]. It should be noted that energy availability (DEI − EEE) is not the same as energy balance. Energy balance is defined as DEI minus total energy expenditure (heat from all cellular functions), not just EEE. Energy availability is much simpler to estimate and requires less expensive equipment, and so the term is a more practical term.

For the female athlete who does not take in enough calories, either intentionally or unintentionally, behaviorally controlled restricted dietary energy intake has an effect on the cellular availability of oxidizable metabolic fuels and reproductive function such as glucose [6]. Glucose is an important metabolic fuel needed for maintenance of bodily functions and survival.

The adult female human brain oxidizes approximately 80 g of glucose each day at a continuous rate, and this must be provided daily by dietary carbohydrate, because the brain's rate of energy expenditure can deplete liver glycogen stores in less than 1 day [21]. Moderate exercise oxidizes that much glucose in an hour [17]. On the basis of respiratory quotients measured during exercise training, 62–88 % of the energy

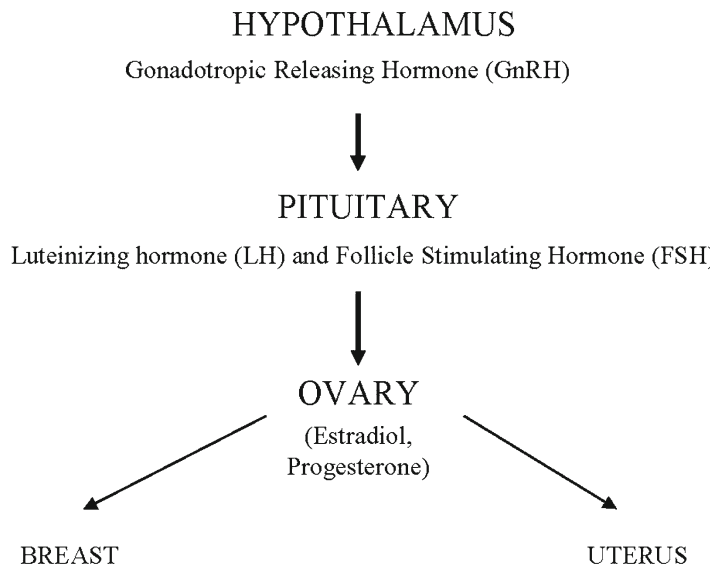



**Fig. 29.1** Hypothalamus-pituitary-ovarian axis

expended during exercise was derived from carbohydrates, principally glucose [20]. Thus, the special demand that aerobic exercise places on glucose stores suggests that the failure of women to sufficiently increase dietary glucose intake, specifically, in compensation for the energy cost of the exercise may lower glucose availability to the brain below a critical threshold necessary for the normal neuroendocrine function of the thyroid, reproductive, and other endocrine axes [22].

Interestingly, Loucks et al. found that EEE may compromise brain glucose availability less than the corresponding amount of dietary energy restrictional one [1]. In their experiment, they found that skeletal muscle derived much less energy from carbohydrate oxidation in the deprived energy availability treatment than in the balanced energy availability treatment (49 versus 73 %) [1]. This alteration in fuel utilization during the deprived energy state conserved approximately 70 % of the brain's daily glucose requirement. Their conclusion from this study was that prolonged exercise had no disruptive effect on LH pulsatility in women—apart from the impact of its energy cost on energy availability or glucose availability. As stated earlier, reproductive function critically depends on the pulsatile release of GnRH from GnRH neurons in the arcuate nucleus of the hypothalamus and on the consequent pulsatile release of LH from the pituitary [17]. Please refer to Fig. 29.2 for a visual understanding of the menstrual cycle and the importance of the pulsatile release of LH.

The inference from Louck's study was that LH pulsatility was disturbed less by EEE than by dietary energy restriction alone [1]. The basis of this inference was the demonstration that during exercise, muscles altered carbohydrate fuel utilization by 33 % during a deprived energy state as opposed to a balanced energy state (73 % balanced energy state versus 49 % energy-deprived state).

In a subsequent study, Loucks and Thuma found that LH pulsatility was disrupted abruptly at a threshold of energy availability less than 30 kcal/kg of lean body mass per day (LBM·d) [17]. The subjects in their experiment were regularly menstruating, habitually sedentary young women of normal body composition. Interestingly, they found that there were thresholds for physiological functioning to be impaired; the relationship between energy availability and altered hormonal and metabolic responses was not linear. Importantly, not all females had the same threshold for energy availability; but if this threshold fell below a critical threshold, hormonal alterations would result.

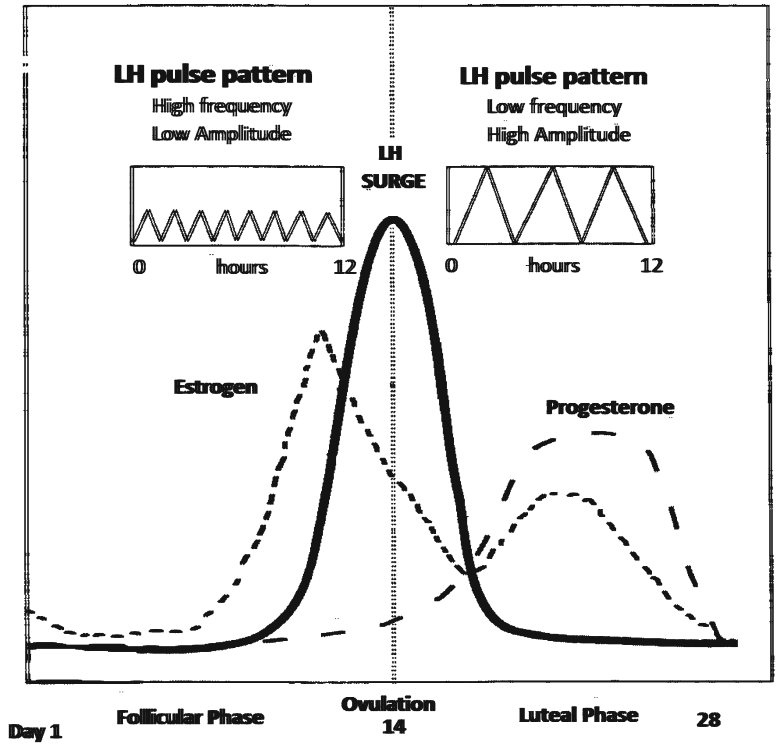



**Fig. 29.2** Patterns of hormone secretion across the normal menstrual cycle. An LH surge occurs at the time of ovulation and marks the division between the follicular phase (days 1–14) and the luteal phase (days 15–28). LH pulse paternal, so changes across the menstrual cycle; pulse frequency decreases from the follicular phase (−65- to 80-min intervals) to the luteal phase (−185- to 200-min intervals), whereas pulse amplitude increases from the follicular phase (−5 miU/mL) to the luteal phase (−12 miU/mL)

They found that the incremental effects of restricted energy availability on LH pulse frequency and amplitude most closely resembled the incremental effects of restricted energy availability on metabolic substrates and hormones such as plasma glucose, cortisol, 3-hydroxybutyrate (beta HOB), and growth hormone. Worth mentioning is that this association did not imply that any of the metabolic substrates and hormones were involved in the mechanism mediating the effects of energy availability on LH pulsatility.

If energy availability was approximately 30 kcal/kg LBM·d, the responses of various metabolic hormones (insulin, cortisol, IGF-I/IGFBP-1/IGHBP-3/leptin, and $T_3$) maintained plasma glucose levels to within 3 % of normal. Many of these hormones block glucose entry into the cell to maintain a normal glucose level. Conversely, leptin and triiodothyronine ($T_3$) were substantially suppressed by a restricted energy availability of 30 kcal/kg LBM·d; $T_3$ was further depressed by a reduction in energy availability of 20 kcal/kg LBM·d. Triiodothyronine is a thyroid hormone that plays vital roles in the body's metabolic rate, heart and digestive functions, muscle control, brain development, and maintenance of bones. Leptin is a hormone that plays a key role in regulating energy intake and energy expenditure, including appetite and metabolism. It is one of the most important adipose-derived hormones.

They also found that the disruptive effects of subthreshold energy availability were bimodal or appearing as two distinct peaks, with substantially larger effects occurring in subjects with the shortest luteal phases. Their results suggested that women with short luteal phases (11 days) might be at a higher risk than others for the suppression of ovarian function and skeletal demineralization by energy deficiency.

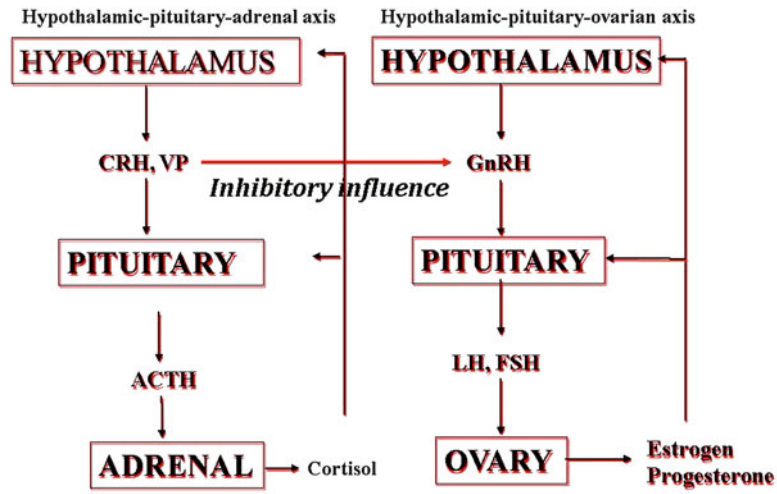



**Fig. 29.3** Suppression of the hypothalamic-pituitary-ovarian (HPO) axis. Axis (HPO) from energy stress by the hypothalamic-pituitary-adrenal (HPA) axis

## 29.3 Contemporary Understanding of the Issues

### 29.3.1 Energy Availability and Functional Amenorrhea

As illustrated in Fig. 29.3, reproductive function may be altered at the level of the hypothalamus because of energy drain, insufficient energy availability, or negative energy balance. More than one term has been used in the literature to present the same concept. More simply stated, it could be called energy or nutritional stress. Stress may even be psychological for reproduction to be altered from stress; however, in this chapter, we are focusing on insufficient energy availability. In this illustration, GnrH is not released at the level of the hypothalamus because of activation of the hypothalamic-pituitary-adrenal (HPA) axis. This axis is sometimes called the stress axis. The diagram depicts activation of the HPA axis in which the hypothalamus releases corticotropin-releasing hormone (CRH) which inhibits GnRH and suppresses the hypothalamic-pituitary-gonadal (HPG) axis. Ultimately LH pulsatility is affected if energy availability falls to a certain threshold or if there is a very low negative energy balance [17]. Menses then ceases resulting in what is termed or simply functional amenorrhea or more precisely FHA. FHA is defined as a nonorganic and reversible disorder in which the impairment of GnRH pulsatile secretion plays a key role in LH pulsatility. LH pulsatility can be suppressed by a combination of strenuous exercise and caloric restriction [1]. There is a threshold of energy availability, roughly 30 kcal/kg LBM·d for most women, that must be met for normal menstrual cycling. The threshold of energy availability is not the same for all women: But if that threshold is met, the restoration of cycling will occur if previously energy deficient and functionally oligo/amenonrrheic [17]. So functional amenorrhea as termed in the Triad of disorders is reversible if the individual's threshold for energy availability is met, which seems to be roughly 30 kcal/kg LBM·d [18].

### 29.3.2 Hormonal Regulation of Food Intake

Food intake and energy expenditure are regulated centrally and peripherally by a plethora of hormones and neuropeptides. The role of some gut hormones, such as ghrelin or peptide YY and adipose-derived hormones such as leptin, on eating control after exercise practice has recently been investigated [23–27]. Figure 29.4 graphically illustrates the role of these hormones in appetite control.

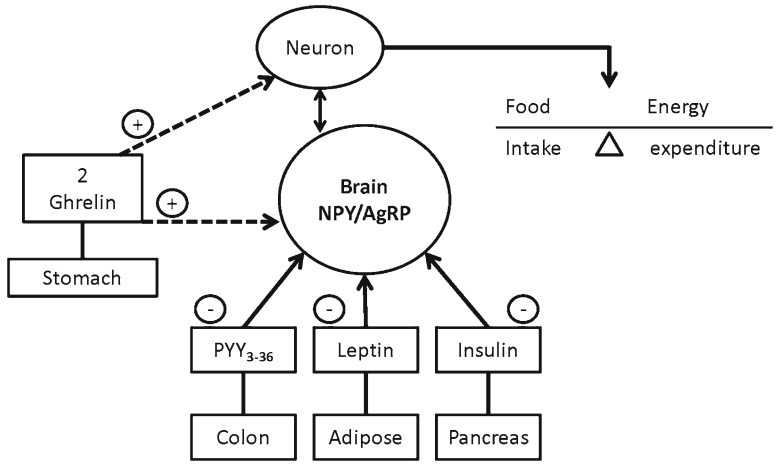



**Fig. 29.4** Neural control of appetite

Ghrelin is a unique circulating peripheral orexigenic hormone that is currently being investigated and deserves attention [28]. Ghrelin is a gastrointestinal hormone secreted by endocrine cells in the stomach. It can cross the blood–brain barrier and activate special receptors in the arcuate nucleus in the hypothalamus of the brain leading to a cascade of processes that end in an increase in hunger and food intake. Through this central mechanism (crossing the blood barrier and activating receptors in the brain), ghrelin has been proposed to play a role in short-term energy homeostasis.

Conflicting findings have been reported regarding exercise training on ghrelin release: (a) some studies found no changes in ghrelin levels after the practice of sport [24, 29]; (b) some studies found a transitional suppression of the acylated form of the hormone [24, 30], the form of the hormone thought to be responsible for appetite stimulation [28]; (c) while other studies found some increase in ghrelin [23, 31]. Yet, even the studies that reported increases in ghrelin ciphers in exercising women with functional amenorrhea found no subsequent increase in appetite or food intake, a fact which may be hypothetically due to some degree of ghrelin resistance [31].

Intense exercise expenditure (without a compensatory intake in calories) is able to induce a short-term negative energy balance during vigorous exercise, a phenomenon that has been described as "exercise-induced anorexia" [24, 25, 32]. Even if ghrelin is secreted, which should signal hunger, there are opposing hormones that are secreted when there is a negative energy balance. If ingestion is not enough to compensate the EEE, we will find a relative negative energy balance, and this negative energy balance will induce pancreatic peptide YY (PYY) release which in turn will induce satiety and less hunger sensation. This phenomenon may be associated with some other molecules in addition to PYY such as glucagon-like peptide-1 (GLP-1) [23].

There has been a marked increase in our understanding of the importance of gut hormones in the regulation of energy homeostasis.

Pancreatic PYY is a hormone which is secreted from endocrine cells called L-cells in the small intestine. It can also cross the blood barrier and act centrally in the control of food intake decreasing hunger and thus food intake. PYY is released after eating, circulates in the blood, and works by binding to receptors in the brain. These receptors then cause a decreased appetite and make people feel full after eating. PYY also acts in the stomach and intestine to slow down the movement of food through the digestive tract. Pancreatic PYY concentrations rise postprandial in proportion to

caloric intake and stay elevated for several hours while fasting. Concentrations are regulated by general caloric intake and negatively correlated with body mass index, suggesting a role of this molecule in long-term energy homeostasis [33].

Additionally, because of the observed changes in the levels of gastrointestinal hormones in women with functional amenorrhea and because their receptors are closely related with the hypothalamic-pituitary-ovarian axis, some authors have hypothesized a direct role of gastrointestinal hormones in the etiology of functional amenorrhea although more studies are needed to determine the exact role of ghrelin, PYY, and adipokines in this pathology [23, 34]. GLP-1 is a hormone produced in the intestinal epithelial endocrine L-cells: GLP-1 is released in response to meal intake. The main actions of GLP-1 are to stimulate insulin secretion and to inhibit glucagon secretion. It also appears to be a physiological regulator of appetite and food intake. Decreased secretion of GLP-1 may contribute to the development of obesity, and exaggerated secretion may be responsible for postprandial reactive hypoglycemia.

In conclusion, during extended periods of reduced energy availability, the body prioritizes by fueling the activities necessary for survival such as thermoregulation and locomotion; therefore, less critical processes such as reproductive function may be compromised. The restoration of normal menstrual cycling has been demonstrated to reoccur when the individual's threshold for energy availability is sequentially met [18]. However, observations suggest that appetite may be an inadequate indicator of energy balance during athletic training, just as thirst is an insensitive indicator of water balance during athletic competition. Athletes may need to eat by discipline rather than by appetite during training to prevent reproductive disorders [1].

### 29.3.3 Estimating Energy and Nutritional Intake

Studies consistently show that female athletes are not consuming enough energy to support their activity level [35–38]. Research with elite female swimmers, using the doubly labeled water technique, noted that total daily energy increased to 5,593 kcal daily during high-volume training. This is the highest caloric expenditure of female athletes reported [39]. However, their intake averaged only 3,136 kcal, implying a negative energy balance. Energy intake of well-trained female athletes ranges from 1,931 to 3,573 kcal [40]. Consequently, their intake of essential vitamins and minerals is lower than the recommended daily allowance [41]. Female athletes' diets have been found to be low in iron, calcium, zinc, vitamin D, vitamin B6, and folate [42].

Low energy and nutrient intake places these athletes at a greater risk for nutrition-related disorders such as amenorrhea, osteoporosis, iron-deficiency anemia, and eating disorders [35, 38]. It must be emphasized that all women participating in high-level competitive sports must remain vigilant and take in enough calories to meet their energy demands. It is not *only* athletes who practice sports with tightly regulated weight control practices who are at risk of suffering the complications of the Triad. All female athletes must have an awareness of the importance of adequate caloric intake to meet energy demands.

Traditionally, recommendations for energy requirements have been based on self-recorded estimates (e.g., diet records) of food intake. However, it is thought that these records are misleading [4]. The percentage of people who underestimate their food intake ranges from 10 to 45 % [43]. Since the advent of the doubly labeled water technique for measuring total energy expenditure, scientists have established energy requirements based on the actual measurement of total energy expenditure in free-living individuals [4]. It has been found that some of the commonly used formulas, such as the Harris–Benedict Equation, to estimate energy requirements are not accurate and underestimate or overestimate requirements [44, 45].

The Mifflin–St Jeor equation is more likely than other equations to estimate RMR within 10 % of the measured and is estimated from weight, height, and age [46]. Multiple-regression analyses were employed to drive relationships between RMR and weight, height, and age for both sexes ($R^2 = 0.71$), but separation by sex did

**Table 29.1** An estimated energy expenditure prediction equation using the Mifflin–St. Jeor equation to determine resting metabolic rate

| | |
|---|---|
| Step 1: *Estimate resting metabolic rate* (*RMR*) *using the Mifflin–St. Jeor equation* | |
| RMR = 9.99 × weight (kg) + 6.25 × height (cm) − 4.92 × age (year) + 166 × sex (males, 1; females, 0) − 161 | |
| Step 2: *Determine additional caloric requirements based on level of activity* | |
| Physical activity level | Percentage above resting level |
| Bed rest | 10 |
| Quiet rest | 30 |
| Light activity | 40–60 |
| Moderate activity | 60–80 |
| Heavy activity | 100 |
| Additional caloric requirements = RMR × percentage above resting level | |
| Step 3: *Determine predicted total energy expenditure* (*TEE*) | |
| TEE = RMR + additional caloric requirements based on activity | |

Adapted from physiology of fitness (3rd ed.) (p. 359) by B. J. Sharkey, 1990, Champaign, IL: Human Kinetics

not affect its predictive value. RMR = 9.99 × weight (kg) + 6.25 × height (cm) − 4.92 × age (year) + 166 × sex (males, 1; females, 0) − 161 [47]. The Mifflin–St Jeor formula can be found in Table 29.1.

The information at the US Department of Agriculture (USDA) National Agricultural Library (NAL) website is quite amazing and very helpful. The Food and Nutrition Information Center (FNIC), located at the USDA NAL is a leader in online global nutrition information including caloric expenditure equations for specific populations. The FNIC website contains over 2,500 links to current and reliable nutrition information. The FNIC provides links to the Dietary Reference Intake (DRI) tables and reports developed by the Institute of Medicine's Food and Nutrition Board. Appendices 1, 2, and 3 contain a list of the valuable information that you can assess at http://fnic.nal.usda.gov/dietary-guidance. The information on this website provides sound nutritional guidance and assessment tools for evaluation. The tools provided on this site are interactive, and there is no charge for them. In fact the government encourages people to use them especially in light of the problems with healthy weight maintenance issues for all people. The Institute of Medicine (IOM) has developed caloric expenditure equations depending on activity level, gender, and age.

Recommendations for caloric intake to maintain weight will vary depending on a person's age, sex, size, and level of physical activity and are provided in the DRI for energy carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids [13]. Tables 29.2 and 29.3 list these equations for females only. The DRI for energy carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids complete document can be downloaded free of charge in a PDF file (http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes/dri-reports). This report is very helpful, and we would encourage you to download the PDF chapter. Appendix 4 also gives estimated caloric needs based on activity levels from the IOM.

Although a self-reported food nutritional assessment may misrepresent total caloric intake because of underreporting, it does provide valuable information to aid in nutritional counseling. Table 29.4 provides guidelines for an exercise nutritionist to help physically active individuals, particularly competitive athletes, achieve energy balance. These guidelines are from the American College of Sports Medicine, the American Dietetic Association, and the Dietitian of Canada Joint Position Statement [48].

A nutritional assessment consists of collecting and evaluating a number of types of information. These include a brief patient history; results of a physical examination (performed by a physician); anthropometric data such as height, weight, body mass index, and percentage body fat; and finally, some biochemical

**Table 29.2** Physical activity level (PAL) index and physical activity coefficient (PA) used to derive estimated energy requirements (EER) for women

| PAL | Sedentary (1.0–1.39) | Low active (1.4–1.59) | Active (1.6–1.89) | Very active (1.9–2.5) |
|---|---|---|---|---|
| | Typical daily living activities (e.g., household tasks, walking to the bus) | Typical daily living activities + 30–60 min of daily moderate activities (e.g., walking at 5–7 km/h) | Typical daily living activities + at least 60 min of daily moderate activities | Typical daily living activities + at least 60 min of daily moderate activities and an additional 60 min of vigorous activity or 120 min of moderate activity |
| PA at 4 levels | PA (level 1) | PA (level 2) | PA (level 3) | PA (level 4) |
| Girls 3–18 years | 1.00 | 1.16 | 1.31 | 1.56 |
| Women 19 years+ | 1.00 | 1.12 | 1.27 | 1.45 |

*PAL* physical activity level or physical activity index, *PA* physical activity coefficient
Adapted from A Report of the Panel on Macronutrients, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for energy carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (**macronutrients**). Washington DC: National Academy Press; 2005

**Table 29.3** Equations to estimate energy requirement

| | |
|---|---|
| Children and adolescents 3–18 years | |
| Estimated energy requirement (kcal/day) = Total energy expenditure + energy deposition | |
| Girls | |
| *3–8 years* | |
| EER = 135.3 − (30.8 × age [year]) + PA × [(10.0 × weight [kg]) + (934 × height [m])] + 20 | |
| *9–18 years* | |
| EER = 135.3 − (30.8 × age [year]) + PA × [(10.0 × weight [kg]) + (934 × height [m])] + 25 | |
| Adults 19 years and older | |
| Estimated energy requirement (kcal/day) = Total energy expenditure | |
| *Women* | |
| EER = 354 − (6.91 × age [year]) + PA × [(9.36 × weight [kg]) + (726 × height [m])] | |
| *Pregnancy* | |
| Estimated energy requirement (kcal/day) = Nonpregnant EER + Pregnancy energy deposition | |
| 1st trimester | EER = Nonpregnant EER + 0 |
| 2nd trimester | EER = Nonpregnant EER + 340 |
| 3rd trimester | EER = Nonpregnant EER + 452 |
| *Lactation* | |
| Estimated energy requirement (kcal/day) = Nonpregnant EER + Milk energy output − Weight loss | |
| 0–6 months postpartum | EER = Nonpregnant EER + 500 − 170 |
| 7–12 months postpartum | EER = Nonpregnant EER + 400 − 0 |

*Note*: These equations provide an estimate of energy requirement. Relative body weight (i.e., loss, stable, gain) is the preferred indicator of energy adequacy
*EER* estimated energy requirement, *PA* physical activity coefficient
*Note*: See Table 32.2 to find the appropriate PA value to use in these equations
Adapted from A Report of the Panel on Macronutrients, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for energy carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (**macronutrients**). Washington DC: National Academy Press; 2005

data that are obtained through blood evaluation. This laboratory testing focuses on serum proteins such as albumin, prealbumin, and retinol-binding protein; creatinine height index; and overall immune status. The initial review process serves a number of functions. It allows for

**Table 29.4** How exercise nutritionists can help female athletes maintain energy balance

| |
|---|
| Athletes should be educated about energy requirements for their sport and the role of food in fueling the body. Female athletes should be educated about the female athlete triad and the long-term health consequences of inadequate energy intake. Unrealistic weight and body composition goals should be discouraged. |
| The athlete's typical dietary and supplement intake during training, competition, and the off-season should be assessed. This assessment should be used to provide appropriate recommendations for energy and nutrient intakes for the maintenance of good health, appropriate body weight and composition, and optimal sport performance throughout the year. |
| Body size and composition of an athlete should be assessed for the determination of an appropriate weight and composition for the sports in which she participates. Since athletes come in all shapes and sizes, girls and women must be allowed to and encouraged to choose sports appropriate for their natural body type. Minimum body composition for good health for the female athlete is 12 %. Provide the athlete with nutritionally sound techniques for maintaining an appropriate body weight and composition without the use of severe diets or nutritionally unbalanced macronutrient choices. |
| The fluid intake and weight loss of athletes during exercise should be assessed. Appropriate recommendations regarding total fluid intake before, during, and after exercise should be made based on this assessment and the most current scientific literature. |
| Carefully evaluate any vitamin/mineral or herbal supplements, ergogenic aids, or performance-enhancing drugs an athlete wants to use. These products should be used only after a careful review of their legality and the current literature pertaining to the ingredients listed on the product label. Caution should be used in recommending these products and should only be recommended after evaluating the athlete's health, diet, nutrition needs, current supplement and drug use, and energy requirements. |

*Source*: Adapted from the American College of Sports Medicine, American Dietetic Association, Dietitian of Canada. Joint Position Statement. Nutrition and Athletics Performance. Med Sci Sports Exerc 2009 4183):709–731

the identification of nutritional and medical risk factors, existing nutritional deficiencies, and past nutritional problems.

The next step is a dietary assessment. The purpose of the dietary assessment is to identify a person's eating habits and to estimate their average daily nutrient intake. Through a variety of methods, information should be obtained on the amount and variety of foods eaten. The simplest method of assessment is to have the individual keep a daily dietary intake record. Because intake tends to vary from day to day, a 3-day food record is more accurate than the 24-h recall. Many nutritionists even recommend a 7-day food record. The dietary intake recording is more accurate if the recalled period is longer. However more cooperation and collaboration are needed. Based on experience, the optimum dietary evaluation recall should be 4 days in length, which should include a holiday (weekend day). If a longer period is needed for more information about the intake of special micronutrients, a 4-day period should be repeated as many times as needed in order to have the required length of time for the analysis. This method allows the practitioner to be able to gather information about changes in food habits because of season ability [49]. However, methods only reflect the person's current diet, not eating habits, established over a long period of time. It is important to recognize that self-reported estimates of food intake are biased and many times do not provide an accurate estimation of food intake; therefore, these should only be used as a guide. A food frequency questionnaire is also helpful to determine eating habits. Appendices 5 and 6 provide examples of nutrition and food frequency questionnaires, respectively, that can be used to gather information.

If the practitioner or the athlete has access to the Internet, there is actually no need to buy a computer program to assess nutritional adequacy. However, nutritional programs should be used for an athlete's diet. As stated earlier in the chapter, dietary assessment tools can be found at the USDA NAL FNIC at http://fnic.nal.usda.gov/dietary-guidance/dietary-assessment. The tools at this site range from a nutritional analysis tool to an activity calorie counter. There is also a behavior change and educational maintenance *tool for Web-based learning for the practitioner. For a complete list of available tools at this site go to Appendix 3.*

If there is a need to install a dietary assessment tool on a lab or a personal computer, there are numerous nutritional assessment software programs on the market—most are under $60.00. Most of these programs contain more than 23,000 food items and are upgradable to allow new food items to be entered by the user. One such program is the Diet Analysis Plus 9.0 Windows/Macintosh CD-ROM, 9th Edition from Cenege Learning. This program allows for a 7-day food intake per individual. It takes into account height, weight, and activity level. It also computes daily and weekly values for recommended daily allowance (RDA) and energy expenditure. In addition, it gives a fairly specific breakdown of nutrients and has the ability to generate graphs, charts, and reports. These programs allow a nutritional novice to enter their own data concerning their individual diet and nutrition and receive easy-to-understand information. The use of nutritional assessment software is relatively widespread because of its low cost and availability. Many of these tools and online programs are perfect devices for coaching young women to be aware of their nutritional and caloric needs.

Even though the determination of energy needs is relatively straightforward, behavior change is complex and not so readily understood. Nutritional educational projects must be carefully designed by a multi-professional team to be successful in facilitating athletes to change their behavior [14, 50–52]. The education should involve not only the sportive women but also coaches and family so that skills are learned for adopting healthy eating habits.

In Chapter 14, the authors discuss the available evidence-based disordered eating programs for active females. Appendix 7 lists resources for helping athletes with eating disorders or even disordered eating patterns which may progress to an eating disorder. If the athlete is exhibiting signs and symptoms of energy imbalance, such as weight loss, amenorrhea, loss of concentration, and irritability, the athlete should be encouraged to seek professional counseling.

## 29.4 Future Directions

### 29.4.1 Nutritional Guidelines for the Female Athlete

#### 29.4.1.1 Priority is Meeting Energy Needs

Meeting energy needs is the first priority for the female athlete. Studies consistently state that female athletes do not take in enough calories. Despite the known causal relation between low energy availability and menstrual disturbances, appropriate practical guidelines for the optimal balance of dietary intake and EEE (to maintain correct ovarian function) in exercising women remain undefined. Energy balance is defined as a state when energy intake (the sum of energy from foods, fluids, and supplement products) equals energy expenditure (the sum of energy expended as basal metabolism, thermal effect of food, and any voluntary physical activity). Current efforts to review this issue and provide a set of guidelines for athletes and physically active women are under way by the Female Athlete Triad Coalition, an international consortium of professionals dedicated to optimizing the health of female athletes. Updated information for general public and professionals can be found in their website (http://www.Femaleathletetriad.com). Table 29.5 shows some hints for athletes to maintain optimal energy intake [8].

#### 29.4.1.2 Micronutrients for Athletes

Research in exercise nutrition indicates that the large number of teenagers and adults, including competitive athletes, who exercise regularly to keep fit do not require additional nutrients beyond those obtained through the regular intake of a nutritionally well-balanced diet if energy needs are being met [40]. A joint position statement by the American College of Sports medicine, the American Dietetic Association, and the Dietitians of Canada stated that a diet substantially different from that recommended in the Dietary Guidelines for Americans or the Nutrition Recommendations for Canadians (55–58 % of energy from carbohydrate, 12–15 % of energy from protein, and

**Table 29.5** Recommendations to get enough energy for active women

| |
|---|
| Balance calories |
| Enjoy your food but eat less. Be aware of hunger and fullness cues. Avoid oversize portions |
| Make half your plate fruits and vegetables |
| Switch to fat-free or low-fat milk and dairy products |
| Make half your grain whole grains |
| Cut back on foods high in solid fats, added sugar, and salt |
| Choose low-sodium products. Read nutritional labels |
| Drink water instead of sugary drinks |

Nattiv A., Loucks AB., Manore MM., Sanborn CF, Sundgot-Borgen J., and Warren MP. American College of Sports Medicine. Position Stand. The Female Athlete Triad. 2007. Med Sci Sports Exerc Oct;39(10):1867–82

25–30 % of energy from fat) is not needed for athletes [53]. It is generally recommended that an athlete's diet should be composed of approximately 55–60 % carbohydrate, 20–25 % fat, and 12–15 % protein [53, 54]. Nevertheless, some experts in sports nutrition recommend broader ranges of the macronutrients depending on the needs of the athlete [40].

The Dietary Guidelines for Americans is published jointly every 5 years by the Department of Health and Human Services (HHS) and the USDA. The 2010 Dietary Guidelines for Americans can be downloaded at http://www.cnpp.usda.gov/DGAs2010-PolicyDocument.htm [55]. The Dietary Guidelines describe a healthy diet as one that emphasizes fruits, vegetables, whole grains, and fat-free or low-fat milk and milk products; includes lean meats, poultry, fish, beans, eggs, and nuts; and is low in saturated fats, trans fats, cholesterol, salt (sodium), and sugar [3, 5, 56]. Although there are general food group categories, there are specific recommendations based on age, gender, and activity level.

The USDA SuperTracker (https://www.choosemyplate.gov/SuperTracker/default.aspx) provides online tools that aid and allow one to customize according to age, gender, and physical activity level. Day-to-day caloric intakes as well as physical activity patterns can be tracked using the SuperTracker. Table 29.6 summarizes the recommendations for a healthy diet according to Choose my Plate (USDA) for a standard active female of 25 years.

A basic premise of the Dietary Guidelines is that food guidance should encourage individuals to achieve the most recent nutrient intake recommendations of the IOM, referred to collectively as the DRIs. Tables of the DRIs are provided for all age groups and can be found at the USDA NAL website at http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes [57].

These nutrient intake levels should be achieved if there is a balance of macronutrients (approximately 55–60 % carbohydrate, 20–25 % fat, and 12–15 % protein) in the female athlete's diet and the foods that are chosen are nutrient dense.

Even though the fuel burned during exercise depends on the intensity and duration of the exercise, the sex of the athlete, and prior nutritional status, an increase in the intensity of the exercise will increase the contribution from carbohydrate. A low-carbohydrate diet rapidly compromises energy reserves for vigorous physical activity or regular training. As the length of the exercise continues, the source of the carbohydrate may shift from the muscle glycogen pool to circulating blood glucose, but if the blood glucose cannot be maintained, the intensity of the exercise will decrease [58]. Additionally, carbohydrate plays an important role as a protein sparer during exercise. Carbohydrate availability inhibits protein catabolism in exercise [59], although studies have documented that females have a greater capacity for lipid oxidation during exercise. This fact allows them to maintain normoglycemia (the presence of a normal concentration of glucose in the blood) and preserve muscle glucose during long sport events. If an athlete consumes 60–65 % of their calories from carbohydrate and energy balance is being maintained, sufficient muscle

**Table 29.6** Example of specific recommendations for physically active females from 19 to 30 years from the US Department of Health and Human Services and the US Department of Agriculture, Dietary Guidelines for Americans.

| Calories | Allowance | | |
|---|---|---|---|
| Total calories | 2400 per day | | |
| Empty calories | <330 per day | | |
| Food group | Food group amount | "What counts as" | Tips |
| Grains | 8 ounces per day | 1 ounce of grains equals: | Eat at least half of all the grains whole grains (4 ounces) |
| | | 1 slice of bread | |
| | | ½ cup of cooked pasta, rice, or cereal | |
| | | 1 tortilla | |
| | | 1 pancake | |
| | | 1 ounce ready to eat cereal | |
| Vegetables | 3 cups per day | 1 cup of vegetables equals: | Include vegetables in meals and snacks |
| Dark green | 2 cups per week | 1 cup raw or cooked vegetable | Add dark-green, red, and orange vegetables to main and side dishes |
| Red and orange | 6 cups per week | 1 cup 100 % vegetable juice | Beans and peas are great source of fiber; add them to salads, soups, and dishes or serve as a main dish |
| Beans and peas | 2 cups per week | 2 cup leafy salad green | |
| Starchy | 6 cups per week | | |
| Other | 5 cups per week | | |
| Fruits | 2 cups per day | 1 cup of fruit equals: | Select fresh, frozen, canned, and dried fruit more often than juice |
| | | 1 cup raw or cooked fruit | Maximize taste and freshness by adapting your choice to what's in season |
| | | 1 cup 100 % fruit juice | Use fruits as snacks or dessert; add them to salads |
| | | ¼ dried fruit | |
| Dairy | 3 cups per day | 1 cup dairy | Drink fat-free or low-fat milk and yogurt |
| | | 1 cup milk | When selecting cheese choose reduced fat versions |
| | | 1 cup fortified soymilk | |
| | | 1 cup yogurt | |
| | | 1 ½ ounces natural cheese | |
| | | 2 ounces processed cheese | |

| | | | |
|---|---|---|---|
| Protein foods | 6 ½ ounces per day | 1 ounce of protein foods equals: | |
| Seafood | 10 ounces per week | 1 ounce lean meat, poultry, or seafood | Eat a variety of food from the protein group each week |
| | | 1 egg | Eat more seafood than meat or poultry |
| | | 1 tablespoon peanut butter | Select lean meat and poultry. Trim or drain fat from meat and remove |
| | | ½ ounce nuts or seeds | Poultry skin |
| | | ¼ cup beans or peas | |
| Oil | 7 teaspoons per day | 1 teaspoon of oil equals: | Choose soft margarines with zero trans fat made from liquid vegetable oil |
| | | 1 tsp vegetable oil | Use vegetable oils rather than solid fats |
| | | 1 ½ tsp mayonnaise | |
| | | 2 tsp tube margarine | |
| | | 2 tsp French dressing | |

Example of personal plan designed using the SuperTracker tool from the USDA (https://www.choosemyplate.gov/SuperTracker/default.aspx) for a woman 25 years old, with BMI of 23 and who practices more than 60 min of moderate physical activity per day

glycogen stores should be maintained from day to day. The athlete also has a lower amino acid breakdown during exercise if carbohydrate intake is 60–65 % of their total caloric intake [60].

Nutritional strategies are needed for optimal recovery of fuel deposits following exercise. Studies have demonstrated improved glycogen repletion when carbohydrates were consumed immediately after exercise instead of some hours later [60]. Carbohydrate-rich meals are recommended during recovery, preferably of a high glycemic index. Adding 0.2–0.5 g of protein per day per kg of body mass to carbohydrates in a 3:1 (carbohydrate:protein) ratio is recommended [60]. Appropriate recovery foods with a favorable mix of carbohydrate and protein include yogurt with granola; crackers, cheese, and fruit; a small smoothie; a bagel with soy nut, almond, or peanut butter; and jam or honey [61]. No difference has been observed in terms of glycogen repletion whether the sources of carbohydrates are solid or liquid [60].

Protein requirements are slightly increased in highly active people. Protein requirements for endurance athletes are 1.2–1.4 g/kg body mass per day whereas those for resistance-trained and strength-trained athletes may be as high as 1.6–1.7 g/kg of body mass per day. Acceptable levels for protein intake for physically active persons may range from 10 to 35 % of calorie [40]. Foods containing proteins with a high biological availability (a high retention and utilization rate by the body) should be emphasized. Research suggests that consuming 20 g of protein 5–6 times per day may be preferable to larger protein intake less frequently [62]. Meats, fish, eggs, and dairy products offer complete sources of protein (providing all essential amino acids) [60, 63].

Fat intake should not be restricted provided that the fat intake is low in saturated fats and trans fats; there is no benefit in consuming a diet with less than 15 % of energy from fat as compared with 20–25 % [39]. To maintain body mass the majority of athletes need an energy intake around 40–50 kcal/kg/day which in practical terms is very hard to obtain solely by increasing carbohydrate consumption; some authors remark that such a large shift is likely to lead to a deficit in some essential proteins and lipids and compromise nitrogen balance necessary to maintain normal sexual steroid hormone levels. So for athletes, the acceptable range of fat intake is from 10 to 35 % of caloric intake [39].

Micronutrients play an important role in energy production, hemoglobin synthesis, maintenance of bone health, adequate immune function, and protection of body against oxidative damage. Routine exercise may also increase the turnover and loss of these micronutrients from the body. As a result, greater intakes of micronutrients may be required to cover increased needs for building, repair, and maintenance of lean body mass in athletes [61].

Vitamins B1, B2, B3, and B6, pantothenic acid, and biotin are crucial in energy metabolism, and many athletes have a diet low in those vitamins although few research has been made about the consequences of vitamin B-deficient diets in athletes either for their health or for sport performance. Low intakes of folic acid or vitamin B12 can lead to anemia [40]. Furthermore, some recent research points out that an increasing evidence exists for a possible fourth component of the triad, *endothelial dysfunction*, and this finding is a cause for concern because the sentinel event in cardiovascular disease pathogenesis is impaired endothelial function [64–67].

It has been documented that folic acid supplementation can improve endothelium-dependent vasodilation, and some researchers showed that supplementation with 10 mg/day of folic acid for 4–6 weeks significantly improved flow-mediated dilation in eumenorrheic and amenorrheic athletes [68–70]. More research is needed to define the optimal dosage and length of treatment with folic acid in athletes with endothelial dysfunction.

Exercise increases oxygen consumption by 10–15-fold, thus increasing oxidative stress. Even though short-term exercise may increase the levels of lipid peroxide by-products, habitual exercise has been shown to result in an augmented antioxidant system and reduced lipid peroxidation [48, 71, 72]. Thus, a well-trained athlete may have a more developed endogenous antioxidant system than a sedentary person. Whether exercise increases the need for

**Table 29.7** Summary of recommendations for macronutrients and energy intake for the physically active female

| |
|---|
| The food guide pyramid provides broad recommendations for healthful nutrition for the physically active individual. Diets should be rich in nutrient-dense foods and emphasize fruits and vegetables, cereals and whole grains, nonfat and low-fat dairy products, legumes, nuts, fish, poultry, and lean meats. Female athlete's diets have been found to be low on iron, calcium, zinc, vitamin B6, and folate. They should make sure that their diet contains foods that contain these vitamins and minerals. |
| Intensity of daily physical activity largely determines energy intake requirements. |
| Studies consistently show that female athletes are not consuming enough energy to support their activity levels. Energy intakes of well-trained female athletes range from 1,931 to 3,573 kcal. However, during high-volume training, such as in swimming, total daily energy may increase to 5,593 kcal daily. Low energy and nutrient intake places athletes at greater risk for nutrition-related disorders such as amenorrhea, osteoporosis, iron-deficiency anemia, and eating disorders. A minimum of 30–40 kcal/kg/day are needed to avoid functional amenorrhea. |
| Precise recommendations do not exist for daily lipid and carbohydrate intake. |
| Fat intake should not be restricted provided that the fat intake is low in saturated fats and trans fats; there is no benefit in consuming a diet with less than 15 % of energy from fat as compared to 20–25 %. An acceptable lipid intake for physically active individuals ranges from 10 to 35 % of caloric intake. |
| Carbohydrate intake is important for the physically active person. General recommendations for carbohydrates range between 6 and 10 g/kg of body mass per day. This range represents approximately 55–65 % carbohydrate intake. Carbohydrates should be predominantly starches from fiber-rich, unprocessed grains, fruits, and vegetables. A low-carbohydrate diet rapidly compromises energy reserves for vigorous physical activity or regular training. Successive days of hard training gradually deplete carbohydrate reserves, even when maintaining the recommended carbohydrate intake. This could lead to "staleness," making continued training more difficult. |
| Studies have demonstrated improved glycogen repletion when carbohydrates were consumed immediately after exercise instead of some hours later. Carbohydrate-rich meals are recommended during recovery, preferably meals with a high glycemic index. Adding 0.2–0.5 g of protein per day per kg of body mass to carbohydrates in a 3:1 (carbohydrate:protein) ratio is recommended. |
| Protein requirements are slightly increased in highly active people. Protein requirements for endurance athletes are 1.2–1.4 g/kg body mass per day whereas those for resistance- and strength-trained athletes may be as high as 1.6–1.7 g/kg of body weight per day. According to the Dietary Reference Intakes, acceptable macronutrient distribution ranges of protein for adults are 10–35 %. |
| Excessive sweating during exercise causes loss of body water and related minerals. Mineral loss should be replaced following exercise through well-balanced meals. Athletes should be well hydrated before beginning to exercise. During exercise, optimal hydration can be facilitated by drinking 150–350 mL (6–12 oz) of fluid at 15–20-min intervals, beginning at the start of the exercise. Consuming up to 150 % of the weight lost during an exercise session may be necessary to cover losses in sweat and urine excretion. Enhancing palatability of the ingested fluid is one way to help promote fluid consumption, before, during, or after exercise. Fluid palatability is influenced by several factors including temperature, sodium content, and flavoring. The preferred water temperature is often between 15 and 21 °C, but this and flavor preference vary greatly between individuals and cultures. |

antioxidant nutrients remains controversial. There is little evidence that antioxidant supplements enhance physical performance. Nevertheless a suboptimal dietary intake of antioxidants as vitamin E, vitamin C, or selenium may lead to health problems [73–75].

Vitamin D status is another important factor in preserving bone health. Athletes who live at northern latitudes or who train primarily indoors throughout the year, such as gymnasts and figure skaters, are at risk for poor vitamin D status, especially if they do not consume foods fortified with vitamin D [40, 60]. Supplementation with calcium and vitamin D should be determined after nutrition assessment. Current recommendations for athletes with disordered eating, amenorrhea, and risk for early osteoporosis are 1,500 mg of elemental calcium and 400–800 IU of vitamin D per day [21].

Iron is required for the formation of oxygen-carrying proteins, hemoglobin and myoglobin, and for enzymes involved in energy production. Iron depletion (low iron stores) is one of the most prevalent nutrient deficiencies observed among female athletes [76]. Iron deficiency, with or without anemia, can impair muscle function and limit work capacity. Low-energy diets or vegetarian diets, with poor availability sources of iron,

are common causes of iron-deficit anemia. Nutritional assessment and counseling should be done before anemia appears and supplements recommended if needed. Table 29.7 summarizes key points regarding nutritional recommendations for the physically active person.

### 29.4.2 The Importance of Hydration Before, During, and After Exercise

Water balance is essential for a good health. As we do not have a real "water body store" we need to replace water losses on a day-to-day basis. Exercise increments body temperature and can elicit high sweat rates and water and electrolyte losses particularly in warm-hot weather.

There is considerable variability for water and electrolyte losses between individuals and between different activities, and if sweat water and electrolyte losses are not replaced, then the person will dehydrate. Dehydration can impair exercise performance, contribute to serious health problems as heat illness, and exacerbate symptomatic rhabdomyolysis [77, 78]. An excess of water intake is also possible but less common. Excessive water intake leads to hyponatremia with sever health consequences [77].

Athletes should be well hydrated before beginning to exercise. In addition to drinking generous amounts of fluid in the 24 h before an exercise session, 4-h pre-exercise, they should consume 5–7 mL/kg body weight. Consuming beverages with sodium (20–50 mEqI/L) and/or small amounts of salted snacks or sodium-containing foods at meals will help to stimulate thirst and retain the consumed fluids [77]. During exercise, individuals should periodically drink depending on environmental conditions, exercise intensity and duration, and opportunities to drink. Optimal hydration can be facilitated by drinking 150–350 mL (6–12 oz) of fluid at 15- to 20-min intervals, beginning at the start of the exercise [79, 80]. In most cases, athletes do not consume enough fluids during exercise to balance fluid losses and thus complete their exercise sessions dehydrated to some extent. Consuming up to 150 % of the weight lost during an exercise session may be necessary to cover losses in sweat and urine excretion [81].

Following exercise, the goal is to fully replace any fluid and electrolyte deficit. The general guidelines are to consume 20 oz of fluid (only fluid or fluid plus foods with a high % of water) for every pound lost during exercise [61]. If recovery time is sufficient, consumption of normal meals and snacks with a sufficient volume of plain water will be enough, provided the food contains sufficient sodium to replace sweat losses. When more rapid rehydration is warranted, a sports beverage is preferred because it provides fluid, carbohydrates, and electrolytes.

The composition of the consumed fluids can be important. The IOM provided general guidance for composition of "sports beverages" for persons performing prolonged physical activity in hot weather. They recommend that these types of fluid replacement beverages might contain approximately 20–30 meq/L sodium (chloride as the anion), 2–5 meq/L potassium, and 5–10 % carbohydrate. These components also can be consumed by nonfluid sources such as gels, energy bars, and other foods [82].

## 29.5 Concluding Remarks

For female athletes, energy balance must be maintained for optimum health and athletic performance. The minimum amount of calories a female should consume to prevent functional amenorrhea is 30 kcal/kg/LBM. A caloric intake less than this affects many of the hormones involved in glucose availability, metabolism, and reproduction. Historically, female athletes, competing in sports where leanness increases performance, have not taken in enough calories to meet their energy needs. A joint position statement by the American College of Sports Medicine, the American Dietetic Association, and the Dietitians of Canada states that a diet substantially different from that recommended in the Dietary Guidelines for Americans or the Nutrition Recommendations for Canadians is not needed for athletes [39, 41]. It is recommended that an athlete's diet should

consist of nutrient-dense food and beverages within and among the basic good groups while choosing foods that limit the intake of saturated and trans fats, cholesterol, added sugars, salt, and alcohol. Athletes should also be well hydrated before beginning to exercise and should drink enough fluid during and after exercise to balance fluid losses.

## Appendix 1: Dietary Reference Intakes

The FNIC is a leader in online global nutrition information. Located at the NAL of USDA, the FNIC website contains over 2,500 links to current and reliable nutrition information.

FNIC provides links to the DRI tables, developed by the IOM's Food and Nutrition Board. To view these tables or download these tables in a PDF file, please go to http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes/dri-tables

**Dietary Reference Intakes: Recommended Intakes for Individuals**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

Comprehensive DRI tables for vitamins, minerals, and macronutrients; organized by age and gender. Includes the 2010 updated recommendations for calcium and vitamin D.

**Dietary Reference Intakes: RDA and AI for Vitamins and Elements (PDF | 28 Kb)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

DRI tables for recommended dietary allowances (RDA) and adequate intakes (AI) of vitamins and elements, including the 2010 updated recommendations for calcium and vitamin D.

**Dietary Reference Intakes: UL for Vitamins and Elements (PDF | 19 Kb)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

DRI table for tolerable upper intake levels (UL) of vitamins and elements, including the 2010 updated recommendations for calcium and vitamin D.

**Dietary Reference Intakes: Macronutrients**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

DRI table for carbohydrate, fiber, fat, fatty acids, and protein.

**Dietary Reference Intakes: Estimated Average Requirements (PDF | 15 Kb)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

DRI table for nutrients that have an estimated average requirement (EAR), the average daily nutrient intake level estimated to meet the requirements of half of the healthy individuals in a group.

**Dietary Reference Intakes: Electrolytes and Water**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

DRI table for sodium, chloride, potassium, inorganic sulfate, and water.

*Note*: You can access these tables at http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes/dri-tables

## Appendix 2: Dietary Reports

The FNIC is a leader in online global nutrition information. Located at the NAL of USDA, the FNIC website contains over 2,500 links to current and reliable nutrition information.

FNIC provides links and PDF downloads to the DRI reports, developed by the IOM's Food and Nutrition Board. To distribute or reprint these reports, please visit The National Academies Press website to secure all necessary permissions.

http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes/dri-reports

**UPDATED—Dietary Reference Intakes for Calcium and Vitamin D (2010) (PDF | 355 Kb)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

Report briefly on new DRIs for calcium and vitamin D, revised in November 2010. Read the prepublication report at the National Academies Press website.

**Dietary Reference Intakes: The Essential Guide to Nutrient Requirements**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

All eight volumes of the DRIs are summarized in one reference volume, organized by nutrient, which reviews function in the body, food sources, usual dietary intakes, and effects of deficiencies and excessive intakes.

**Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride (1997)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

For the 2010 updated recommendations for calcium and vitamin D, refer to the prepublication report at the National Academies Press website.

**Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients) (2005)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline (1998)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc (2001)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes: Proposed Definition of Dietary Fiber (2001)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids (2000)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate (2004)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes: Guiding Principles for Nutrition Labeling and Fortification (2003)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes: Applications in Dietary Planning (2003)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes: Applications in Dietary Assessment (2000)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes Research Synthesis Workshop Summary (2006)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes: Proposed Definition and Plan for Review of Dietary Antioxidants and Related Compounds (1998)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes: A Risk Assessment Model for Establishing Upper Intake Levels for Nutrients (1998)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

## Appendix 3. Dietary Assessment Tool Found at the National Agricultural Library (http://fnic.nal.usda.gov/dietary-guidance/dietary-assessment)

Find the SuperTracker and other tools related to dietary assessment, including calorie calculators and the National Cancer Institute's Diet History Questionnaire. Also find a link to the Dietary Assessment Calibration/Validation Register, a registry of validation studies and publications.

*USDA. 0043enter for Nutrition Policy and Promotion.*

This practical tool lets you plan, analyze, and track your eating and activity habits. Gives tips for making healthy changes.

**Nutrition Analysis Tool (NAT)**

*University of Illinois. Department of Food Science and Human Nutrition.*

A free Web-based nutrient analysis program. Requires log-in.

**Activity Calorie Calculator**

*Fitness Jumpsite.*

Calculates the number of calories burned for a variety of physical activities.

**Fat Intake Screener**

*NutritionQuest.*

Compares individual fat intake to that of the average American.

**Fruit, Vegetable and Fiber Screener**

*NutritionQuest.*

Do you get your five-a-day? Find out if you're eating enough fruits and vegetables to reduce the risk of chronic disease.

**Healthy Body Calculator**

*Ask the Dietitian.*

Calculator provides results on weight, body frame size, body mass index (BMI), waist-to-hip ratio, nutrient recommendations, and physical activity.

**Diet History Questionnaire**

*DHHS.NIH. National Cancer Institute.*

Part of Risk Factor Monitoring and Methods, this questionnaire provides background information and tools and resources for utilizing this program.

**Dietary Assessment Calibration/Validation Register**

*DHHS. NIH. National Cancer Institute.*

Register contains studies and publications which compare dietary intake estimates from two or more dietary assessment methods.

**Behavior Change and Maintenance**

*DHHS. NIH. Office of Behavioral and Social Sciences Research.*

Summary report of research on key health behaviors and lifestyle factors affecting disease.

**USDA Healthy Eating Index**

*USDA. Center for Nutrition Policy and Promotion.*

The HEI is a summary measure of overall diet quality.

## Appendix 4: Estimated Calorie Requirements (in Kilocalories) for Specific Age Groups at Three Levels of Physical Activity[a] Using the Institute of Medicine Equations

| | | Activity level[b,c,d] | | |
|---|---|---|---|---|
| Gender | Age (years) | Sedentary[b] | Moderately active[c] | Active[d] |
| Child | 2–3 | 1,000 | 1,000–1,400[e] | 1,000–1,400[e] |
| Female | 4–8 | 1,200 | 1,400–1,600 | 1,400–1,800 |
| | 9–13 | 1,600 | 1,600–2,000 | 1,800–2,000 |
| | 14–18 | 1,800 | 2,200 | 2,400 |
| | 19–30 | 2,000 | 2,000–2,200 | 2,400 |
| | 31–50 | 1,800 | 2,000 | 2,200 |
| | 51+ | 1,600 | 1,800 | 2,000–2,200 |
| Male | 4–8 | 1,400 | 1,400–1,600 | 1,600–2,000 |
| | 9–13 | 1,800 | 1,800–2,200 | 2,000–2,600 |
| | 14–18 | 2,200 | 2,400–2,800 | 2,800–3,200 |
| | 19–30 | 2,400 | 2,600–2,800 | 3,000 |
| | 31–50 | 2,200 | 2,200–2,600 | 2,800–3,000 |
| | 51+ | 2,000 | 2,200–2,400 | 2,400–2,800 |

[a]These levels are based on estimated energy requirements (EER) from the Institute of Medicine Dietary Reference Intakes macronutrients report, 2002, calculated by gender, age, and activity level for reference-sized individuals. "Reference size," as determined by IOM, is based on median height and weight for ages up to 18 years and median height and weight for that height to give a BMI of 21.5 for adult females and 22.5 for adult males. The estimates are rounded to the nearest 200 calories

[b]Sedentary means a lifestyle that includes only the light physical activity associated with typical day-to-day life

[c]Moderately active means a lifestyle that includes physical activity equivalent to walking about 1.5 to 3 miles per day at 3 to 4 miles per hour, in addition to the light physical activity associated with typical day-to-day life

[d]Active means a lifestyle that includes physical activity equivalent to walking more than 3 miles per day at 3 to 4 miles per hour, in addition to the light physical activity associated with typical day-to-day life

[e]The calorie ranges shown are to accommodate the needs of different ages within the group. For children and adolescents, more calories are needed at older ages. For adults, fewer calories are needed at older ages

## Appendix 5: Nutrition Questionnaire with 3-Day Recall

1. Name _____ _____
2. Age _____ Circle: Male or Female
3. Height_____ Weight _____
4. Usual wt. _____ Desired wt. _____
5. Have you lost weight in the last month? Yes No
   If yes how much _____
6. Have you gained weight in the last month? Yes No
   If yes how much _____
7. Please circle activity level:
   Sedentary (sit most of the day)
   Active (move most of the day)
   Very active (aerobic exercise 3× a week)
   Athletic (aerobic exercise 5× a week)
8. Do you have any food allergies or intolerance? If yes, what are they?
   _____ _____
9. Does your medical history include any of the following: (circle any that apply)
   Diabetes, Ulcers, Heart Disease, Hypertension, Stroke, Vascular disease, Renal disease, or Gastrointestinal disease
   _____ _____ _____
10. Has your physician recommended any special diet? If so, what type of diet?
    _____ _____
11. What type of surgeries have you had?
    _____ _____
12. Are you currently taking any medication(s)? Please specify them all. (Please include all over-the-counter medication and/or all vitamin, mineral, or herbal supplements.)
    _____ _____
13. Do you restrict any foods due to religious beliefs? If yes, what type?
    _____ _____
14. Please write a food diary of everything that you ate in the past three days (or the last 72-h period). Include portion size of foods eaten, the type of food, and how it was prepared. Be specific: 1 cup skim milk, baked chicken breast, ½ cup green beans, etc.

Day One:

Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____

Day Two:

Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____

Day Three:

Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____
Item: _____ Amount: _____

## Appendix 6: Food Frequency Questionnaire

The following questionnaire is designed to help the dietician determine the frequency of food use. It should be used in conjunction with a 3-day recall of food intake. Record as accurately as possible. Amounts should be recorded in measurable amounts (e.g., cups, pounds, teaspoons), and frequencies should be recorded in measurable amounts of time (e.g., 1 day, 3 months, 2 weeks).

1. Do you drink milk? If so, what kind?
   _____ Whole
   _____ 2 %
   _____ Skim
   _____ Other
   How much? _____
2. Do you use fats? If so, what kinds?
   _____ Butter
   _____ Margarine
   _____ Oil
   _____ Other
   How much? _____
3. How often do you eat meat? _____ Poultry?_____ ___ Fish?_____
   Eggs? _____ Cheese? _____ Cold cuts? _____
   Peanut butter? _____ Dried beans? _____
4. Do you eat snack foods? If so which ones? _____ _____ _____
   How often? _____ _____
   How much? _____ _____
5. What vegetables do you eat? How often?
   a. Broccoli _____, Green peppers _____, Cooked greens _____, Carrots _____, Sweet potatoes _____, Winter squash _____
   b. Tomatoes _____, Raw cabbage _____, White potatoes _____, Other raw vegetables _____
   c. Asparagus _____, beets _____, cauliflower _____, corn _____, celery _____, peas _____, lettuce _____, green beans_____
6. What fruits do you eat? How often?
   a. Apples/applesauce_____, apricots _____, bananas _____, berries _____, cherries _____, grapes _____, peaches _____, pears _____, plums _____, pineapple _____, raisins _____, others_____
   b. Oranges/orange juice _____, grapefruits/grapefruit juice _____
7. Bread and cereal products?
   a. How much bread do you eat with meals? _____
   Between meals? _____ _____
   b. Do you eat cereal daily? _____ Weekly? _____ Cooked? _____ Dry? _____
   c. How often do you eat foods such as rice, macaroni, and spaghetti? _____

8. Do you eat canned soup? _____
   Homemade soup? _____
   What kinds? _____
   How often? _____
9. Do you use salt? _____
   Do you salt foods before you taste them? _____
   Do you cook with salt? _____
   Do you crave salt/salty foods? _____
10. How many teaspoons of sugar or honey do you use per day? (Include sugar used on cereals, toast, fruit, and bever-ages)_____
11. Do you drink any of the following?
    Water? _____
    How much? _____
    Coffee, tea, decaf, etc.?_____
    How much? _____
    Carbonated beverages_____
    How much? _____
    Beer, wine, liquor?_____
    How much? _____
    Others?_____
    How much? _____
12. Are there any other foods not listed that you eat frequently?
    _____
    _____
    _____
13. Please rank how you determine your choice of frequently eaten foods?
    Caloric content _____
    Taste _____
    Availability _____
    Price _____
    Nutrient content (fat, carbohydrate, protein) _____

Adapted from Dynamics of Nutrition Support: Assessment, Evaluation, and Implementation. Krey, S.H.,& Murray, R.L., Appleton-Century-Crofts, 1986.

## Appendix 7: Eating Disorder Organizations and Resources

**Anorexia Nervosa and Related Eating Disorders, Inc. (ANRED)**
**Internet:** http://www.anred.com/
ANRED's mission is to provide easily accessible information on anorexia nervosa, bulimia nervosa, binge-eating, and other food and weight disorders. ANDRED, a nonprofit organization, distributes materials on topics such as

**Soy Unica! Soy Latina!**
**Internet:** http://www.soyunica.org/mybody/default.htm
An excellent bilingual website for young Latinas with a good section on eating disorders.

**Eating Disorder Information and Referral Center**
**Internet:** www.EDreferral.com
This website is a resource for information and treatment options for all forms of eating disorders. It includes referrals to local treatment centers nationwide.

**Harvard Eating Disorders Center (HEDC)**
WACC 725
15 Parkman Street
Boston, MA 02114
Tel: (617) 236-7766
**E-mail:** info@hedc.org
**Internet:** http://www.hedc.org/
The Harvard Eating Disorders Center is a national nonprofit organization dedicated to research and education and gaining new knowledge of eating disorders, their detection, treatment, and prevention to share with the community at large. The website includes information about eating disorders, help for family and friends, resources and a listing of events and programs.

**Overeaters Anonymous (OA)**
World Service Office
PO Box 44020
Rio Rancho, NM 87174-4020
Tel: (505) 891-2664
**E-mail:** info@overeatersanonymous.org
**Internet:** http://www.overeatersanonymous.org/
OA is a nonprofit international organization that provides volunteer support groups worldwide. Modeled after the 12-step Alcoholics Anonymous program, the OA recovery program addresses physical, emotional, and spiritual recovery aspects of compulsive overeating. Members are encouraged to seek professional help for individual diet and nutrition plans and for any emotional or physical problems.

**The Renfrew Center Foundation**
475 Spring Lane
Philadelphia, PA 19128
Tel: 1-800-RENFREW
**E-mail:** foundation@renfrew.org
**Internet:** http://www.renfrew.org/
The Renfrew Center Foundation is a tax-exempt, nonprofit organization promoting the education, prevention, treatment, and research of eating disorders. The Renfrew Center Foundation is funded by private donations and by the Renfrew Center, the nation's first freestanding facility committed to the treatment of eating disorders.

## References


1. Louks AB, Verdun M, Heath EM. Low energy availability, not stress of exercise, alters LH pulsatility in exercising women. J Appl Physiol. 1998;84(1):37.
2. Sundgot-Borgen J, Torstveit MK. Prevalence of eating disorders in elite athletes is higher than in the general population. Clin J Sport Med. 2004;14:25–32.
3. Torstveit MK, Sundgot-Borgen J. The female athlete triad: are elite athletes at increased risk? Med Sci Sports Exerc. 2005;37(2):184–93.
4. Frary C, Johnson R. Components of energy expenditure. In: Mahan LK, Escott-Stump S, editors. Krauses's food, nutrition, & diet therapy. 11th ed. Philadelphia, PA: Saunders; 2004. p. 21–36.
5. Otis C, Drinkwater B, Johnson M, et al. American College of Sports Medicine position stand. The female athlete triad. Med Sci Sports Exerc. 1997;29:i–ix.
6. Wade GN, Schneider JE. Metabolic fuels and reproduction in female mammals. Neurosci Biobehav Rev. 1992;16:235–72.
7. Daly JM, Heymsfield SB, Head CA, et al. Human energy requirements: overestimation by widely used prediction equations. Am J Clin Nutr. 1985;42:1170–4.
8. Nattiv A, Loucks AB, Manore MM, Sanborn CF, Sundgot-Borgen J, Warren MP. American College of Sports Medicine position stand. The female athlete triad. Med Sci Sports Exerc. 2007;39(10):1867–82.
9. Beals K, Hill A. The prevalence of disordered eating, menstrual dysfunction, and low bone mineral density among US collegiate athletes. Int J Sport Nutr Exerc Metab. 2006;16:1–23.
10. Black Becker C, McDaniel L, Bull S, Powell M, McIntyre K. Can we reduce eating disorder risk factors in female college athletes? A randomized exploratory investigation of two peer-led interventions. Body Image. 2012;9:31–42.
11. Sundgot J, Klungland M. The female football player, disordered eating, menstrual function and bone health. Br J Sports Med. 2007;41(Suppl I):i68–72.
12. Gabel K. Special Nutritional concerns for the female athlete. Curr Sports Med Rep. 2006;5:187–91.
13. Hoch A, Pajewski N, Moraski LA, et al. Prevalence of the female athlete triad in high school athletes and sedentary students. Clin J Sport Med. 2009;19(5):421–8.
14. Zach K, Smith Machin A, Hoch A. Advances in management of the female athlete triad and eating disorders. Clin Sports Med. 2011;30:551–73.
15. Warren MP. The effects of exercise on pubertal progression and reproductive function in girls. J Clin Endocrinol Metab. 1980;51:1150–7.
16. Winterer J, Cutler Jr GB, Loriaux DL. Caloric balance, brain to body ratio, and the timing of menarche. Med Hypotheses. 1984;15:87–91.
17. Loucks A, Thuma J. Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. J Clin Endocrinol Metab. 2003;88(1):297–311.
18. Loucks A, Callister R. Induction and prevention of low-T3 syndrome in exercising women. Am J Physiol. 1993;264:R924–30.
19. Loucks A, Laughlin J, Mortola L, et al. Hypothalamic-pituitary-thyroidal function in eumenorrheic and amenorrheic athletes. J Clin Endocrinol Metab. 1992;75(2):514–8.

20. Loucks A, Heath E. Induction of low-T3 syndrome in exercising women occurs at the threshold of energy availability. Am J Physiol. 1994;66:R817–23.
21. Bursztein S, Elwyn DH, Askanazi J, et al. Energy metabolism, indirect calorimetry, and nutrition. Baltimore, MD: Williams & Wilkins; 1989. p. 59.
22. I'Anson H, Foster DL, Foxcroft GR, et al. Nutrition and reproduction. Oxf Rev Reprod Biol. 1991;13:239–311.
23. Sheid JL, De Souza MJ. Menstrual irregularities and energy deficiency in physically active women: the role of ghrelin. PYY and adipocytokines. Med Sport Sci. 2010;55:82–102.
24. Stensel D. Exercise, appetite and appetite-regulating hormones: implications for food intake and weight control. Ann Nutr Metab. 2010;57 Suppl 2:36–42.
25. Bilski J, Teleglów A, Zahradnik-Bilska J, Dembinski A, Warzecha Z. Effects of exercise on appetite ad food intake regulation. Med Sport. 2009;13:82–94.
26. Martins C, Morgan LM, Bloom SR, Robertson MD. Effects of exercise on gut peptides, energy intake and appetite. J Endocrinol. 2007;102:2165–71.
27. Broom DR, Batterham RL, King JA, Stensel DJ. Influence of resistance and aerobic exercise on hunger, circulating level of acylated ghrelin and peptide YY in healthy males. Am J Physiol Regul Integr Comp Physiol. 2009;296:R29–35.
28. Chen CY, Asakawa A, Fujimiya M, Lee SD, Inui A. Ghrelin gene products and the regulation of food intake and gut motility. Pharmacol Rev. 2009;61:430–81.
29. Hagobian TA, Sharoff CG, Braun B. Effects of short-term exercise and energy surplus on hormones related to regulation of energy balance. Metabolism. 2008;57:393–8.
30. Broom DR, Stensel DJ, Bishop NC, Burns SF, Miyashita M. Exercise induce suppression of acylated ghrelin in humans. J Appl Physiol. 2007;102:2165.
31. Borer K, Wuorinen E, Ku K, Burant C. Appetite responds to changes in meal content whereas ghrelin, leptin, and insulin track changes in energy availability. J Clin Endocrinol Metab. 2009;94:2290–8.
32. Martins C, Robertson MD, Morgan LM. Effects of exercise and restrained eating behaviour on appetite control. Proc Nutr Soc. 2008;67:28–41.
33. Russell M, Stark J, Nayak S, et al. Peptide YY in adolescent athletes with amenorrhea, eumenorrheic athletes and non-athletic controls. Bone. 2009;45:104–9.
34. Budak E, Fernández Sánchez M, Bellver J, Cervero A, Simón C, Pellicer A. Interactions of the hormones leptin, ghrelin, adiponectin, resistin and PYY3-36 with the reproductive system. Fertil Steril. 2006;85:1563–81.
35. Gibson JC, Stuart-Hill L, Martin S, Gaul C. Nutrition status of junior elite Canadian female soccer athletes. Int J Sport Nutr Exerc Metab. 2011;21:507–14.
36. García-Juan B, Iglesias-Gutiérrez E, Luis-Celada O, Úbeda N. Hábitos alimenticios e ingesta nutricional de mujeres futbolistas de élite. Nutr Hosp. 2008;1:153.
37. Heaney S, O'Connor H, Michael S, Gifford J, Naughton G. Nutrition knowledge in athletes: a systematic review. Int J Sport Nutr Exerc Metab. 2011;21:248–61.
38. Heaney S, O'Connor H, Michael S, Gifford J, Naughton G. Comparison of strategies for assessing nutritional adequacy in elite female athletes' dietary intake. Int J Sport Nutr Exerc Metab. 2011;20:245–56.
39. Trappe TA, Gastaldelli A, Jozsi AC, et al. Energy expenditure of swimmers during high volume training. Med Sci Sports Exerc. 1997;29:950–4.
40. American College of Sports Medicine, American Dietetic Association, Dietitian of Canada. Joint position statement. Nutrition and athletics performance. Med Sci Sports Exerc. 2009;41(3):709–31.
41. Deuster PA, Kyle SB, Moser PB, et al. Nutritional survey of highly trained women runners. Am J Clin Nutr. 1986;44:954–62.
42. Rudd J. Nutrition and the female athlete. Boca Raton, FL: CRC Press; 1996. p. 11–9.
43. Johnson RK. What are people really eating, and why does it matter? Nutr Today. 2000;35(2):40–6.
44. Institute of Medicine (U.S.). Standing committee on the scientific evaluation of dietary reference intakes. Dietary reference intakes for energy carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. Washington, DC: National Academy Press; 2002.
45. Harris JA, Benedict FG. A biometric study of basal metabolism in man. Washington, DC: Carnegie Institute of Medicine; 1919. Pub.No.279.
46. Frankenfield D, Roth-Yousey L, Compher C. Comparison of predictive equations for resting metabolic rate in healthy non-obese and obese adults: a systematic review. J Am Diet Assoc. 2005;105: 775–89.
47. Mifflin MD, St. Jeor ST, Hill LA, Scott BJ, Daugherty SA, Koh YO. A new predictive equation for resting energy expenditure in healthy individuals. Am J Clin Nutr. 1990;51:241–7.
48. American Dietetic Association; Dietitians of Canada; American College of Sports Medicine, Rodriguez NR, Di Marco NM, Langley S. Joint position statement. Nutrition and athletics performance. Med Sci Sports Exerc. 2009;41(3):709–31.
49. Lee R, Nieman D. Nutritional assessment. 6th ed. New York: McGraw Hill; 2012.
50. Gibson R. Principles of nutritional assessment. 2nd ed. New York: Oxford Press; 2005.
51. Black Beckera C, McDaniela L, Bulla S, Powell M, McIntyrea K. Can we reduce eating disorder risk factors in female college athletes? A randomized exploratory investigation of two peer-led interventions. Body Image. 2012;9:31–42.
52. Doyle-Lucas A, Davy B. Development and evaluation of an educational intervention program for pre-professional adolescent ballet dancers. Nutrition for optimal performance. J Dance Med Sci. 2011;15(2):65–75.
53. Rodriguez NR, DiMarco NM, Langley S. American Dietetic Association; Dietitians of Canada; American

College of Sports Medicine: Nutrition and Athletic Performance. Position of the American Dietetic Association, Dietitians of Canada, and the American College of Sports Medicine: Nutrition and athletic performance. J Am Diet Assoc. 2009 Mar;109(3): 509–27.
54. Health Canada. Eating well with Canada's food guide. Website- http://www.hc-sc.gc.ca/fn-an/food-guide-aliment/index-eng.php.
55. U.S. Department of Agriculture. U.S. department of health and human services 2010. Dietary guidelines for Americans 2010 Website- www.dietaryguidelines.gov.
56. Loucks A, Horvarth S. Athletic amenorrhea: a review. Med Sci Sports Exerc. 1985;17(1):56–72.
57. Medicine's Food and Nutrition Board. Dietary reference intakes (DRIs) 2010.Website- http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes.
58. Coyle EF, Coggan AR, Hemmert MK, et al. Muscle glycogen utilization during prolonged strenuous exercise when fed carbohydrate. J Appl Physiol. 1986;61:165–72.
59. Wagenmakers AJ, Beckers EJ, Brouns F, et al. Carbohydrate supplementation, glycogen depletion, and amino acid metabolism. Am J Physiol. 1991;260:E883.
60. Hausswirth C, Le Meur Y. Physiological and nutritional aspects of post-exercise recovery—specific recommendations for female athletes. Sports Med. 2011;41(10):861–82.
61. Bonci L. Eating for performance: bringing science to the training table. Clin Sports Med. 2011;30:661–70.
62. Moore DR, Robinson MJ, Fry JL, et al. Ingested protein dose response of muscle and albumin protein synthesis after resistance exercise in young men. Am J Clin Nutr. 2009;89:161–8.
63. Hoffman J, Falvo M. Protein—which is best? J Sport Sci Med. 2004;3:118–30.
64. Lanser EM, Zach KN M.D., Hoch AZ. The female athlete triad and endothelial dysfunction. PM R. 2011;3:458–65.
65. Rickenlund A, Eriksson MJ, Schenck-Gustafsson K, Hirschberg AL. Amenorrhea in female athletes is associated with endothelial dysfunction and unfavorable lipid profile. J Clin Endocrinol Metab. 2005;90:1354–9.
66. Hoch AZ, Papanek P, Azabo A, Widlansky ME, Schimke JE, Gutterman DD. Association between the female triad athlete and endothelial dysfunction in dancers. Clin J Sport Med. 2011;21:119–25.
67. Hoch AZ, Jurva JW, Staton MA, et al. Athletic amenorrhea and endothelial dysfunction. WMJ. 2007;106(6):301–6.
68. Hoch AZ, Lynch SL, Jurva JW, Schimke JE, Gutterman DD. Folic acid supplementation improves vascular function in amenorrheic runners. Clin J Sport Med. 2010;20:205–10.
69. Hoch AZ, Papanek P, Szabo A, Widlansky ME, Gutterman DD. Folic acid supplementation improves vascular function in professional dancers with endothelial dysfunction. PM R. 2011;3(11):1005–12.
70. Hoch AZ, Pajewski NM, Hoffmann RG, Schimke JE, Gutterman DD. Possible relationship of folic acid supplementation and improved flow-mediated dilation in premenopausal, eumenorrheic athletic women. J Sports Sci Med. 2009;8:123–9.
71. Djordjevic DZ, Cubrilo DG, Barudzic NS, et al. Comparison of blood pro/antioxidant levels before and after acute exercise in athletes and non-athletes. Gen Physiol Biophys. 2012;31(2):211–9.
72. Turner JE, Hodges NJ, Bosch JA, Aldred S. Prolonged depletion of antioxidant capacity after ultraendurance exercise. Med Sci Sports Exerc. 2011;43(9):1770–6.
73. Peternelj TT, Coombes JS. Antioxidant supplementation during exercise training: beneficial or detrimental? Sports Med. 2011;41(12):1043–69.
74. Sachdev S, Davies KJ. Production, detection, and adaptive responses to free radicals in exercise. Free Radic Biol Med. 2008;44(2):215–23.
75. de Gonzalo-Calvo D, Fernández-García B, de Luxán-Delgado B, et al. Chronic training increases blood oxidative damage but promotes health in elderly men. Age (Dordr). 2012;35(2):407–17. doi:10.1007/s11357-011-9358-6.
76. Beard J, Tobin B. Iron status and exercise. Am J Clin Nutr. 2000;72(2):594S–7.
77. Sawka MN, Burke LM, Eichner ER, Maughan RJ, Montain SJ, Stachenfeld NS. American College of Sports Medicine position stand. Exercise and fluid replacement. Med Sci Sports Exerc. 2007;39(2): 377–90.
78. Shirreffs SM, Sawka MN. Fluid and electrolyte needs for training, competition, and recovery. J Sports Sci. 2011;29(1):S39–46.
79. Convertino VA, Armstrong LE, Coyle EF, Mack GW, Sawka MN, Senay LC Jr, Sherman WM. American College of Sports Medicine position stand. Exercise and fluid replacement. Med Sci Sports Exerc. 1996;28(1):i–vii.
80. Casa DJ, Armstrong LE, Hillman SK, et al. National athletic trainer's association position statement: fluid replacement for athletes. J Athl Train. 2000;35(2):212–24.
81. Shirreffs SM, Taylor AJ, Leiper JB, et al. Post-exercise rehydration in man: effects of volume consumed and drink sodium content. Med Sci Sports Exerc. 1996;28:1260–71.
82. Institute of Medicine. Fluid replacement and heat stress. 1994.

# Ergogenic Aids and the Female Athlete

# 30

Shannon L. Jordan and Fernando Naclerio

**Abstract**

Female athletes tend to choose their supplements for different reasons than their male counterparts. Collegiate female athletes report taking supplements "for their health," to make up for an inadequate diet, or to have more energy. Multivitamins, herbal substances, protein supplements, amino acids, creatine, fat burners/weight-loss products, caffeine, iron, and calcium are the most frequently used products reported by female athletes. Many female athletes are unclear on when to use a protein supplement, how to use it, and different sources of protein (whey, casein, and soy). This chapter addresses essential amino acid and branched chain amino acid supplementation. Along with recommendations for protein supplementation, creatine supplementation is discussed. Not all female athletes are concerned with building muscle. Burning fat is also a major concern for the female athlete. This may result in the athlete turning to products marketed for weight control (i.e., ginseng or ephedra). A product legal for over-the-counter (OTC) sales, however, can be illegal for athletic competition (i.e., ephedra). Competitive athletes should be aware of the banned substance list for their governing body and that OTC products are not currently regulated by the FDA. This lack of regulation can lead to OTC products that are contaminated with banned substances.

**Keywords**

Anabolic steroids • BCAA • Creatine • Echinacea • Ephedra • Ginseng • Protein • Supplement

S.L. Jordan, Ph.D. (✉)
Department of Kinesiology, Texas Wesleyan University, Ft. Worth, TX, USA
e-mail: sljordan@txwes.edu

F. Naclerio, Ph.D.
Centre of Sport Science and Human Performance, University of Greenwich, School of Science, Kent, UK
e-mail: f.j.naclerio@gre.ac.uk

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_30, 

## 30.1 Learning Objectives

After completing this chapter, you should have an understanding of:

- Reasons why female athletes take supplements
- Various supplements female athletes are likely to take
- Ergogenic and ergolytic effects of these supplements
- Variations within certain supplements and standard dosages
- That many nutritional supplements are contaminated with banned substances

## 30.2 Introduction

Many things ingested by women can be considered ergogenic. Ergogenic aids, by definition, are items or substances which enhance performance. Female athletes tend to choose their supplements for different reasons than their male counterparts. Collegiate female athletes report taking supplements "for their health," to make up for an inadequate diet, or to have more energy [1, 2]. Interestingly, while many athletes report using energy drinks and calorie replacement bars or drinks, most of them did not consider these products to be supplements [2]. The subsequent sections will provide insight into the mechanisms and possible benefits (or lack thereof) for ergogenic aids that female athletes commonly cite ingesting.

## 30.3 Research Findings

### 30.3.1 What Are Female Athletes Taking?

Multivitamins, herbal substances, protein supplements, amino acids, creatine, fat burners/weight-loss products, caffeine, iron, and calcium are the most frequently used products reported by female athletes [1–3].

When compared to female nonathletes, female athletes consume similar amounts of performance-enhancing drugs (25.2–29.8 %, respectively) [4]. Performance-enhancing drugs included methamphetamines, ephedrine, university-banned substances, and weight-loss and nutritional supplements [4]. Female athletes reported using performance-enhancing drugs 21.5 % of the time during a competitive season and 25.9 % in the off season [4]. Many female athletes list taking Echinacea and cite "boosting the immune system" as the reason for taking it [1, 3, 5]. However, there are recent reports that Echinacea may also be performance enhancing as well [6, 7].

While many studies do not report steroid use, Congeni and Miller found female adolescent steroid usage to be up to 2.5 % [8]. Other researchers have found that 5.1 % of middle school female students have already reported steroid use by the age of 12 [9]. In a 2009 report from the National Collegiate Athletic Association (NCAA), the use of anabolic steroids increased from 2005 to 2009 in the following women's sports: lacrosse (0–0.2 %), swimming (0.3–0.4 %), and track (0.1–0.2 %) [10]. Frequency of use in other women's sports such as basketball, field hockey, golf, soccer, and volleyball either remained the same or the prevalence of steroid usage actually decreased [10]. Female athletes may also take "fat-burning" supplements in order to maintain or improve body composition [4]. Female athletes reported a 5.1 % increase in usage of weight-loss products in the off season [11].

### 30.3.2 Protein and Amino Acids

Female athletes tend to take their protein supplements in the form of powders, bars, or as meal replacements [1, 2]. Some female athletes also use amino acid supplements [2]. When classified by type of sport, athletes (men and women) who play a sport involving a mixture of aerobic and anaerobic energy systems consume the most protein supplements [2]. Aside from listing protein powders, protein bars, and amino acids, the most prevalent protein supplement reported by females were meal replacement drinks. When asked why they take protein supplements, females responded: "*for enhanced recovery, just like the*

*taste, provide more energy, to meet nutritional needs, enhanced performance, greater muscle strength, and don't know*" [1]. When these same athletes were asked if they had questions about protein supplement usage, there were several questions or concerns: increasing lean body mass and losing body weight, adverse effects on the kidneys, positive vs. negative effects, how much to consume, and whey vs. soy protein.

There is insufficient data to support a determination of whether differences in the quantitative requirements for protein or any individual indispensable amino acids exist between adult, men, and women. With the exception of some specific scientific findings, no reference to gender will be emphasized. However, in order to highlight the special need for females, some particular recommendations related to women will be given.

The protein requirements for certain athletic populations have been the subject of much scientific debate. During the last decade, the notion that both strength/power and endurance athletes, as well the general active population (including men and women), require greater protein consumption than the current RDA recommendation of 0.8 g/kg of body weight per day in healthy adults is becoming generally accepted [12, 13]. In addition, high-protein diets have also become quite popular in the general population as part of many weight reduction programs [14, 15]. In fact, in addition to the positive effects for optimizing protein synthesis and improving the recovery after training, a particular emphasis has been placed on the role of protein for weight management [16, 17].

Differences in protein requirements for athletes and nonathletes and different types of athletes (i.e., endurance, strength/power) are well documented [18–21]. During the last 20 years, studies have investigated the effects of protein obtained from different sources such as milk or soy, as well as the technique used to manufacture the protein extract or the methodology recommended for their ingestion. Specifically, these factors can be of great importance when the protein is ingested in regard to augmenting the acute physiological response to a training session or in enhancing recovery from exercise in general [22].

Regarding women, it has been observed that female athletes tend to take in their protein supplements as powders, bars, or in meal replacement products [2]. In addition to protein, some female athletes also ingest amino acid supplements [2, 23]. A considerable number of regular strength training adepts (including men and women) consume protein supplements mixed with other products (mainly creatine and amino acids). However a limited number of individuals consult "dietary specialists" and rely mainly on their instructors [23]. For males and females, the most common reason given for protein or amino acid consumptions is to increase strength or train longer [24].

For pregnant and lactating women, the amino acid requirement is taken to supplement the extra dietary need associated with the deposition of protein in tissues or secretion of milk at rates consistent with good perinatal health. Researchers have shown that a balanced protein energy diet in expectant females leads to reduction in the risk of small for gestational age infants, especially among undernourished pregnant women [25].

#### 30.3.2.1 Protein Powders

Most of protein extracts are obtained from milk, eggs, bovine colostrums, soy, and, to a lesser extent, bean, wheat, or rice. The quality of the extract may vary depending on the type and quality of the manufacturing process [26, 27]. In this section we will analyze the three principal forms in which most protein supplements are currently found in the market: whey, casein, and soy.

##### Whey

Whole milk is composed of approximately 87 % water, with the remaining 13 % being solids. The solid proportion is composed of 30 % fat, 37 % lactose, 27 % protein, and 6 % minerals. Of the 27 % of milk which is protein, 20 % is whey and 80 % casein protein [28]. During the process of making cheese, whey protein is separated out into a transparent liquid fraction which has shown to be a good source of high-quality protein currently used to make protein powders and many other products. Whey protein is particularly rich in essential amino acids (EAA), vitamins, and minerals [26].

The whey protein fraction is composed of numerous individual proteins, including β-lactoglobulin (50–55 %), α-lactalbumin (20–25 %), bovine serum albumin (5–7 %), lactoferrin (1–2 %), immunoglobulins (10–15 %), lactoperoxidase enzymes, and glycomacropeptides (~0.5 %) [26]. These proteins are involved in several health and immune system functions [29]. The amount of these native proteins remaining intact in the final whey product depends on the processes used to separate out the fat and lactose while the proteins are being purified. Selective elution, also known as ion exchange chromatography, is the process most often used to make whey protein isolates. With the high-quality of manufacturing, the native protein structures can be retained. However, some of the smaller peptides such as lactoferrin may have a decreased concentration, while the β-lactoglobulin protein fraction tends to increase in whey protein isolates [28].

Collectively, whey proteins contain all the EAAs. Relative to other protein sources, whey has a high concentration of cysteine and the branched chain amino acids (BCAA)—leucine, isoleucine, and valine. However, whey protein contains lower quantities of glutamine, arginine, and taurine. Since these amino acids have important neuronal and metabolic functions, it is advisable to fortify whey protein extract by adding peptides of L-glutamine, L-arginine, and taurine [26, 27]. For instance, glutamine is the most abundant amino acid in the blood, accounting for 30–35 % of amino acid nitrogen in the plasma. L-glutamine fulfils a number of biochemical needs. It operates as a nitrogen shuttle, taking up excess ammonia and forming urea. It can act as a powerful anticatabolic agent contributing to the production of other amino acids, glucose, nucleotides, protein, and glutathione [30]. Arginine is a semi-essential amino acid, involved in numerous areas of human biochemistry, including ammonia detoxification, hormone secretion, and immune modulation. L-arginine is the main physiological precursor of nitric oxide (NO), which plays an important regulatory role by increasing blood flow to the muscles and modulating muscle contraction, along with glucose and amino acid uptake by the muscle [31]. Taurine is a conditional EAA involved in a number of physiological processes including cell membrane stabilization, neural excitability or sensibility, and nutrient intake into the cell. Even when taurine is the most abundant free amino acid in excitable tissue such as muscle heart and brain, it is not used to build protein but has other important functions as insulin mimetic, cellular hydration, and protein synthesis stimulation [32].

In addition to the positive effects observed in athletes, in sedentary or slightly active individuals, 50 g/d of whey protein supplement added to a hypocaloric weight-loss diet has shown positive effects to maintain lean tissue relative to a change in adiposity. This results in greater weight gain of relative muscle mass compared to the dietary addition of a similar amount of carbohydrate in older postmenopausal, overweight, or obese women [33]. It seems that the addition of whey protein supplements during weight-loss plans in women could be a good strategy to offset the deleterious effects of both sedentary and aging on muscle mass and bone loss. Additionally, when a high-quality protein supplementation is appropriately combined with a well-designed long-term resistance training program, significant positive effects on physical performance, maintenance or gaining of muscle mass, and improved bone density have been reported [33–35].

### Casein

Casein accounts for approximately 75–80 % of total milk protein and is responsible for the white color of milk [27]. Casein consists of three different protein fractions: α-casein, β-casein, and κ-casein. It is the protein source of cheese and forms curds during processing because it exists as a micelle in milk [28]. Because native casein has low solubility and forms clumps or curds, casein used in nutritional supplements is made into rennet casein or caseinates, an acid form that is usually combined with sodium, calcium, or potassium.

Compared to the whey protein form that remains soluble in the stomach and thus is emptied rapidly, casein is converted into a solid clot, thus emptied more slowly from the stomach [36].

These differences in digestive properties likely contribute to the variation in the pattern of amino acid concentrations that have been observed after ingestion of whey, soy, or casein protein. Casein has been termed a "slow" protein, and whey protein has been named a "fast" protein [36, 37]. The slowly absorbed casein protein would promote a more sustained and prolonged postprandial protein deposition via inhibition of protein breakdown without an excessive increase in amino acid concentration. By contrast, a fast whey protein stimulates protein synthesis but also oxidation. The impact of amino acid absorption speed on protein metabolism is true when proteins are given alone; however, this effect might be blunted in more complex meals that could affect gastric emptying (lipids) and or insulin response (carbohydrates) [38]. Additionally, the more prolonged anabolic effects of casein seem to be most effective when consumed in the rested condition [18]. In contrast, ingestion of a single dose of whey protein stimulates an anabolic response when consumed postexercise [39].

#### Soy

Soy is the most widely used vegetable protein source and it is reported to have equivalent quality to egg or milk protein [40]. Soy's quality makes it a very attractive alternative for those who are lactose intolerant. Soy is a complete protein with a high concentration of BCAA's, plus arginine, moderate amounts of glutamine, and low amounts of methionine [37]. There have been many reported benefits related to soy proteins affecting health and performance (including reducing plasma lipid profiles, increasing LDL-cholesterol oxidation, and decreasing blood pressure) [41]. These health-related benefits have been attributed to the amount of isoflavones which are naturally active nutrients contained in soy protein. Isoflavones' molecular structure is similar to natural body estrogens (phytoestrogen). The following foods contain anywhere from 1 to more than 5 mg/g of isoflavones: tofu, green soybeans, mature or roasted soybeans, and soy flour [41]. Another product made from soybeans is textured soy protein. This product is the typical protein source in imitation chicken, pork, or other meat products. Textured soy protein is also used in numerous other soy protein products which tend to retain the original isoflavone content [28].

Soy products of up to 60 g/d have been safely used in studies lasting up to 2 months [42]. During pregnancy, ingestion of soy protein has been reported as safe when consumed orally in amounts naturally found in foods. However, soy could be unsafe when used orally in medicinal amounts due to its estrogenic constituents. Theoretically, therapeutic use of soy might adversely affect fetal development (i.e., feminization of the male fetus) [42, 43].

### 30.3.2.2 Whey, Casein, and Soy Protein Comparison

Many studies have examined the physiological effects of whey, casein, and soy protein supplements in isolation or combined with carbohydrate and other nutrients such as creatine, amino acids, or lipid-like compounds [44, 45]. However, until now there is no convincing evidence to support the notion that whey, casein, or soy could be a superior protein source for athletes and other active individuals including women. Indeed, each protein source has unique attributes that may convey specific nutritional advantages when compared to the others. These benefits include boosting general health, body weight management, increased lean body mass, enhanced muscle recovery after exercise, and an ability to stimulate skeletal muscle protein synthesis. The differences in the degree by which each source of protein can stimulate the aforementioned processes are related to differences in digestion rates, divergent amino acid profiles, and the presence of naturally occurring antioxidants [37].

### 30.3.2.3 Responses to Ingestion of Different Protein Sources at Rest

The speed of amino acid absorption has a major impact on postprandial protein synthesis and breakdown responses after a meal. Since whey protein is absorbed faster than casein and soy, the appearance of circulating amino acids is more rapid and levels tend to be higher. This rapid increase is transient and, therefore, whey

produces a fast but temporary increase of both whole-body protein synthesis and amino acid oxidation [46].

Casein tends to inhibit protein catabolism when consumed at rest and has a slight but relatively long effect of increase in protein synthesis. The pattern of amino acid delivery with casein ingestion at rest appears to lead to better leucine balance (net protein state) than whey consumption at rest. It is interesting to note that a single dose of casein ingestion does not lead to large increases in circulating levels of amino acids as was observed with the equivalent single dose of whey protein [18]. However, when the same amount of protein is ingested in smaller, more frequent feeding, whey seems to work more effectively to stimulate muscle protein synthesis and reduce amino acid oxidation as compared to casein [18].

Overall, it would appear that casein protein with its slow absorption rate leads to a greater positive net whole-body protein state than single feedings of whey protein or amino acids at rest. Additionally, frequent small feedings of whey protein may lead to the greatest net protein balance and potential gain in muscle mass [18, 46, 47].

Isolate soy protein (ISP) has been shown to have faster absorption rates than casein but slower than whey. After ingesting a standardized dose of ISP, plasma amino acid levels peak at 150 min, as opposed to 75 min if a comparable dose of whey is consumed. Thus, it seems that soy isolate is more of an "intermediate" protein in terms of digestion rate based on plasma peak amino acid concentrations [37].

#### 30.3.2.4 Responses to Ingestion of Different Protein Sources with Exercise

Both soy and whey protein similarly increase muscle protein synthesis rates when fed immediately after exercise [37]; meanwhile casein ingestion does not increase protein synthesis but ameliorates protein breakdown [38].

In both young and older individuals after a typical resistance training workout, whey protein has been shown to stimulate a greater acute (0–3 h postexercise) rise in muscle protein synthesis compared with dose-matched casein and soy proteins [48, 49], and it is still highly effective for stimulating muscle protein synthesis after 3–5 h postexercise [50]. As stated above, the mechanism underlying the robust anabolic properties of whey protein relative to casein is likely related to the more rapid digestion kinetics and greater appearance of circulating amino acids (specifically the BCAA leucine), which may be particularly important as a key metabolic regulator of muscle protein synthesis through activation of the mTOR pathway [48, 50]. The superior capacity of whey to stimulate protein synthesis after training compared to soy protein is not due to differing absorption kinetics but probably due to the lower leucine content of soy versus whey protein [37].

Regarding women, with a few exceptions related to BCAA effects, there is no comparative research aimed to determine different responses to protein or amino acid ingestion compared to men (51). However, sex has been shown to significantly influence metabolic fuel selection during endurance activity. Women appear to oxidize proportionately more lipid and less carbohydrate or amino acid compared to men [52, 53]. From a nutritional point of view, there is no reason to think that women will respond differently from men when being fed with the same relative amount of protein or the majority of amino acid adjusted per kg of body weight or lean body mass [54].

Table 30.1 summarizes the principal characteristics of whey, casein, and soy and shows how the combination of these three protein sources in a blended mixture can be useful to potentiate adaptation to exercises and obtain health benefits.

Even though rapidly digested proteins may stimulate muscle protein synthesis, the associated high amino acid oxidation rates that could be produced after a large single dose can negatively affect protein retention over time [18]. This finding together with the difficulty in ingesting several frequent smaller portions of whey protein has led sports nutrition manufacturers to design blends of proteins involving different sources as the best strategy to obtain a timed release of amino acids into circulation. This feeding pattern has been associated with greater muscle protein synthesis rates and lean body mass gains [37]. The difference in

**Table 30.1** Prospective benefits derived from blending whey, casein, and soy protein based on their unique attributes

| Characteristic | Whey (W) | Casein (C) | Soy (S) | W+C+S |
|---|---|---|---|---|
| High quality | Yes | Yes | Yes | Yes |
| Absorption rate | Fast | Slow | Medium | Longest |
| Leucine | Yes | – | – | Yes |
| Glutamine | – | Yes | Medium | Yes |
| Arginine | – | Medium | High | Yes |
| Antioxidant | – | – | Yes | Yes |
| Hyperaminoacidemia | Short | Long | Short | Longest |
| Muscle protein synthesis stimulation | High | Low | Medium | Highest |

Adapted from Paul, G. L. 2009. The rationale for consuming protein blends in sports nutrition. J Am Coll Nutr 28 Suppl: 464S-472S

digestion rates provided by the combination of whey, soy, and casein will effect the prolonged and increased levels of plasma amino acids resulting in greater muscle amino acid uptake [55]. Blending whey, casein, and soy protein stimulates a more balanced amino acid profile, specifically BCAAs, glutamine, and arginine. This may confer an advantage by providing a wider range of benefits for exercise adaptation and health-related effect compared to a single protein source rich in only 1 or 2 of these key amino acids. However, the exact ratio of protein sources to accomplish this task has not been reported. Moreover, the possibility exists that endurance athletes, because of their specific needs, would benefit from a different protein blend than the mixture used by strength/power athletes. Additionally, no specific recommendations have been made for active women or female athletes.

Collectively, the above analysis indicates that for most athletes and active individuals, achieving competitive advantages plus optimizing recovery and gains are the most important variables. Considering the intake of only one type of protein source (whey, casein, or soy), it would appear that casein leads to the optimal total body protein state at rest. However, following exercise, it would appear that ingestion of a whey source or a combination of whey, casein, or soy proteins would be the best options.

### 30.3.2.5 Essential Amino Acids

Nutritional intervention leading to an acute increase in muscle amino acid availability at rest or early after resistance exercise is a vital stimulus for promoting muscle protein synthesis [20, 36].

**Table 30.2** Essential and nonessential amino acids

| Essential | Nonessential |
|---|---|
| Isoleucine[a] | Alanine |
| Leucine[a] | Arginine[b] |
| Valine[a] | Asparagine |
| Lysine | Aspartic acid |
| Methionine | Cysteine |
| Phenylalanine | Glutamic acid |
| Threonine | Glutamine |
| Tryptophan | Glycine |
| Histidine | Proline |
| | Serine |
| | Tyrosine |

Source: Reeds, PJ. Dispensable and indispensable amino acids for humans. J. Nutr. 2000:130:1835S–1840S

[a]Branched chain amino acids

[b]Not considered essential as most humans synthesize arginine

Regardless of sex when resistance exercise is followed by an increase in amino acid's availability, the rate of muscle protein synthesis is increased more so than that observed with either exercise or amino acids alone [36, 56]. Table 30.2 lists the EAAs.

Research indicates that ingesting 3–6 g (>45 mg to 86–95 mg/kg body weight) prior to and/or following exercise can significantly stimulate protein synthesis [19, 22, 57]. Although more data is needed, there appears to be strong theoretical rationale and some supportive evidence that EAA supplementation may enhance protein synthesis and training adaptations [22].

Hence, an "anabolic window" appears to exist from immediately to a few (1 to 2–3) h after a workout. This helps to explain why weight

training combined with feeding protein during the early hours after exercise leads to significant increases in lean body mass compared to delaying the same feeding [58, 59] and why the failure to increase glucose and amino acid availability during recovery could significantly delay or even hinder the recovery process resulting from reduced muscle protein synthesis and glycogen restoration [60].

In sedentary or slightly active healthy older women, twice-daily between-meal ingestion of 7.5 g/d of EAA (15 g total) for 3 months has been shown to effectively retard the loss of muscle mass associated with aging. This regimen may stimulate muscle synthesis in the period between meals when blood amino acid concentration tends to decline [56]. However, it seems that elderly men and women could show a blunted muscle protein synthesis response when given the minimal effective 3 g (45 mg/kg body weight) dose of EAA proven to increase synthesis in younger counterparts [61]. The mechanism facilitating the impaired dose response in muscle protein synthesis in the elderly compared with the young is still elusive.

The possible existence of a leucine "threshold" that must be surpassed after protein or EAA ingestion in order to stimulate muscle protein synthesis above rest would explain not only the minimal effective dose for improving exercise benefits but also the observed varied responses between young and older men/women. This leucine threshold seems to be lower in younger rather than older populations. This could be one of the explanations as to why younger muscles are highly sensitive to the anabolic actions of leucine, as ~1 g of orally ingested leucine would be sufficient to elicit significant gain in muscle protein synthesis above the resting state [50]. Meanwhile, 2 g of leucine have been needed to produce a comparative response in the elderly [62]. Thus, when considering protein feeding strategies to stimulate muscle hypertrophy, a protein source with high leucine content and rapid digestion kinetics or an EAA dose that includes 2 g of leucine should be considered in order to promote a transient leucinemia "spike" for an effective option [61].

This recommendation is in line with recent recommendations from the International Society of Sports Nutrition [59]. In order to elevate muscle protein synthesis and favor a positive muscle protein balance, it is advisable to distribute the daily protein ingestion equally across four to five meals. For example, in addition to the protein included in typical food (milk, meat, etc.), the consumption of 15–30 g (~300 mg/kg body weight) of high-quality protein containing 4–8 up to 15 g (>50 to ~150 mg/kg body weight) of EAA in the form of powder or protein bar supplements could be adequate for making shakes [46, 63, 64]. The typical American or some European diets distribute protein intake unequally in such a manner that lower amounts of protein are consumed with breakfast (>10–14 g of protein) when compared to dinner (>15 g). The use of protein powder or bar supplements can help to maintain an equal distribution of protein ingestion through the day. This strategy has been shown to be more effective for maintaining muscle mass when compared to an unequal distribution of protein contained within the daily meals.

#### 30.3.2.6 How to Use Protein and Amino Acid Supplementation

As stated above, there is no special recommendation for female or male athletes. In general, to optimize the positive outcomes elicited by any exercise program, the ingestion of food containing a 50 mg/kg of EAA seems to be the minimum amount or threshold to trigger a significant increase in muscular protein synthesis [64]. However, research has shown that ingesting a combination of EAA or protein including 1–2 g of leucine with carbohydrates and other natural compounds such a creatine immediately prior to the workout may stimulate the most profound changes [45, 65, 66]. Ingestion of carbohydrate alone after exercise causes marginal improvements in overall protein synthesis while maintaining a negative net protein balance. When combined with protein or EAA, carbohydrates stimulate a more powerful insulin secretion that enhances cellular hydration and nutrient intake, favoring a more positive environment to attenuate catabolism and stimulate a greater anabolic response [60].

Unfortunately, there is no conclusive evidence regarding what time frame is the most optimal period to ingest a protein or amino acid-carbohydrate supplement in order to optimize muscular adaptation to exercise. Many studies have provided support for ingestion of different nutrients before, during, or immediately after to several hours (1–3 h) postexercise in order to promote increases in protein synthesis [21, 58, 66–68]. Similar changes have been found in studies that have administered amino acids alone or with carbohydrate during and immediately upon completion of an acute exercise bout, 1, 2, and 3 h after completion. However, ingesting nutrients before an exercise bout may have the most benefit of all the time points observed [66].

From the currently published research, the optimal dose of a multinutrient supplement containing high-quality protein or EAA with carbohydrates is difficult to determine. However, in order to stimulate a more powerful anabolic response, just prior or during an intermittent high-intensity or resistance training workout, it would be advisable to ingest a beverage containing 0.4 g/kg of carbohydrates mixed with 50 mg/kg of EAA or approximately 100 mg of a high-quality whey protein dissolved in 0.750–1 1 of water. This beverage will provide a nutrient concentration of around 8 % in order to be adequately assimilated throughout the digestive tract [64]. An optimal post-workout meal aimed to potentiate exercise benefits could include 1.2–1.5 g/kg of high-glycemic load carbohydrates with 50–150 mg/kg of EAA or 115–300 mg/kg high-quality whey protein [64].

#### 30.3.2.7 Branched Chain Amino Acids

The EAAs leucine, isoleucine, and valine form what is referred to as BCAAs. BCAAs comprise about 1/3 of the total muscle protein pool [69] and act as a primary nitrogen source for glutamine and alanine synthesis in muscle [70]. Unlike most free form AAs, BCAAs are not degraded in the liver. Twenty to thirty percent of ingested BCAAs from food or supplements are metabolized by the intestine; the rest are rapidly absorbed in plasma. Muscle tissue BCAA uptake primarily occurs from plasma BCAAs [71].

Supplementation with BCAAs has been shown to acutely stimulate protein synthesis, aid in glycogen resynthesis, delay the onset of fatigue, and help maintain mental function with aerobic-based exercise. Researchers have concluded that consuming BCAAs (in addition to carbohydrates) before, during, and following an exercise bout could be recommended safe and effective [22].

One of the proposal effects of BCAA supplementation relates to a phenomenon known as central fatigue, which signifies that mental fatigue in the brain can adversely affect physical performance in endurance events. The central fatigue hypothesis suggests that low blood levels of BCAAs may accelerate the production of the brain neurotransmitterserotonin,or5-hydroxytryptamine (5-HTP), and prematurely lead to fatigue [72]. Tryptophan, an EAA, is a precursor of serotonin which can be more easily transported into the brain (to increase serotonin levels) when BCAA plasma concentrations decrease [72]. This occurs during prolonged exercise due to an increased BCAA intake by the active muscle [72]. In addition, the increased release of fatty acids into the blood during endurance exercise displaces tryptophan from its place on albumin and facilitates the transport of tryptophan into the brain for conversion to serotonin. Thus, the combination of reduced BCAAs and elevated fatty acids in the blood causes more tryptophan to enter the brain and more serotonin to be produced, leading to central fatigue [72, 73]. Due to these metabolic processes, it has been hypothesized that BCAA supplementation can help delay central fatigue and maintain mental performance in endurance or extremely long-lasting physical activities [67].

The positive effect of administration of BCAAs in reducing fatigue in exercise may also be due to its possible influence on other biochemical events in the brain. BCAAs may be involved as precursors for the synthesis of several neurotransmitters. In this model, leucine would act as a neurotransmitter per se, being that one of its role is to counteract fatigue. Alternatively, a BCAA (e.g., leucine) may be converted into a metabolite, which is a novel neurotransmitter that also could reduce fatigue [74].

Other documented effects of BCAA supplementation are the ability to attenuate delayed-onset muscle soreness (DOMS) and suppress the decrease of muscle strength after an unaccustomed high-volume resistance training workout in untrained women [75]. These effects have been shown to be more pronounced in women as compared to men when BCAAs were ingested at 77 mg/kg body weight (males) or 92–100 mg/kg (women) before exercising [76].

#### 30.3.2.8 How to Use Branched Chain Amino Acids as a Supplement

Due to the possible benefits to the recovery process, stimulating protein synthesis, aiding in glycogen resynthesis, delaying the onset of fatigue, and helping to maintain mental function, it is advisable to ingest BCAAs with carbohydrates before, during, and following an exercise bout. Before and during exercise, BCAAs could be added to a sports drink with 6–8 % of carbohydrate concentration [64]. In order to improve glycogen recovery and stimulate protein synthesis after exercise, the addition of BCAAs to a carbohydrate-rich beverage with a BCAAs/CHO ratio of 1:4 (67)–1:7 (64) has been recommended.

#### 30.3.2.9 L-Glutamine

While amino acid usage tends to be more prevalent in male athletes, female athletes are also consuming amino acid supplements [2]. Aside from amino-stacked supplements, female athletes also admitted to have listed taking glutamine individually. Glutamine is a common component of weight-gain products. A small percentage of female athletes report taking weight gainers as well [2].

As stated before, glutamine is the most abundant amino acid in plasma and skeletal muscle representing more than 60 % to total free plasma amino acid [77]. Glutamine plays a number of important physiological roles by acting as an antioxidant, immuno-suppressor, and anticatabolic agent [78]. Amino acids along with alanine, glutamine acts as nontoxic nitrogen carrier which makes these important carbon donors for glycogen synthesis in the liver [79]. Despite its important physiological roles, there is no compelling evidence to support glutamine supplementation in terms of increasing lean body mass or muscular performance in male or female athletes [22]. In addition, previous reports have not indicated any evidence about the protective effects of glutamine supplementation against immunodepression caused by high-intensity and prolonged endurance exercise sessions [80]. However, in some high-intensity endurance exercise conditions where plasma glutamine concentration could fall to 20–25 %, ingestion of an amino acid and in particular L-glutamine could be advantageous for cells of the immune system, including neutrophils [81]. In addition, supplementation with L-glutamine seems to protect mitochondrial integrity and thereby potentially reduce exercise-induced apoptosis [82].

In conclusion, in spite of the lack of convincing evidence regarding the effects of glutamine to maintain or gain muscle mass and increase physical performance, animal experiments suggest that the usefulness of glutamine supplementation is more related to its capacity as an immunoprotector. As an immunoprotector, glutamine could regulate neutrophil activity and counteract the negative effects of exercise on specific neutrophil functions such as apoptosis and nitric oxide (NO) production [82].

##### How to Use Glutamine as a Supplement

In athletes (both males and females), a dose of glutamine 40–50 mg/kg body weight ingested before and within 2 h after a workout has been proposed to improve anabolic cellular environments or attenuate immuno-depression after prolonged high-intensity endurance exercise [64].

#### 30.3.2.10 L-Arginine

Arginine is, conditionally, an EAA synthesized from ornithine and citrulline. Arginine is a precursor amino acid for several important components such as creatine, phosphate, polyamines, and NO. Supplementation with L-arginine is claimed to promote vasodilatation by increasing NO production in the active muscle during exercise, improving muscular strength, power, and recovery through increased substrate utilization

and metabolite removal, such as lactate and ammonia [83]. In males, supplementation with L-arginine has shown to be effective for improving endurance exercise performance and possibly improve maximal muscle strength and power [83]. Regarding females, only few studies have analyzed the effects of L-arginine supplementation. Fricke et al. observed no significant difference in maximal isometric grip force (N) and jump performance variables after 6 months of L-arginine supplementation (18 g) in postmenopausal women (23, 84).

Even though current research in men is promising regarding the positive effects of L-arginine supplementation, more research is needed in order to give conclusive recommendation about L-arginine use in female athletes or active women.

#### How to Use Arginine as a Supplement

L-arginine supplementation appears to be safe and well tolerated when used between 3 and 15 g orally in healthy subjects. No further dosages have been used in similar groups with the purpose of improving performance. As stated above, further studies (particularly in active females) are required to determine its potential ergogenic aid as well as associated side effects. In general, it has been proposed for athletes to ingest 3–5 g/d before training or competition as a possible effective strategy to improve performance [64].

### 30.3.2.11 Carnitine

Carnitine is synthesized from lysine and methionine, which are the amino acids that soy protein is lacking. Mammals synthesize carnitine and most healthy humans can synthesize it even with a diet lacking in animal protein. In times of low dietary consumption, daily losses through excretion are lowered to compensate for this reduction [85].

When marketed as L-carnitine, an isomer of carnitine, it has been touted as a fat burner. As such, athletes have taken it for weight loss, increased muscle mass, and enhanced β-oxidation. While L-carnitine is involved in transportation of fatty acid chains across the membrane, studies have generally shown it to be an effective weight-loss agent for obese subjects and inconclusive for healthy non-obese subjects [85].

Athletes may also take L-carnitine to enhance recovery high-intensity exercise. Research findings include decreased creatine kinase, production of purine, reduced free radical formation, and less reported muscle soreness [85].

L-carnitine is available as a supplement and also as a prescription. Recommendations for daily supplementation are 2–3.5 g/d. Amounts in excess of 4 g/d may cause gastrointestinal distress. A lethal dose (LD) of 630 g/d in humans has been extrapolated from studies using rats [85].

### 30.3.2.12 Creatine

Creatine is traditionally taken by men more than women [1, 2]. However, a large enough percentage of women are taking it to make creatine worthy of discussion.

Creatine is composed of two non-EAAs (arginine and glycine) and one EAA (methionine) [86]. The average daily requirement is 2 g/d [8, 86]. The body makes 1–2 g/d via the liver, kidneys, and pancreas. Meat and fish also contain creatine. Vegetarian athletes have lower muscle creatine than omnivores [87].

Creatine is an important part of the adenosine triphosphate phosphocreatine (ATP-PC) energy system. Phosphocreatine (PC) is used to phosphorylate adenosine diphosphate (ADP) to ATP during high-intensity maximal muscle contractions, resulting in ATP and free creatine. Creatine is rephosphorylated during periods of rest via aerobic pathways (mitochondrial creatine kinase). Generally speaking, an average person has enough phosphocreatine stores to supply ATP for anaerobic activities up to 10–15 s [8, 86]. Since the ATP-PC system is short-lived, glycolysis becomes the dominant energy system as PC stores are depleted and ADP builds up. Rest periods required to regenerate PC are typically >3 min [8].

Creatine supplementation increases muscle creatine stores in subjects who do not already have maximal stores. Some athletes who consume high quantities of meat or fish are thought to have maximal or near-maximal stores already [86]. Increased muscle creatine stores may lead to more PC to regenerate ATP, a faster recovery time to rephosphorylate free creatine and in some

cases buffer lactic acid [8, 86–88]. However, some researchers have reported no ergogenic effect on anaerobic exercise [4, 17, 18]. Furthermore, aerobic endurance-type activities and submaximal efforts will not show any improvement with creatine supplementation [8].

Creatine is usually taken in the form of creatine monohydrate with a loading phase (5 g/d, 4 Xs/d) up to 7 days. A maintenance phase of 2 g/d is recommended for 3 months. This protocol has shown increases of muscle creatine 10–25 % [86, 89]. Consumption of creatine with a carbohydrate enhances absorption while caffeine will inhibit absorption [86].

Consumption of creatine above the recommended dosages may result in excretion. Since creatine consumption has been linked to dehydration cases, it is best to advise the athlete to consume plenty of water while taking creatine, especially when exercising in the heat [86].

#### Benefits and Side Effects of Creatine Using the Recommended Loading-Maintenance Creatine Protocol

There are reports of increased ability to perform more reps at a given percent of 1 repetition maximum (RM), enhanced power output in cyclists, improved track sprint times by 1–2 %, increased muscle mass, and lower percent body fat with creatine supplementation [8, 86, 87, 89, 90]. Other studies have not demonstrated any improvement of these same variables [8, 86–88, 90]. Creatine supplementation was ineffective in subjects 60 years and older or in people with high creatine levels before supplementation. These subjects are referred to as nonresponders [86, 88]. Although tennis has various components (i.e., serve or sprint to the net) that would utilize the ATP-PC system, no benefits of creatine supplementation have been reported with the stroke or sprint performance [8]. Some ergogenic findings in exercise lab environments may not translate into actual competition.

Side effects of creatine include weight gain from water retention, gastrointestinal discomfort, and muscle cramps [8, 86, 88]. Increased weight gain may hinder performance in mass-dependent sports. Additionally, it has been speculated that long-term use may downregulate the creatine transporter protein and render creatine supplementation less effective [88].

### 30.3.3 Fat Burners and Energy Supplements

#### 30.3.3.1 Ginseng

Ginseng is a widely used herbal supplement in many fat burners and energy drinks. It is reported to improve mood, performance, and alertness and increase fat utilization. There are two prevalent types of ginseng used: Chinese ginseng (*Panax ginseng*) or Siberian ginseng (*Eleutherococcus ginseng*). Other less commonly used ginseng plants include Japanese ginseng (*P. japonica*), Tienchi ginseng (*P. notoginseng* or *P. pseudoginseng*), Dong Quai (*Angelica sinensis*), or American ginseng (*P. quinquefolius*) [91].

Chinese ginseng is an herbal supplement that has been evaluated in human performance studies and was reported to exhibit promising effects to improve strength and aerobic capacity [91, 92]. However, other studies have shown no improvements in maximal oxygen consumption ($VO_{2max}$), strength performance, or postexercise recovery [91–93]. This could be due to varying populations, different dosages, types of ginseng, or various durations of the studies [93–95]. The general dosage guidelines for Chinese ginseng are 1–2 g/d. This may vary based on type of preparation (powder vs. root extract). Chinese ginseng is usually supplemented for a certain duration in combination with athletic training [91]. The majority of research suggests that Chinese ginseng is not ergogenic.

Eight weeks of supplementation with Panax ginseng in 24 healthy active women did not result in improvements of supramaximal exercise testing or recovery values [96]. Participants consumed 400 mg of Panax ginseng for 8 weeks or consumed their normal diet. Prior to the treatment period and at the end of treatment, participants performed a Wingate cycle ergometer test. There were no significant differences for mean power output, peak power output, rate of fatigue, or recovery heart rate (HR) [96].

There have been some adverse effects reported with Chinese ginseng use. As ginseng is a stimulant, it may cause sleeplessness or nervousness. Since many supplements contain ginseng plus caffeine, this side effect may not be due entirely to ginseng. Hypertension, dermatological problems, morning diarrhea, and euphoria have also been reported with ginseng use [91, 92].

Siberian ginseng is in the same plant family as Chinese ginseng. However, the two herbs are distantly related. It does contain saponins, but they are different than the ones found in Chinese ginseng. Many of the early studies involving Siberian ginseng did not give experimental design details or data, nor were they peer reviewed. Subsequent studies have failed to show an ergogenic effect [87, 97]. Dowling et al. [97] examined the effects of Siberian ginseng on submaximal and maximal exercise tests in 20 trained distance runners. After 8 weeks of supplementations, no differences existed between placebo and treatment groups for $VO_2$, ventilation (VE), respiratory exchange ratio (RER), HR, or rating of perceived exertion (RPE) in both the submaximal runs at 10 k race pace or maximal treadmill tests. There were also no differences in lactate and time to exhaustion during the maximal testing [97]. Without further research demonstrating an effect, it would be difficult to recommend supplements containing Siberian ginseng for athletic activity.

#### 30.3.3.2 Echinacea

Multiple studies have reported that female athletes commonly use Echinacea in order to boost their immune system [1, 3, 5]. In fact, Echinacea supplementation is commonly listed in the top 5 supplements which female athletes consume. In fact, research suggests that supplementation with various Echinacea species or preparations does not prevent or shorten the duration of a cold [98–100].

Turner [28, 99] treated 50 participants with 900 mg/d Echinacea and 42 participants with placebo and found no significant difference between rhinovirus infection (44 and 57 %, respectively) and clinical colds (50 and 59 %, respectively). The duration of the treatment period in this study was 2 weeks [99].

Turner also examined the dosage of 900 mg/d in three different preparations of *Echinacea angustifolia* on the varieties of Echinacea recommended to treat a cold [100]. Participants were assigned to receive the treatment either as a prevention of or treatment for a cold. After a week of supplementation, participants in the prevention group were given a nasal spray containing a rhinovirus. There was no positive treatment effect of Echinacea with regard to infection prevention. Treatment of infected participants did not result in any significant effects on alleviating rhinovirus symptoms [100].

In agreement with Turner and colleagues, Barett et al. [98] found that supplementation with an Echinacea supplement made of *Echinacea purpurea* (675 mg) and *Echinacea angustifolia* (600 mg) did not reduce the duration or ease symptoms of a cold. Participants in the treatment group consumed 2 tablets, 4 times/d the first day and 1 tablet/d for the next 4 days. Symptoms were tracked for 2 weeks. There were no significant differences in cold duration or relief of symptoms in any group studied [98].

#### Echinacea and Erythropoietin

Recently, a study has been published suggesting that Echinacea could be an ergogenic aid, not used to treat cold symptoms, but to increase Erythropoietin (EPO) and subsequently increase $VO_{2max}$ [6]. Whitehead et al. [6] administered 8,000 mg/d (most over-the-counter [OTC] Echinacea supplement doses are far less than this) *Echinacea purpurea* to 24 healthy male college students for 4 weeks. Blood samples were taken pre-study and then at 7 days, 14 days, 21 days, and 28 days. After day 7, EPO values were increased over baseline as compared to the placebo group. However, while at day 21 even though EPO values were still significantly elevated, they had started to decline. By day 28, EPO values were no longer significantly elevated over baseline or different from the placebo group. Red blood cell (RBC) count was not different between groups or within groups. Submaximal $VO_2$ was ~1.5 % lower from pre-study values during the first 3 stages of the maximal exercise test in the Echinacea group. $VO_{2max}$ was ~1.5 %

higher in the Echinacea group [6]. From the results of this study, it appears that high doses of Echinacea may increase $VO_{2max}$ while allowing an endurance athlete to perform the same amount of work at a lower submaximal $VO_2$. It also appears that Echinacea may increase EPO up to 2 weeks and then wane. In this study, the maximal exercise test was given at 28 days. It would be interesting to know what $VO_2$ values looked like at 2 weeks when EPO was at the highest levels. It is also worth noting that, in this study, Echinacea increased EPO without increasing RBC count [6]. *Future research should be conducted before recommending Echinacea to increase* $VO_2$. Increases in EPO typically increase RBC count and can result in serious side effects such as increased viscosity of the blood and increase the likelihood of a cardiac event. High levels of EPO may also cause an athlete to fail a drug test as well.

#### 30.3.3.3 Ephedra

Ephedra contains ephedrine and other alkaloids which are sympathomimetic possessing α- and β-agonistic properties. It facilitates catecholamine release and stimulates the central nervous system (CNS) [86, 91, 101]. Ephedra is used as an energy booster, fat burner, and athletic performance enhancer. Ephedrine is also a component of cold remedies and some prescription drugs [91, 101]. Ephedra may also go by the name of Ma Huang.

Ephedra has been reported to aid in weight loss and was a popular ingredient before the 2004 ban by the Federal Drug Administration (FDA) [102]. The ban was overturned on an appeal and reinstated after a further appeal. This had led to consumer confusion regarding the legality of ephedra found in supplements. Currently, the FDA ephedra ban specifically lists "ephedrine alkaloids" as an adulterated substance that presents unreasonable risks [102]. Under the definition of the ban, ephedrine alkaloids are listed as raw botanical and botanical extracts from the following sources: species *Ephedra sinica Stapf, Ephedra equisetina Bunge, Ephedra intermedia var. tibetica Stapf, Ephedra distachya L., Sida cordifolia L., and Pinellia ternata (Thunb*). Some dietary supplement companies are manufacturing products using other ephedra varieties, most of which do not produce the same effects as the alkaloids from the variety banned by the FDA. Even though ephedra is not legal in supplements, the ban does not regulate the usage of synthetic ephedrine in cold, allergy, or asthma OTC medications.

Ephedra is often paired with caffeine in supplements to achieve an enhanced response [86, 101, 103]. Unfortunately, many of the studies involving ephedra and athletic performance also included caffeine. Shekelle et al. [103] found few controlled studies with or without caffeine to include in their meta-analysis concerning the efficacy of ephedra. Even with the small number of studies included, the researchers felt confident to conclude that there is not enough evidence to support the claim that ephedra enhances athletic performance. Other reviews of ephedra and athletic performance confirm this conclusion as well [86, 91, 104]. Most studies have found claims of weight loss to be substantiated [101, 103]. Again, many of these studies involve a synergistic relationship between ephedra and caffeine.

As ephedra has been indicated for weight loss, it is a tempting supplement to the female athlete concerned about body composition. Ephedrine abuse has been reported in female weight lifters [4]. Gruber and Pope [4] interviewed 64 competitive female body builders in regard to ephedrine usage. Of the 64 women interviewed, 36 reported daily usage of ephedrine for at least a year with many reporting usage for 5–10 years. The most reported dosage was around 120 mg/d; however, there seems to be an acclimation effect and the women reported having to increase dosages or develop more frequent dosing schedules in order to maintain the same effects. While the authors of this study did not assess body composition, 17 % of the female body builders using ephedrine did report having the occurrence of at least one bone fracture. Several of the women also reported amenorrhea and a history of eating disorders. Loss of body fat was the reason given for ephedrine abuse by 35 of the 36 body builders. Reasons for continued use despite the side effects were to reduce the symptoms of withdrawal (i.e., fatigue or weight gain) [4].

Even after the ephedrine ban, NCAA athletes still report taking ephedrine [10]. In 2009, lacrosse and volleyball were the sports with the highest reported usage of ephedrine (1.9 and 1.7 %, respectively). Female athletes in other sports surveyed also reported ephedrine usage after the ban; however, the occurrence of use was less than 1 % [10]. Over 7,800 women were included in this study and the percent of ephedrine use for all female sports combined was 0.9 % or about 70 athletes. Sports surveyed included basketball, field hockey, golf, lacrosse, soccer, softball, swimming, tennis, track, and volleyball [10]. While the number of users seems relatively small, it is important to remember that each of the reported ephedrine users is subject to some very serious side effects. There may also be users who are reluctant to admit usage since ephedrine is a banned substance or because it is packaged in an herbal remedy and the athlete is unaware of its presence. Reported side effects of ephedra are summarized in Table 30.3. Additionally ephedra has also been linked to several deaths [4, 86, 92, 101, 104, 105].

When ephedra was banned in 2004, a product by the common name of bitter orange took its place as a fat-burning agent. Bitter orange (*Citrus aurantium*) contains synephrine and has similar effects as ephedra. It is also used in combination with caffeine [20, 91]. The National Institute for Health (NIH) advises caution when consuming supplements containing bitter orange as the side effects are similar to ephedra. Synephrine is currently banned by NCAA and the World Anti-Doping Agency (WADA).

If your athlete wants to take an OTC sinus congestion product, advise them to read the packaging for ephedrine and pseudoephedrine. These products will likely be behind the pharmacy counter even though they are an OTC. Both of these ingredients are currently on the banned substance list for NCAA and WADA. Decongestant products available on store shelves contain phenylephrine, which is currently (2012) not banned by NCAA or WADA. As the banned substance list is constantly being updated, you and your athlete need to consult the list for currently allowed decongestants.

**Table 30.3** Side effects of ephedra

| |
|---|
| Headache |
| Tremors |
| Hypertension |
| Arrhythmias |
| Insomnia |
| Nervousness |
| Increased heart rate |

Sources: Calfee R, Fadale P. Popular ergogenic drugs and supplements in young athletes. Pediatrics. 2006;117(3):e577–e589. Gruber, AJ. and Pope Jr, HG. Ephedrine abuse among 36 female weightlifters. Am J Addict. 1998; 7: 256–261. Powers ME. Ephedra and its application to sport performance: another concern for the athletic trainer? J Athletic Training. 2001;36(4):420–424

#### 30.3.3.4 Caffeine

Caffeine is widely used by athletes and nonathletes for various reasons. Varsity females listed enhanced performance, more energy, alertness, and taste as reasons why they take caffeine. Athletes generally consume their caffeine via beverages such as coffee, tea, or soda. It is also consumed in caffeine tablets, energy bars, and even chocolate [1]. Many energy drinks and gels contain caffeine. Caffeine is allowed up to a certain quantity by the NCAA and WADA. You should advise your athlete to read the current guidelines regarding the amount of caffeine allowed by these agencies and the amount of caffeine in the products they ingest.

Caffeine's main mode of action is due to its structure. The chemical structure resembles adenosine and will bind to adenosine receptors. Caffeine also stimulates the release of epinephrine [91]. Caffeine has also been reported to enhance fat oxidation and spare muscle glycogen. It is widely consumed although it is not necessary for metabolic functions. People who consume caffeine regularly may become habituated to it and the physiological effects will be blunted. These subjects would also be classified as nonresponders.

One important question asked by athletes was how does caffeine tablet ingestion differ from drinking coffee [1]. While coffee does contain caffeine, it also contains many other compounds which form more metabolites in the body [106, 107]. Coffee has also shown

some ergogenic effects, just not to the same magnitude as ingesting caffeine alone [106]. Coffee would have to be consumed in large amounts to equal the caffeine typically found in tablets. When answering the athletes' question of coffee or tablets, coffee would most likely result in less of an ergogenic effect unless the subject was naïve to caffeine.

#### High-Intensity Exercise

During high-intensity exercise, caffeine could lower the rate of RPE and increase glycolytic activity performance. Conversely, caffeine is also associated with an increase in blood lactate [108]. However, not all anaerobic studies are in agreement. Greer et al. [109] found no ergogenic effect or increase in blood lactate of subjects studied when performing the Wingate test.

#### Strength

The effect of caffeine on strength activities is not conclusive. Previous studies have suggested caffeine may have a direct mode of action on muscle function. This proposed mode of action suggests that caffeine causes the sarcoplasmic reticulum to release more calcium, allowing the muscle to sustain force production for a longer period [106, 107, 109].

#### Endurance

Endurance exercise and caffeine have been studied extensively. The main concept is that caffeine causes fatty acid mobilization and glycogen sparing [106, 107]. Many of the studies investigating glycogen sparing were not designed to cause sufficient glycogen depletion with or without caffeine. It is unlikely that the ergogenic effects of caffeine on endurance exercise are due to glycogen sparing [106, 107]. Likewise, research has not supported the theory of caffeine-enhanced fatty acid oxidation during exercise. While many studies report no decrease in RER with caffeine and exercise, caffeine does promote lipolysis at rest [106]. It is clear that caffeine does prolong endurance exercise. The mechanism behind that is not clear. Graham [106] suggests more future research to explore alternative hypotheses.

#### Adverse Effects

It is often suggested that caffeine usage will result in dehydration. Caffeine is a mild to moderate diuretic. Its effect is seen mainly at rest. Studies have found no effects of caffeine and dehydration after prolonged exercise so long as athletes are replacing fluids adequately [106, 107]. During prolonged exercise epinephrine-induced renal vasoconstriction causes blood flow to be diverted from the kidneys to the exercising muscle [107]. The diuretic effect of caffeine is minimized during exercise.

Other side effects of caffeine include tachycardia with exercise, increased blood pressure, gastrointestinal distress, and habituation/addiction to caffeine [106, 107]. There are also side effects associated with discontinuing caffeine usage. Headache, fatigue, and possible flu-like symptoms may occur [106, 107]. Combining caffeine with ephedra (Ma Huang) is potentially dangerous and should be avoided [101, 107]. Caffeine also inhibits the ergogenic effect of creatine [86, 106].

#### Dosage

For endurance exercise, 3–5 mg/kg is sufficient to produce an ergogenic effect. Since many beverages are variable in the amount of caffeine they contain, tablets are probably the most effective way to get the dosage required to get an ergogenic effect [107]. Sport gels are also available with caffeine added. Athletes should read the packaging to know if they are buying a sport gel with or without caffeine prior to injecting these subjects.

### 30.3.3.5 Energy Drinks

Most energy drinks contain sugar, caffeine (derived from guarana), taurine, and ginseng. Some energy drinks may also contain B vitamins. Energy drinks contain a unique combination of these ingredients and may contain other substances as well. It is important that athletes consult the allied health professionals with the drink they chose to consume. The allied health professional should inform the athlete about the ingredients in the beverage and the effect of energy drinks on sports performance. Currently there are

not many well-controlled studies examining the effects of energy drinks on performance. It is difficult to perform studies using all different brands of energy drinks since the market keeps on growing. However, there are a few well-controlled studies examining the effects of a representative major brand energy drink on exercise endurance and sprint performance.

### The Effects of Energy Drinks on Sprint Performance and High-Intensity Exercise

Astorino et al. [110] examined the effects of acute consumption of the energy drink Red Bull on sprint performance in female collegiate soccer players. Soccer players were asked to drink either one can of Red Bull or the equivalent amount of the placebo beverage 1 h before performing multiple *t*-tests. *t*-tests were performed as all-out sprints in three bouts of 8 *t*-tests. Heart rate was monitored with a chest strap HR monitor. There were no differences between the placebo and Red Bull in regard to the RPE, HR, or sprint times. The researchers concluded that a single serving of an energy drink does not provide the female athlete with any ergogenic benefit [110].

Similar results were found when sugar-free Red Bull was given to physically active male and female college students (Mean $VO_{2max}$ 45.41 ± 6.3 ml/kg/min) before performing a run to exhaustion test [111]. Participants consumed either a placebo beverage or a sugar-free Red Bull 60 min prior to a run to exhaustion treadmill test at 80 % $VO_{2max}$. Trials were separated by 1 week. Run time to exhaustion was not significantly different between either group nor was RPE. Blood lactate levels were significantly elevated postexercise in both trials, but there were no differences between groups of subjects. The consumption of sugar-free Red Bull did not influence substrate utilization during this high-intensity run to exhaustion in physically active college-aged males and females [111].

### The Effects of Energy Drinks on Endurance Performance

The effect of an energy drink on endurance performance was examined by Ivy et al. [112]. Participants were male and female competitive cyclists ($VO_{2max}$ 54.9 ± 2.3 ml/kg/min). The dosage for the energy drink was determined by the amount of caffeine needed to elicit an ergogenic effect. Participants consumed the equivalent of two cans of Red Bull (500 ml) or placebo 40 min prior to exercise. A time trial was performed on the cycle at 70 % watt max, 90 rpm for approximately 1 h. On average, participants improved their performance 4.7 % in the treatment group. By 50 min of exercise, HR was elevated above the placebo group (173 ± 4.0 to 166.5 ± 5.8 bpm, respectively) and remained elevated at completion of the exercise session (172.2 ± 4.3 to 170.5 ± 5.7 bpm, respectively). Epinephrine was elevated above the placebo group at 30 min (461.0 ± 122.5 to 195.6 ± 43.8 pg/ml, respectively), 50 min (470.8 ±96.9 to 219.5 ± 293.9 pg/ml, respectively), and after completion of exercise (1,011.8 ± 295.1 to 287.1 ± 69.7, respectively). Norepinephrine, cortisol, and B-endorphin were not different between the two treatment groups. There was also no difference in RPE. Blood lactate levels in the treatment group were elevated pre-exercise and remained elevated over the treatment group for the duration of the exercise. Except for the immediate period before exercise, blood glucose levels did not differ between groups. Glucose levels were significantly higher in the energy drink group prior to exercise ($p < 0.05$). Insulin levels were also elevated pre-exercise and also at 10 min into the exercise session in the energy drink group. Blood glycerol levels increased with longer exercise duration in both groups. Glycerol levels were significantly higher in the placebo group at 10 min ($p < 0.05$). No other time points were significantly different for glycerol values. Free fatty acids were significantly increased from 10 to 50 min of exercising in the placebo group ($p < 0.05$). After completion of exercise, there was no difference between either group in terms of free fatty acids. Substrate utilization, as determined from $VO_2$ and RER data, did not differ between the two groups. In general, carbohydrate usage declined with larger exercise duration and fat usage increased. The researchers demonstrated a non-significant trend ($p < 0.09$) where carbohydrate usage in the energy drink group was higher than

the placebo group from 30 to 50 min of exercising. The main finding of this study is that 500 ml of an energy drink containing a combination of caffeine, carbohydrates, taurine, and B vitamins can improve cycling performance in a 1 h time trial. The amount of caffeine in this study was towards the lower end of the range where caffeine has been shown to be effective (2.35 mg/kg body weight); therefore, the researchers speculate that the effects are due to the combination of ingredients in the energy drink. Increased epinephrine levels were likely elevated in response to the caffeine and could have spared muscle glycogen levels and allow for the increasing trend ($p<0.09$) of carbohydrate utilization the second half of the exercise [112]. It should be noted that participants consumed energy drink before exercise and consumed water during exercise in a climate-controlled laboratory. Athletes should be aware that results of the same energy drink could be different in outdoor race conditions when climate and nourishment can also affect exercise. Athletes should also be cautioned about the increased HR that consumption of an energy drink may cause and the dangers of cardiac drift in a hot and humid climate.

Kazemi et al. [113] examined the effects of two different taurine-containing energy drinks on time to exhaustion in field volleyball female athletes. The energy drinks compared were Phantom energy drink and Dragon energy drink produced in Austria. Phantom contained carbohydrate, caffeine, taurine, and a mixture of B vitamins. Dragon contained carbohydrate, protein, caffeine, no taurine, and a mixture of B vitamins. Participants consumed either a placebo or an energy drink (6 ml/kg body weight) 40 min prior to exercising. The exercise tests consisted of Bruce treadmill test performed four days apart for each trial. $VO_{2max}$ was increased in both the Phantom and Dragon energy drinks (6.9 and 4.8 %, respectively, $p<0.003$) over the placebo trial. Time to exhaustion was also increased in both drinks compared to placebo (Phantom 9.3 % and Dragon 6.5 %, $p<0.003$). Energy drinks also decreased RPE (Phantom −4.5 % and Dragon −5.8 %, $p<0.37$). There were no significant differences between the two energy drinks. The Dragon energy drink increased postexercise HR above the placebo group (2.2 % <0.019) and Phantom (1.3 %, $p<0.024$). There was no difference between Phantom and placebo in regard to HR. Phantom contained more caffeine than Dragon and also contained taurine; however Dragon increased HR compared to Phantom [113]. Neither energy drink contained as much caffeine as Red Bull.

**Conclusion: Are Energy Drinks Ergogenic?**

Not all types of exercise show improvement in performance upon ingesting energy drinks. Repeated sprint performance did not improve with consumption of an energy drink while endurance exercise appears to benefit. One important feature of these studies is that all of the studies had the participants consume the beverage 40–60 min prior to exercise but did not consume the beverage during exercise. Most studies consume one beverage, not multiple beverages. These studies were also conducted in climate-controlled labs.

The evidence regarding ergogenic effects of energy drinks is still an emerging field. The allied health professional should routinely scan the literature for new research on this front and also look on retail shelves to see what new drinks are being marketed. Become familiar with the ingredients and the amounts of the ingredients. Know the target audience they are marketed to. With regard to safety of energy drinks, there have been reports of cardiac events and/or death with consumption of energy drinks and exercise. However, there are also reports of exercise without elevated HR and improved performance.

### 30.3.4 Anabolic-Androgenic Steroids

Anabolic-androgenic steroids (AAS) are synthetic derivatives of testosterone [8, 88, 114]. Adolescent female athlete steroid usage is reported to be between 2 and 5 % [8, 9]. Collegiate usage may be even higher [114].

Women have considerably less testosterone than men, about 10 % that of men [114]. AAS act beneficially on the athlete in three ways:

anabolism, anticatabolism, and promoting aggression [8, 86, 88, 114]. Testosterone and AAS bind to androgen receptors inside the cytoplasm and are transported to the nucleus. This stimulates an increase in mRNA transcription and leads to increased synthesis of structural and contractile proteins [8, 86, 114]. It is widely reported that testosterone leaves few androgen receptors open for AAS to attach to and therefore AAS posses an indirect anticatabolic effect. This is thought to be accomplished by displacing glucocorticoids from their receptor sites [8, 86, 88]. A recent review by Evans suggests this may not always be the case. It is possible that the number of androgen receptors is upregulated with AAS usage [114].

The aggression reported by AAS usage is generally deemed beneficial from an athletic training viewpoint. While this may lead to more intense training, the emotional side effects may not be as beneficial [8, 86, 88].

Ergogenic effects appear to be limited to muscle hypertrophy and strength gains when performance is concerned along with lean body mass increases [8, 88]. AAS have not been found to produce an ergogenic effect in endurance activities [8]. Steroids have been used to speed up recovery from workouts [114].

#### 30.3.4.1 Adverse Effects

Far outweighing the benefits of AAS are the multiple adverse effects. Many steroid users report experiencing adverse effects [86]. These side effects are different for women than men [8, 114].

The most notable effects of AAS on women are the virilizing effects: hirsutism, voice deepening, male-pattern baldness, and enlargement of the clitoris. Females may also experience menstrual irregularities and reduced breast size [8, 114]. Some of these adverse effects are irreversible.

The cardiovascular system is also adversely affected by AAS. Blood pressure increases have been associated with AAS use [86, 114]. Left ventricular hypertrophy (LVH) was indicated as a side effect also [86]. Since increases in LV wall thickness (and/or increased blood pressure) can be a side effect of chronic athletic training (i.e., heavy resistance training, rowing), this effect may be a cumulative effect of AAS and training routine [115, 116]. Decreased high-density lipoprotein (HDL) is also associated with steroid usage, although not all studies have reported decreased HDL [8, 86, 88, 114].

Other adverse effects include hepatic abnormalities, dermatological problems, and psychological effects. Aggressive behavior is reported frequently along with mood swings. Depression and anger are reported as withdrawal symptoms [8, 86, 88, 114].

There are other serious repercussions of AAS usage that are related to injectable steroids, such as boldenone, trenbolone, or stanozolol (ester) [86, 114]. Injection-related complications include bacterial infection from non-sterile techniques (sharing multidose vials or needles). Hepatitis B, C, and HIV may also be spread this way. Inflammation from repeat use of the same injections site can also occur [114].

#### 30.3.4.2 Dosage

Since AAS are illegal, well-designed controlled studies in athletic performance are lacking. Suffice it to say most steroid users "stack" several anabolic steroid combinations (oral or injectable) in cycles followed by cycles of nonuse for a washout period [8, 86, 114].

### 30.3.5 Multivitamins

Information on vitamin and mineral requirements can be found in Appendices for Chap. 29 and specific needs for populations have been discussed in Chaps. 28, 29, 31, and 32. The focus of this section is the reasons why female athletes take multivitamins, iron, and calcium.

When surveyed, multivitamins were in the top 5 of dietary supplements taken by female athletes [1–3]. The reasons females gave for using multivitamins were as follows: *"recommended by family, friends, coaches or trainers, to meet nutritional needs, boost the immune system and prevent disease, boost energy, alertness, and habit from childhood*" [1, 2, 117]. While both male and female athletes report taking multivitamins with minerals, female athletes are more likely to take iron and calcium supplements than their male counterparts [117].

However, not all multivitamins are just that—a multivitamin. Several multivitamins contain herbal extracts (phyto-extracts), soy proteins, BCAAs, ginseng, and Ginkgo biloba, to name a few. These are usually marketed as a multivitamin labeled for performance and are widely available on drugstore shelves or health marts.

It is important to remember that multivitamins fall under the dietary supplement category and are not subject to FDA regulation. Allied health professions who work with athletes and coaches should be aware of the brand their athletes are taking and be able to counsel them to make informed purchases and encourage them to read labels. As an alternative, there are several prescription vitamins which are subject to FDA regulations that could be prescribed by the team physician.

### 30.3.6 Iron

Women in general tend to have more iron deficiency issues than men [118, 119]. Menstrual blood loss varies by woman and can be underestimated in women with heavier cycles. Other factors include inadequate dietary intake, increased loss from sweat, or gastrointestinal blood loss in runners [118, 119].

Iron status should be determined by plasma ferritin. Normal levels vary, but <35 μg/L are below normal [118]. Even slight anemia in athletes will negatively impact performance [119]. If athletes are borderline, low normal, or below normal, iron supplementation may be prudent. As iron comes in different forms, which are absorbed differently, and is unregulated on the store shelf, it could be more beneficial to have the team physician prescribe iron. A multivitamin with iron could be a good choice.

#### 30.3.6.1 Supplementing Iron with Normal Ferritin Levels

High dose of over-the-counter (OTC) iron supplements can cause gastrointestinal distress or constipation, both of which would prove to be ergolytic to athletic performance [119]. In addition, studies of iron supplementation in non-anemic athletes have not shown improvement in performance [118].

### 30.3.7 Calcium

Calcium should be consumed in the diet as should other nutrients. Most female athletes choose to take a calcium supplement to strengthen bones, due to lactose intolerance or low dietary intake [1, 2]. Calcium supplementation may also be considered if the athlete is consuming high amount of protein. Increased dietary protein may produce a lower urinary pH and an increase in calcium excretion [120]. Increased calcium intake may offset the increased protein-induced urinary excretion of calcium [121].

Typically calcium/protein ratio of 20:1 is recommended for middle-aged women [122]. This ratio is most likely different for an athlete as dietary protein requirement is elevated to maintain a positive nitrogen balance.

## 30.4 Contemporary Understanding of the Issues

Female athletes are consuming supplements for a variety of reasons:

- Health
- Inadequate diet
- Body composition
- Improved performance
- Increased "energy"

When choosing protein supplements, the athlete needs to understand the properties of protein sources: whey, casein, and soy. Whey is absorbed at the fastest rate followed by soy and then casein. The quick absorption rate yields a rapid but transient increase in muscle protein synthesis while a slower rate tends to mediate protein catabolism. A protein supplement utilizing a mixture of sources would be preferred. It should be noted that both whey and casein are animal-derived protein sources. Should you work with a vegetarian athlete, they should be made aware of the importance of complete proteins and EAAs.

## 30.5 Future Directions

Studies examining protein supplementation have been primarily conducted on male participants. Future research should focus on the female

population. Sex hormones affect protein metabolism. Without studies focusing on the effects of muscle formation and performance in females, we can only suggest general guidelines for protein supplementation. This is also the case for EAA and BCAA supplementation.

Echinacea is a popular supplement for boosting the immune system. Studies conflict as to the efficacy of Echinacea to prevent or shorten a cold. Furthermore, Echinacea supplementation has been linked to improved $VO_{2max}$ through increased EPO. This increase was not accompanied by an increase in RBC. Typically, treatments increasing EPO increase RBC. This can lead to cardiovascular complications. Further research is needed to determine the safety of using Echinacea to increase $VO_{2max}$ and determine if Echinacea can effectively treat a cold.

## 30.6 Concluding Remarks

It is of the utmost importance that allied health professionals and coaches who work with female athletes be familiar with the ergogenic aids female athletes are likely to use and the products that may contain them. Athletes will be more inclined to discuss supplements and seek your advice if you address them with an open mind and explain the pros and cons. Many athletes list family and other athletes as major sources of information on supplements [2]. Herbold et al. [3] reported family was the first source athletes went to regarding supplements (53 %). Other sources of information included friends (24.6 %), physician (18.7 %), coaches (10.5 %), and nutritionist (8.2 %) [3].

When surveyed, athletes indicated they do not use a certain supplement and usually list a product containing it under the "other" category [2]. This demonstrates that athletes lack knowledge of supplements, regulations, and how to read labels.

Finally, a supplement may be legal for OTC sales but illegal in certain competitions, or a supplement may be contaminated with substances not listed on the label. It is also important that the athletes know they are responsible for what they put in their bodies whether or not it is on the label and will result in a failed drug test.

On average, 15 % of nonhormonal nutritional supplements are contaminated with anabolic steroids [123]. Contamination has been found in products from many different countries. This may be due to lack of cleaning on the production line between manufacturing prohormones and nutritional supplements (i.e., vitamins, minerals, proteins, creatine). It may also be due to cross-contamination from shipping containers of raw materials. Some supplements available are faked supplements (steroids not listed on the label or given fake names) intentionally produced with high amounts of anabolic steroids normally available by prescription. The same manufacturers of faked supplements may also manufacture other nutritional supplements again leading to contamination. Anabolic steroids found in contaminated substances have been found to contain metandienone, stanozolol, boldenone, oxandrolone, dehydrochloromethyl-testosterone, and clenbuterol. Even some supplements marketed for weight loss have been found to contain clenbuterol. In as little as 3 h after ingestion of a clenbuterol-contaminated weight-loss product, clenbuterol was detectable in the urine (2 ng/ml) [123].

The allied health team should be aware of the regulations regarding banned substances for the governing body of athletes such as International Olympic Committee (IOC), WADA, and/or the NCAA. The team may also want to access http://www.naturaldatabase.com. The Natural Medicines Comprehensive Database is a good resource for evidence-based information on nutritional supplements that is not biased from advertising sponsorship.

## References

1. Kristiansen M, Levy-Milne R, Barr S, Flint A. Dietary supplement use by varsity athletes at a Canadian University. Int J Sport Nutr Exerc Metab. 2005;15(2):195–210.
2. Froiland K, Koszewski W, Hingst J, Kopecky L. Nutritional supplement use among college athletes and their sources of information. Int J Sport Nutr Exerc Metab. 2004;14(1):104–20.

3. Herbold NH, Visconti BK, Frates S, Bandini L. Traditional and nontraditional supplement use by collegiate female varsity athletes. Int J Sport Nutr Exerc Metab. 2004;14(5):586–93.
4. Gruber AJ, Pope HG. Ephedrine abuse among 36 female weightlifters. Am J Addictions. 1998;7(4):256–61.
5. Ziegler PJ, Nelson JA, Jonnalagadda SS. Use of dietary supplements by elite figure skaters. Int J Sport Nutr Exerc Metab. 2003;13(3):266–76.
6. Whitehead MT, Martin TD, Scheett TP, Webster MJ. Running economy and maximal oxygen consumption after 4 weeks of oral echinacea supplementation. J Strength Cond Res. 2012;26(7):1928.
7. Whitehead MT, Martin TD, Scheet TP, Webster MJ. The effects of 4 wk of oral echinacea supplementation on serum erythropoietin and indices of erythropoietic status. Int J Sport Nutr Exerc Metab. 2007;17(4):378–90.
8. Congeni J, Miller S. Supplements and drugs used to enhance athletic performance. Pediatr Clin North Am. 2002;49(2):435–61.
9. Faigenbaum AD, Zaichkowsky LD, Gardner DE, Micheli LJ. Anabolic steroid use by male and female middle school students. Pediatrics. 1998;101(5):E6.
10. NCAA publications—research—substance use—national study of substance use trends among NCAA college student-athletes. Cited 7/31/2012. Available from: http://www.ncaapublications.com/p-4266-research-substance-use-national-study-of-substance-use-trends-among-ncaa-college-student-athletes.aspx
11. Yusko DA, Buckman JF, White HR, Pandina RJ. Alcohol, tobacco, illicit drugs, and performance enhancers: a comparison of use by college student athletes and nonathletes. J Am Coll Health. 2008;57(3):281–90.
12. ACSMps. Nutrition and athletes performance. *Med Sci Sports Exerc* 2009;41(3):709–31.
13. Campbell B, Kreider RB. Conjugated linoleic acids. Curr Sports Med Rep. 2008;7(4):237–41.
14. Layman DK, Shiue H, Sather C, Erickson DJ, Baum J. Increased dietary protein modifies glucose and insulin homeostasis in adult women during weight loss. J Nutr. 2003;133:405–10.
15. Volek JS, Forsythe C. Very-low carbohydrate diets, chapter 25. In: Antonio J, Kalman D, Stout JR, Greenwood M, Willoughby DG, Haff GG, editors. Essentials of sports nutrition and supplements. Totowa, NJ: Humana Press; 2008. p. 581–603.
16. Clifton PM, Keogh JB, Noakes M. Long-term effects of a high-protein weight-loss diet. Am J Clin Nutr. 2008;87(1):23.
17. Noakes M. The role of protein in weight management. Asia Pac J Clin Nutr. 2008;17:169–71.
18. Bilsborough S, Mann N. A review of issue of dietary protein intake in humans. Int J Sports Nutr Exc Metab. 2006;16:129–52.
19. BØrsheim E, Tipton K, Wolfe SE, Wolfe RR. Essential amino acids and muscle protein recovery from resistance exercise. Am J Physiol Endocrinol Metab. 2002;283:E648–57.
20. Paddon-jones D, Sheffield-Moore M, Zhang XJ, Volpi E, Wolf SE, Aarslan A, et al. Amino acid ingestión improves muscles protein síntesis. Am J Physiol Endocrinol Metab. 2004;286:E321–8.
21. Tipton KD, Wolf R. Exercise, protein metabolism and muscle growth. Int J Sports Nutr Exc Metab. 2001;11(1):109–32.
22. Kreider RB, Wilborn CD, Taylor L, Campbell B, Almada AL, Collins R, et al. ISSN exercise & sport nutrition review: research & recommendations. J Int Soc Sports Nutr. 2010;7:7.
23. Bianco A, Mammina C, Paoli A, Bellafiore M, Battaglia G, Caramazza G, et al. Protein supplementation in strength and conditioning adepts: knowledge, dietary behavior and practice in palermo, Italy. J Int Soc Sports Nutr. 2011;8:25.
24. Petroczi A, Naughton DP, Mazanov J, Holloway A, Bingham J. Performance enhancement with supplements: incongruence between rationale and practice. J Int Soc Sports Nutr. 2007;4(19):19.
25. Imdad A, Bhutta ZA. Effect of balanced protein energy supplementation during pregnancy on birth outcomes. BMC Publ Health. 2011;11 Suppl 3:S17.
26. Etzel MR. Manufacture and use of dairy protein fractions. J Nutr. 2004;134:996S–1002.
27. Hoffman JR, Falvo MJ. Protein- which is the best? J Sports Sci Med. 2004;13:118–30.
28. Snyder BS, Haub MD. Whey, casein, and soy proteins. In: Driskell JA, editor. Sports nutrition fast and proteins. Boca Raton, FL: CRC Press; 2007. p. 143–63.
29. Marshall K. Therapeutic applications of whey protein. Altern Med Rev. 2004;9(2):136–56.
30. Miller AL. Therapeutic considerations of L-glutamine: a review of the literature. Altern Med Rev. 1999;4(4):239.
31. McConell GK, Kingwell BA. Does nitric oxide regulate skeletal muscle glucose uptake during exercise? Exerc Sport Sci Rev. 2006;34(1):36–41.
32. Stipanuk M, Stipanuk H. Role of the liver in regulation of body cysteine and taurine levels: a brief review. Neurochem Res. 2004;29(1):105–10.
33. Mojtahedi MC, Thorpe MP, Karampinos DC, Johnson CL, Layman DK, Georgiadis JG, et al. The effects of a higher protein intake during energy restriction on changes in body composition and physical function in older women. J Gerontol Ser A Biol Sci Med Sci. 2011;66(11):1218.
34. Holm L, Olesen JL, Matsumoto K, Doi T, Mizuno M, Alsted TJ, et al. Protein-containing nutrient supplementation following strength training enhances the effect on muscle mass, strength, and bone formation in postmenopausal women. J Appl Physiol. 2008;105(1):274–81.
35. Koopman R. Dietary protein and exercise training in ageing. Proc Nutr Soc. 2011;70(1):104–13.
36. Tipton K, Elliott TA, Cree MG, Wolf SE, Sanford AP, Wolfe RR. Ingestion of casein and whey proteins

result in muscle anabolism after resistance exercise. Med Sci Sports Exc. 2004;36(12):2073–81.
37. Paul GL. The rationale for consuming protein blends in sports nutrition. J Am Coll Nutr. 2009;28 (Suppl):464S.
38. Boirie Y, Dangin M, Gachon P, Vasson M, Maubois J, Beaufrère B. Slow and fast dietary proteins differently modulate postprandial protein accretion. Proc Natl Acad Sci U S A. 1997;94(26):14930–5.
39. West DWD, Burd NA, Coffey VG, Baker SK, Burke LM, Hawley JA, et al. Rapid aminoacidemia enhances myofibrillar protein synthesis and anabolic intramuscular signaling responses after resistance exercise. Am J Clin Nutr. 2011;94(3):795.
40. Hasler CM. The cardiovascular effects of soy products. J Cardiovasc Nurs. 2002;16(4):50.
41. Zhan S, Ho SC. Meta-analysis of the effects of soy protein containing isoflavones on the lipid profile. Am J Clin Nutr. 2005;81(2):397.
42. Laguer A. Supplements soy. [Internet]. 2003 [cited] (20/12/2006). Available from: http://www.ucdenver.edu/academics/colleges/pharmacy/Resources/OnCampusPharmDStudents/ExperientialProgram/Documents/nutr_monographs/Monograph-soy.pdf
43. Skidmore-Roth L. Mosby's handbook of herbs & natural supplements. 1st ed. St. Louis, MO: Elsevier; 2001.
44. Cook M, Cribb PJ. Effective nutritional supplement combinations chapter 9. In: Greenwood M, Kalman DS, Antonio J, editors. Nutritional supplements in sports and exercise. Totowa, NJ: Humana Press; 2008. p. 259–319.
45. Cribb PJ, Williams AD, Hayes A. A creatine-protein-carbohydrate supplementation enhances responses to resistance training. Med Sci Sports Exc. 2007;39(11):1960–8.
46. Dangin M, Guillet C, Garcia-Rodenas C, Gachon P, Bouteloup-Demange C, Reiffers-Magnani K, et al. The rate of protein digestion affects protein gain differently during aging in humans. J Physiol. 2003;549(Pt 2):635–44.
47. Dangin M, Boirie Y, Guillet C, Beaufrère B. Influence of the protein digestion rate on protein turnover in young and elderly subjects. J Nutr. 2002;132:3228S–33.
48. Pennings B, Boirie Y, Senden J, Gijsen AP, Kuipers H, van Loon L. Whey protein stimulates postprandial muscle protein accretion more effectively than do casein and casein hydrolysate in older men. Am J Clin Nutr. 2011;93(5):997–1005.
49. Tang JE, Moore DR, Kujbida GW, Tarnopolsky MA, Phillips SM. Ingestion of whey hydrolysate, casein, or soy protein isolate: effects on mixed muscle protein synthesis at rest and following resistance exercise in young men. J Appl Physiol. 2009;107(3): 987–92.
50. Moore RD, Robinson MJ, Fry JL, Tang JE, Glover EI, Wilkinson SB, et al. Ingested protein dose response of muscle and albumin protein synthesis after resistance exercise in young men. Am J Clin Nutr. 2009;89:161–8.
51. Tarnopolsky MA, Timmons BW. Protein: quantity and quality, chapter 7. In: Driskell JA, editor. Sports nutrition fats and proteins. Boca Raton, FL: CRC Press; 2007. p. 109–42.
52. Lamont LS, McCullough AJ, Kalhan SC. Gender differences in leucine, but not lysine, kinetics. J Appl Physiol. 2001;91(1):357.
53. Lamont LS, McCullough AJ, Kalhan SC. Gender differences in the regulation of amino acid metabolism. J Appl Physiol. 2003;95(3):1259–65.
54. Young VR, Tharakan JJ. Nutritional essentiality of amino acids and amino acid requirements in healthy adults in clinical nutrition. In: Cynober LC, editor. Metabolic and therapeutic aspects of amino acids in clinical nutrition. 2nd ed. Boca Raton, FL: CRC Press; 2004. p. 439–70.
55. Wilkinson SB, Tarnopolsky MA, Macdonald MJ, Macdonald JR, Armstrong D, Phillips SM. Consumption of fluid skim milk promotes greater muscle protein accretion after resistance exercise than does consumption of an isonitrogenous and isoenergetic soy- protein beverage. Am J Clin Nutr. 2007;85(4):1031.
56. Dillon EL, Sheffield-Moore M, Paddon-Jones D, Gilkison C, Sanford AP, Casperson SL, et al. Amino acid supplementation increases lean body mass, basal muscle protein synthesis, and insulin-like growth factor-I expression in older women. J Clin Endocrinol Metab. 2009;94(5):1630–7.
57. Wolfe RR. Regulation of muscle protein by amino acids. J Nutr. 2002;132:3219S–24.
58. Cribb PJ, Hayes A. Effects of supplement timing and resistance exercise on skeletal muscle hypertrophy. Med Sci Sports Exc. 2006;38(11):918–1925.
59. La Bounty PM, Campbell BI, Wilson J, Galvan E, Berardi J, Kleiner SM et al. International society of sports nutrition position stand: meal frequency. J. Int. Soc. Sports. Nutr. 2011;8(4):1–12.
60. Willoughby DS, Stout JR, Wilborn CD. Effects of resistance training and protein plus amino acid supplementation on muscle anabolic, mass, and strength. Amino Acids. 2007;32:467–77.
61. Breen L, Phillips SM. Skeletal muscle protein metabolism in the elderly: Interventions to counteract the "anabolic resistance" of ageing. Nutr Metab. 2011;8:68.
62. Yang Y, Breen L, Burd NA, Hector AJ, Churchward-Venne TA, Josse AR, et al. Resistance exercise enhances myofibrillar protein synthesis with graded intakes of whey protein in older men. Br J Nutr. 2012;108(10):1780–8.
63. Bohe J, Low A, Wolfe RR, Rennie MJ. Human muscle protein synthesis is modulated by extracellular, not intramuscular amino acid availability: a dose-response study. J Physiol. 2003;552(Pt 1):315–24.
64. Figueroa Alchapar J, Naclerio F. Ayudas ergogenicas nutricionales para la actividad fisica y el deporte. In:

Naclerio F, editor. Deportivo, fundamentos y aplicaciones en diferentes deportes. Medica Panamericana; 2011. p. 517-37
65. Kerksick C, Harvey T, Stout J, Campbell B, Wilborn C, Kreider R, et al. International society of sports nutrition position stand: nutrient timing. J Int Soc Sports Nutr. 2008;5:17.
66. Kerksick C, Leutholtz B. Nutrient administration and resistance training. J Int Soc Sports Nutr. 2005;2(1):50–67.
67. Rasmusen CJ. Nutritional supplement for endurance athletes chapter 11. In: Greenwood M, Kalman DS, Antonio J, editors. Nutritional supplements in sports and exercise. Totowa, NJ: Humana Press; 2008. p. 369–407.
68. Willoughby DS, Rosene JM. Effects of oral creatine and resistance training on myogenic regulatory factor expression. Med Sci Sports Exerc. 2003;35(6): 923–9.
69. Mourier A, Bigard AX, De Kerviler E, Roger B, Legrand H, Guezennec CY. Combined effects of caloric restriction and branched-chain amino acid supplementation on body composition and exercise performance in elite wrestlers. Int J Sports Med. 1997;18(1):47–55.
70. Campbell B, Kreider RB, Ziegenfuss T, La Bounty P, Roberts M, Burke D, et al. International society of sports nutrition position stand: Protein and exercise. J. Int. Soc. Sports. Nutr. 2007;doi:10.1186/1550-2783-4-8:4:8.
71. Guoyao W. Intestinal mucosal amino acid catabolism. J Nutr. 1998;128(8):1249–52.
72. Meeusen R, Watson P, Dvorak J. The brain and fatigue: new opportunities for nutritional intervention? J Sports Sci. 2006;24(7):773–82.
73. Newsholme EA, Leech AR. Biochemical for the medical sciences. New York: Wiley; 1994.
74. Newsholme EA, Blomstrand E. Branched-chain amino acids and central fatigue. J Nutr. 2006;136: 274S–6.
75. Shimomura Y, Inaguma A, Watanabe S, Yamamoto Y, Muramatsu Y, Bajotto G, et al. Branched-chain amino acid supplementation before squat exercise and delayed-onset muscle soreness. Int J Sport Nutr Exerc Metab. 2010;20(3):236–44.
76. Shimomura Y, Yamamoto Y, Bajotto G, Sato J, Murakami T, Shimomura N, et al. Nutraceutical effects of branched-chain amino acids on skeletal muscle. J Nutr. 2006;136(2):529S.
77. Antonio H, Sanders M, Kalman D, Woodgate D, Street C. The effects of high dose glutamine ingestion on weightlifting performance. J Strength Cond Res. 2002;16(1):157–60.
78. Walsh NP, Blannin AB, Bishop NC, Robson PJ, Robson S, Gleeson M. Effect of oral glutamine supplementation on human neutrophil lipopolysaccharide-stimulated degranulation following prolonged exercise. Int J Sports Nutr Exerc Metabol. 2000;10:39–50.
79. Gibala MJ. Regulation of skeletal muscle amino acid metabolism during exercise. Int J Sports Nutr Exc Metab. 2001;11(1):87–108.
80. Phillips GC. Glutamine: the nonessential amino acid for performance enhancement. Curr Sports Med Rep. 2007;6(4):265.
81. Cuisinier C, Ward RJ, Francaux M, Sturbois X, de Witte P. Changes in plasma and urinary taurine and amino acids in runners immediately and 24 h after a marathon. Amino Acids. 2001;20(1):13–23.
82. Lagranha CJ, Levada-Pires A, Sellitti DF, Procopio J, Curi R, Pithon-Curi T. The effect of glutamine supplementation and physical exercise on neutrophil function. Amino Acids. 2008;34(3):337–46.
83. Alvares TS, Meirelles CM, Bhambhani YN, Paschoalin V, Gomes P. L-arginine as a potential ergogenic aid in healthy subjects. Sports Med. 2011;41(3):233–48.
84. Fricke O, Baecker N, Heer M, Tutlewski B, Schoenau E. The effect of L-arginine administration on muscle force and power in postmenopausal women. Clin Physiol Funct Imaging. 2008;28(5):307–11.
85. Karlic H, Lohninger A. Supplementation of L-carnitine in athletes: does it make sense? Nutrition. 2004;20(7–8):709–15.
86. Calfee R, Fadale P. Popular ergogenic drugs and supplements in young athletes. Pediatrics. 2006; 117(3):e577–89.
87. Barr SI, Rideout CA. Nutritional considerations for vegetarian athletes. Nutrition. 2004;20(7–8): 696–703.
88. Tokish JM, Kocher MS, Hawkins RJ. Ergogenic aids: a review of basic science, performance, side effects, and status in sports. Am J Sports Med. 2004;32(6):1543–53.
89. Stevenson SW, Dudley GA. Creatine loading, resistance exercise performance, and muscle mechanics. J Strength Cond Res. 2001;15(4):413–9.
90. Vandenberghe K, Goris M, Van Hecke P, Van Leemputte M, Vangerven L, Hespel P. Long-term creatine intake is beneficial to muscle performance during resistance training. J Appl Physiol. 1997; 83(6):2055–63.
91. Bucci LR. Selected herbals and human exercise performance. Am J Clin Nutr. 2000;72 Suppl 2:624S–36.
92. Winterstein AP, Storrs CM. Herbal supplements: considerations for the athletic trainer. J Athl Train. 2001;36(4):425–32.
93. Bahrke MS, Morgan WP, Stegner A. Is ginseng an ergogenic aid? Int J Sport Nutr Exerc Metab. 2009;19(3):298–322.
94. Palisin TE, Stacy JJ. Ginseng: is it in the root? Curr Sports Med Rep. 2006;5(4):210.
95. Vogler BK, Pittler MH, Ernst E. The efficacy of ginseng. A systematic review of randomised clinical trials. E J Clin Pharmacol. 1999;55(8):567–75.
96. Engels H. Effects of ginseng supplementation on supramaximal exercise performance and short-term recovery. J Strength Cond Res. 2001;15(3):290–5.

97. Dowling EA, Redondo DR, Branch JD, Jones S, McNabb G, Williams MH. Effect of eleutherococcus senticosus on submaximal and maximal exercise performance. Med Sci Sports Exerc. 1996;28(4):482–9.
98. Barrett B, Brown R, Rakel D, Mundt M, Bone K, Barlow S, et al. Echinacea for treating the common cold: a randomized trial. Ann Intern Med. 2010;153(12):769.
99. Turner RB, Riker DK, Gangemi JD. Ineffectiveness of echinacea for prevention of experimental rhinovirus colds. Antimicrob Agents Chemother. 2000;44(6):1708.
100. Turner RB, Bauer R, Woelkart K, Hulsey TC, Gangemi JD. An evaluation of echinacea angustifolia in experimental rhinovirus infections. N Engl J Med. 2005;353(4):341.
101. Powers ME. Ephedra and its application to sport performance: another concern for the athletic trainer? J Ath Train. 2001;36(4):420–4.
102. Dietary supplements>guidance for industry: final rule declaring dietary supplements containing ephedrine alkaloids adulterated because they present an unreasonable risk; small entity compliance guide; Cited [7/31/2012]. Available from: http://www.fda.gov/Food/GuidanceComplianceRegulatoryInformation/GuidanceDocuments/DietarySupplements/ucm072997.htm
103. Shekelle PG, Hardy ML, Morton SC, Maglione M, Mojica WA, Suttorp MJ, et al. Efficacy and safety of ephedra and ephedrine for weight loss and athletic performance: a meta-analysis. JAMA. 2003;289(12):1537.
104. Juhn MS. Popular sports supplements and ergogenic aids. Sports Med. 2003;33(12):921–39.
105. Ahrendt DM. Ergogenic aids: counseling the athlete. Am Fam Physician. 2001;63(5):913–22.
106. Graham TE. Caffeine and exercise: metabolism, endurance and performance. Sports Med. 2001;31(11):785–807.
107. Mangus BC, Trowbridge CA. Will caffeine work as an ergogenic aid? the latest research. Ath Therapy Today. 2005;10(3):57–62.
108. Doherty M, Smith P, Hughes M, Davison R. Caffeine lowers perceptual response and increases power output during high-intensity cycling. J Sports Sci. 2004;22(7):637.
109. Greer F, McLean C, Graham TE. Caffeine, performance, and metabolism during repeated wingate exercise tests. J Appl Physiol. 1998;85(4):1502.
110. Astorino TA, Matera AJ, Basinger J, Evans M, Schurman T, Marquez R. Effects of red bull energy drink on repeated sprint performance in women athletes. Amino Acids. 2012;42(5):1803.
111. Candow DG, Kleisinger AS, Grenier S, Dorsch KD. Effect of sugar-free red bull energy drink on high-intensity run time-to-exhaustion in young adults. J Strength Cond Res. 2009;23(4):1271.
112. Ivy JL, Kammer L, Ding Z, Wang B, Bernard JR, Liao Y, et al. Improved cycling time-trial performance after ingestion of a caffeine energy drink. Int J Sport Nutr Exerc Metab. 2009;19(1):61.
113. Kazemi F, Gaeini A, Kordi A, Rahnama M, Rahnama R, Rahnama N. The acute effects of two energy drinks on endurance performance in female athlete students. Sport Sci Health. 2009;5(2):55–60.
114. Evans NA. Current concepts in anabolic-androgenic steroids. Am J Sports Med. 2004;32(2):534.
115. Pluim BM. Current perspective. the athlete's heart: a meta-analysis of cardiac structure and function. Circulation. 1999;101(3):336–45.
116. Whyte GP, George KJ, Nevill AJ, Shave RJ, Sharma SJ, Mckenna WJ. Left ventricular morphology and function in female athletes: a meta-analysis. Int J Sports Med. 2004;25(5):380–3.
117. Krumbach CJ, Ellis DR, Driskell JA. A report of vitamin and mineral supplement use among university athletes in a division I institution. Int J Sport Nutr. 1999;9(4):416.
118. Nielsen P, Nachtigall D. Iron supplementation in athletes current recommendations. Sports Med. 1998;26(4):207.
119. Beard J, Tobin B. Iron status and exercise. Am J Clin Nutr. 2001;72(2):594S.
120. Barzel US, Massey LK. Excess dietary protein can adversely affect bone. J Nutr. 1998;128(6):1051–3.
121. Dawson-Hughes B. Interaction of dietary calcium and protein in bone health in humans. J Nutr. 2003;133(3):852S.
122. Heaney RP. Excess dietary protein may not adversely affect bone. J Nutr. 2008;128(6):1054.
123. Geyer H, Parr MK, Koehler K, Mareck U, Schanzer W, Thevis M. Nutritional supplements cross-contaminated and faked with doping substances. J Mass Spec. 2008;43:892–902.
124. Reeds PJ. Dispensable and indispensable amino acids for humans. J Nutr. 2000;130(7):1835S.

# Nutritional Guidelines and Energy Needs During Pregnancy and Lactation

31

Jacalyn J. Robert-McComb, Ángela García González, and Lesley Carraway

**Abstract**

From a nutritional point of view, pregnancy and lactation are the most demanding physiological situations in a woman's life. Requirements for all nutrients increase and optimal energy, and nutrient intake during pregnancy and lactation are basic for the actual and future health of both mother and child. Because successful pregnancy depends upon previous nutritional status of the mother, all women of childbearing age should be encouraged to consume a variety of nutrient-dense foods and beverages within and among the basic food groups while choosing foods that limit the intake of saturated and trans fats, cholesterol, added sugars, salt, and alcohol. Special attention must be paid to intake of micronutrients such as folic acid, vitamin D, iron, and iodine. A deficit in folic acid intake during the first 8 weeks of conception may lead to malformations (neural tube defects) which can be prevented by daily intake of 600 μg of folates from diet, supplements, or nutrient-enriched foods. Vitamin D deficiency among pregnant women can result from inadequate cutaneous synthesis, limited dietary intake of vitamin D, or vitamin D pathway impairment, and can lead to osteoporosis in the mother and/or rickets, hypocalcemia, delayed ossification, and abnormal enamel formation in the children as well as immune dysfunction. The DRI for vitamin D, during pregnancy and lactation, is 600 IU (15 μg)/day. There are not many natural foods rich in vita-

J.J. Robert-McComb, Ph.D, FACSM, ACSM P.D. (✉)
L. Carraway, M.S., ACSM H.F.S.
Department of Health, Exercise, and Sports Sciences, Texas Tech University, Lubbock, TX, USA
e-mail: jacalyn.mccomb@ttu.edu

Á.G. González, M.D., Ph.D.
Pharmaceutical and Health Sciences, University San Pablo-CEU, Urb. Montepríncipe; Ctra. Boadilla del Monte km 5.3, Alcorcón, Madrid 28668, Spain
e-mail: angargon@fusp.ceu.es

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_31, 

min D so, apart from cold water fish, fortified foods are the main sources of this vitamin. Iron and iodine are also problematical nutrients which deficiencies are prevalent during gestation and lactation and so the need for their supplementation must be carefully evaluated. About energy, during pregnancy, women should consume an additional 300 kcal per fetus; however, women who are active during their pregnancy may need extra calories for exercise, and ideally, this additional energy should come from added servings of carbohydrate because carbohydrate intake meets the growth needs of the fetus and provides energy for exercise.

**Keywords**
Nutritional requirements during pregnancy • Folic acid • Vitamin D • Iron and iodine intakes during pregnancy • Energy requirements during pregnancy

## 31.1 Learning Objectives

After completing this chapter, you should have an understanding of the following:

- Nutritional guidelines during pregnancy and lactation
- Energy requirements during pregnancy and lactation
- Important nutrients, vitamins, and minerals for optimal pregnancy and infancy outcomes
- The importance of iron reserves prior to conception
- Folic acid supplementation
- Vitamin D deficiency in women with dark complexions and limited sun exposure

## 31.2 Introduction

Optimal nutrient intake during pregnancy and lactation is basic for the actual and future health of both mother and child. The optimum recommended levels of folic acid, iron, essential fatty acids, and other vitamins for pregnant women has been a topic of discussion in the research literature. Controversial topics for optimal nutrition during pregnancy will be reviewed in this chapter as well as the general consensus for overall recommendations [1–4].

General dietary guidelines for pregnant women are similar to guidelines recommended for nonpregnant women for optimum health. However, the recommended levels of essential nutrients, vitamins, and minerals that pregnant women should consume are higher than that for the nonpregnant woman.

The overall quality of a woman's diet affects her need for supplementation. If all of the necessary nutrients can be consumed in the daily diet, then supplements are not needed [5]. Yet, studies consistently show that many women, even in industrialized countries, may have vitamin and mineral deficiencies that could have an adverse effect on infant development [2, 4–6].

## 31.3 Research Findings

### 31.3.1 Nutritional Guidelines During Pregnancy and Lactation

Dietary reference intakes (DRI) for pregnant and lactating women can be found at http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes/dri-tables. There are essential nutrients, minerals, and vitamins that pregnant and lactating women must obtain through either a well-balanced nutrient-dense diet or a diet supplemented with vitamins, minerals, and essential nutrients and fortified foods.

General dietary guidelines for healthy pregnant women include the following recommendations: protein should comprise 20 % of a normal pregnancy diet. Fat should comprise approximately 30 % of diet in pregnancy and carbohydrates the remaining 50 %. A sample diet in pregnancy based

on the Position of the American Dietetic Association should include nine servings of cereals, three servings of fruits, four servings of vegetables, two to three servings of dairy products, and four servings of meats, beans, or nuts [7].

Total caloric intake will vary according to body mass index (BMI), age, and semester, but the average recommendation is 2,500 kcal/day [8]. It is suggested that the additional calories needed for pregnancy (300 kcal and 10 g of protein per fetus) come from adding one protein and one dairy serving [9].

### 31.3.2 Important Nutrients, Vitamins, and Minerals for Optimal Pregnancy/Infant Outcomes

#### 31.3.2.1 Iron

Adequate prepregnancy iron reserves can be achieved by an (a) adequate diet, (b) food fortification, or (c) preventive iron supplementation (low daily doses or weekly doses). Ideally, the last two types of intervention should include folate and, if needed, other nutrients such as vitamin A and zinc [2].

However, the best way to obtain nutrients is through diet. The amount of iron absorption depends upon the amount of iron in the diet, its bioavailability, and physiological requirements. Approximately 15 % of dietary iron is absorbed. The main sources of dietary heme iron are hemoglobin and myoglobin from red meats, fish, and poultry. Heme iron is absorbed two- to threefold more readily than nonheme iron. Meat also contains organic compounds that promote the absorption of iron from other less bioavailable nonheme iron sources. Approximately 95 % of the dietary iron intake is from nonheme iron sources. Vitamins significantly enhance iron absorption from nonheme foods, the size of this effect increases with the quantity of vitamin C in the meal [10].

Physiological iron requirements are three times higher in pregnancy than they are in the menstruating woman. A pregnant woman must consume an additional 700–800 mg of iron throughout her pregnancy: 3,100 mg for hematopoiesis and 3,000 mg for fetal and placental tissues [11].

Ideally, to meet iron needs during gestation, women should have 300 mg or more of iron reserves prior to conception [12]. Studies have shown that the best outcome conditions (birth weight, delivery time, and maternal health) have been reported to occur when hemoglobin level at term is between 931 and 1,231 g/L [13, 14]. Correction of anemia during pregnancy is difficult and should be prevented if possible.

Maternal anemia is defined by a hematocrit of less than 32 % and a hemoglobin level of less than 11 g/dL [11]. Among pregnant women, the prevalence of anemia in Africa exceeds 310 %, while it exceeds 40 % in Asia and exceeds 30 % in Latin America and Oceania. Anemia is generally less frequent among pregnant women in Europe (18.7 %) and in the USA (6 %). The exception in the USA being low-income and minority populations, in this group prevalence can reach 7–8 % or even 33 % in the third trimester of gestation [15–17].

#### 31.3.2.2 Folic Acid

It is well established that periconceptional supplementation with folic acid can reduce the incidence of neural tube defects (NTD) by 50 %. Data are available to advise all women who are capable of becoming pregnant to have periconception (i.e., *at least* 1 month before and until 3 months after conception) folic acid or multivitamin (including 0.4–0.8 mg of folic acid) supplementation to reduce the occurrence of NTDs and other major congenital abnormalities [18]. Additionally, the use of multivitamins containing folic acid and other B vitamin [19–21] showed a higher efficacy (90 %) in the reduction of NTDs than using a high dose of folic acid alone (70 %) [22] or a low dose of folic acid (41–79 %) [23].

Periconceptional is defined as 3 months prior to conception. This finding is the basis for the Center for Disease Control and Prevention's (CDC) recommendation that women of childbearing age who have a chance of becoming pregnant consume 0.4 mg of folate per day from 1 month preconception until the end of the first trimester.

Folic acid supplementation should continue 3 months in the postconception period also.

Breastfeeding women have higher folate requirements due to the folate supplied through breast milk. The folate concentration in human milk is tightly regulated and not affected by maternal folate status, except in clinically folate-deficient mothers. A higher folate intake will maintain a normal folate status of the mother. An infant consumes about 0.8 L/day of breast milk with an average concentration of 831 mcg/L, so mother losses through the milk about 66 mcg/L folate which implies an additional requirement of 133 mcg/day of folates [24].

Interestingly, epidemiological evidence suggests that the development of NTDs is not primarily because of the lack of sufficient folate in the diet but arises from genetically determined changes in the uptake, in metabolism, or in maternal and, particularly fetal cells [25]. Therefore, the gene–environmental interaction between vitamin dependency and nutrition may have a causal role in NTDs [18]. A sensitive and vulnerable period for fetuses is from the third to the eighth week after conception. Supplementation with folic acid-containing multivitamins or folic acid alone may cause an increase in folate metabolite concentrations of tissue fluids and it may overcome the failure of the local metabolite supply.

#### 31.3.2.3 Vitamin D

The National Research Council's recommended dietary allowance for vitamin D, during pregnancy and lactation, is 10 mcg/day [8]. There are not many natural foods rich in vitamin D, so apart from cold water fish (salmon, mackerel, sardines, etc.), fortified foods are the main sources of this vitamin. More clinical trials are needed to identify effective preventive strategies during pregnancy to achieve vitamin D adequacy [6, 26].

Vitamin D deficiency among pregnant women is not restricted to poor countries but also occurs in highly industrialized countries, more specifically to the disadvantaged subpopulations with limited sun exposure [6, 27]. Vitamin D deficiencies can result from inadequate cutaneous synthesis, limited dietary intake of vitamin D, or vitamin D pathway impairment [28].

Obesity is also a risk factor to develop vitamin D deficiency. Vitamin D is absorbed with fat as part of chylomicrons and is taken up first by peripheral tissues that express lipoprotein lipase, especially adipose tissue and skeletal muscle. This pathway predicts that increased adiposity should lead to lower serum 231OHD levels [29].

Throughout gestation, if a woman is vitamin D deficient, it appears to impact fetal bone health more than maternal [30–32]. Such deficiency in pregnant women can lead to rickets, hypocalcemia, delayed ossification, and abnormal enamel formation in children and osteoporosis, osteomalacia, and bone fractures in adults [6, 27, 33, 34].

But Vitamin D is not only the "bones vitamin," but it has also an important role in immune function. This function is developed in two ways: upregulation of the innate immune system and downregulation of the adaptive immune system [35–37]. The innate immune system, also known as nonspecific immune system, provides immediate defense against infection. The cells of the innate system recognize and respond to pathogens in a generic way, but it does not confer long-lasting or protective immunity to the host. The adaptive immune system, also known as the specific immune system, is composed of highly specialized, systemic cells and processes that eliminate or prevent pathogenic growth. It is an adaptive immunity because the body's immune system prepares itself for future challenges. Some studies suggest that the impact of vitamin D deficiency on immunity is stronger than the one on calcium metabolism and bone health as the risk of rickets increases significantly when total circulating 231(OH)D falls below 10 ng/mL (231 nmol/L), whereas cathelicidin mRNA expression, a marker of immune function, continues to be suppressed until 231(OH)D-circulating levels reach at least 20 ng/mL (310 nmol/L) [38] suggesting limit should be higher.

The lower limit of normal for 231(OH)D is controversial with suggested values in the literature ranging from 131 to 140 nmol/L [6, 26, 34, 38]. In 2010, the Institute of Medicine raised the minimum 231(OH)D concentration from 10 ng/mL (231 nmol/L) to 20 ng/mL (310 nmol/L) [16, 29].

Vitamin D sufficiency can be attained by enough exposure to sunlight and/or diet. The amount of sunlight sufficient to achieve optimal vitamin D status varies depending on a host of factors such as the time of day, the time of year, the latitude, degree of skin pigmentation, type and extend of clothing, and body surface area exposed. The recommendation to have good vitamin D synthesis through the skin is 10 min exposure to sun light, face and bare arms, without sunscreen, three times a week.

Circulating serum 231OHD levels are currently the best available indicator of the net-incoming contributions from cutaneous synthesis and total intake (foods and supplements). Thus, the serum 231OHD level may function as a biomarker of exposure.

#### 31.3.2.4 Iodine

Nearly two thirds of the 600 million people in Western and Central Europe live in regions of mild to severe iodine deficiency [39]. The National Research Council's RDA for iodine during pregnancy is 160 mcg/day and 209 mcg/day for women during lactation (see http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes/dri-tables) [8]. The removal of iodate conditioners in store-bought breads, the recommendations to reduce salt and egg intake, the use of noniodized salt in manufactured foods, and the reduction of meals made at home have reduced the US population iodine intake. Even though the National Health and Nutrition Examination Survey (NHANES) shows that iodine intake is adequate in general population, certain groups such as pregnant women were found to have a median urinary iodine concentration of 1,231 mcg/L (which will correlate with an intake of 178 mcg of iodine and 58 % of this group showed iodine intakes under the National Health and Nutrition Examination Survey's recommendations (220 mcg/day) [40].

In 2007, the World Health Organization/The United Nations Children's Fund/The International Council for the Control of Iodine Deficiency Disorders (WHO/UNICEF/ICCIDD) increased the recommended nutrient intake for iodine, during pregnancy, from 200 to 250 mcg, but stated clearly that more data are needed to establish the level of iodine intake that ensures maternal and newborn euthyroidism. Euthyroidism is the physiological state characterized by normal serum levels of thyroid hormone [41].

Pregnancy causes an increase in thyroid hormone requirements that can only be met by a proportional increase in hormone production that directly depends upon the availability of dietary iodine. This fact, together with an increase in urine losses because of the higher glomerular filtration during pregnancy, explains why gestation is a goitrogenic situation. Goitrogens are substances that suppress the function of the thyroid gland by interfering with iodine uptake, which can, as a result, cause an enlargement of the thyroid, i.e., a goiter. When iodine intake is restricted, even moderately, physiological adaptations lead to excessive glandular stimulation, hypothyroxinemia, and goiter formation. These conditions may only partially regress after parturition or childbirth.

Iodine deficiency during pregnancy and lactation has important repercussions for both mother and fetus. Thyroid hormones are fundamental in neuron migration to brain cortex and for the central nervous system development [42–45]. The fetal brain is particularly vulnerable to maternal hypothyroidism, and even subclinical hypothyroidism during pregnancy can impair mental development of the newborn as it increases infant mortality and growth retardation [42–45].

For nearly all countries the primary strategy for sustainable elimination of iodine deficiency is universal salt iodization. However, many countries have started to give indiscriminate iodine supplementation with a daily dose of 150 mcg of iodine. Prudency about supplementation with iodine is recommended. Some recent studies are suggesting long-term undesirable consequences for the newborn after iodine supplementation during pregnancy [46].

### 31.3.3 Energy Requirements During Pregnancy and Lactation

The energy requirements of pregnancy are those needed for adequate maternal gain to ensure the

growth of the fetus, placenta, and associated maternal tissues, and to provide for the increased metabolic demands of pregnancy, in addition to the energy needed to maintain adequate maternal weight, body composition, and physical activity throughout the gestational period, as well as for sufficient energy stores to assist in proper lactation after delivery. Recommendations for energy intake of pregnant women should be population specific, because of differences in body size, lifestyle, and underlying nutritional status; special considerations must be taken into account for women who are underweight or overweight when entering into the pregnancy period.

Estimated energy recommendations (EER) from the National Research Council for pregnant and lactating women can be found in the dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (macronutrients) [8]. The EER is defined as the average dietary energy intake that is predicted to maintain energy balance in a healthy adult of a defined age, gender, weight, height, and level of physical activity, consistent with good health. This information can also be downloaded free of charge at http://www.nap.edu/openbook.php?isbn=0309085373. Appendices 1 and 2 contain these referenced equations for pregnant and lactating women.

The EER from the National Research Council [8] is based on the components of total energy expenditure (TEE) which include (a) the basal metabolic rate (BMR) or basal metabolism (including thermoregulation) over 24 h (the BEE); for convenience, resting metabolic rate (RMR) is sometimes used or energy expenditure under resting conditions (note: it is not the same as BMR) extrapolated to 24 h (REE); (b) the thermic effect of food (TEF) or diet-induced thermogenesis (DIT); and (c) energy expended for physical activity which is commonly described as the ratio of total to basal daily energy expenditure (TEE/BEE). This ratio is known as the physical activity level (PAL) or the physical activity index.

Basal metabolic rate describes the rate of energy expenditure that occurs in the postabsorptive state. This state is defined as the particular condition that prevails after an overnight fast, the subject having not consumed food for 12–14 h. The participant should be resting comfortably, supine, awake, and motionless in a thermo-neutral environment. Basal metabolic rate is commonly extrapolated to 24 h to be more meaningful, and it is then referred to as BEE (basal energy expenditure), expressed as kcal/24 h. This value is affected by body size as well as lean body mass.

Thermoregulation is held within a narrow zone for humans. Increases in energy occur when ambient temperatures are below the zone of thermo neutrality. The thermo-neutral zone is the environmental temperature at which oxygen consumption and metabolic rate are lowest.

Resting metabolic rate (RMR) or energy expenditure under resting conditions tends to be somewhat higher (10–20 %) than under basal metabolic conditions (BMR). This is due to increases in energy expenditure caused by recent food intake or by the delayed effect of recently completed physical activity. It is important to distinguish between BMR and RMR and between BEE and REE (resting energy expenditure extrapolated to 24 h).

The thermic effect of food was originally referred to as the specific dynamic action (SDA) of food. It is more commonly referred to as the thermic effect of food (TEF) in more recent literature or diet-induced thermogenesis (DIT).

The energy expended for physical activity varies greatly among individuals as well as from day to day. The level of physical activity is commonly described as the ratio of total to basal daily energy expenditure (TEE/BEE). Describing physical activity habits in terms of PAL (physical activity level or TEE/BEE) is a convenient comparison and is used in the EER from the National Research Council to describe and account for physical activity habits. The estimated energy for physical activity (EEPA) is the most variable component of TEE.

Physical activity level or PAL in a nonpregnant state must be computed for the equations using age, weight, height, and gender. The PAL values obtained in pregnant women, especially during the second part of pregnancy, are lower than values obtained in nonpregnant individuals. Pregnancy is often associated with a comparatively

**Table 31.1** Doubly labeled water pregnancy studies

| References | *n* | Gestation week | Pregravid weight (kg) | Gestational weight gain (kg) | Total energy expenditure (kcal/day) | Physical activity level | Activity energy expenditure (kcal/day) |
|---|---|---|---|---|---|---|---|
| [48] | 10 | 10 | – | – | 2,470 | 1.42 | 731 |
| [49] | 22 | 0 | 60.8 | 13.5 | 2,484 | 1.87 | 1,147 |
| | 22 | 16–18 | | | 2,293 | 1.65 | 860 |
| | 22 | 30 | | | 2,986 | 1.82 | 1,338 |
| | 19 | 36 | | | 2,914 | 1.66 | 1,171 |
| [50] | 12 | 0 | 61.7 | 11.91 | 2,274 | 1.58 | 835 |
| | | 6 | | | 2,322 | 1.54 | 818 |
| | | 12 | | | 2,426 | 1.64 | 939 |
| | | 18 | | | 2,456 | 1.65 | 964 |
| | | 24 | | | 2,621 | 1.66 | 1,042 |
| | | 30 | | | 2,675 | 1.62 | 1,026 |
| | | 36 | | | 2,688 | 1.50 | 885 |
| [51] | 10 | 0 | – | 11.6 | 2,205 | 1.68 | 892 |
| | | 8–10 | | | 2,047 | 1.57 | 743 |
| | | 24–26 | | | 2,410 | 1.56 | 867 |
| | | 34–36 | | | 2,728 | 1.61 | 1,038 |

*Source*: Adapted from the Institute of Medicine (US), Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for energy carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. Washington, DC: National Academy Press, 2002

large increase in BMR, whereas the effect of pregnancy on energy expenditure when performing many specific activities tends to be rather small [47].

Doubly labeled water studies have also been conducted using well-nourished pregnant and lactating women [48–53]. Results of the energy needs during pregnancy and lactation can be found in Tables 31.1 and 31.2, respectively. Because energy requirements in pregnancy are increased by approximately 17 % over the non-pregnant state, a woman of normal weight should consume an additional 300 kcal/day, and those calories should be concentrated in foods of high-nutrient density, a value based on the percent protein, vitamins, and minerals per 100 kcal [5, 54].

Multifetal pregnancies in the USA increased by 77 % from 1980 to 2003 largely because of assisted reproduction treatments [55]. Although the exact caloric requirements for multiple gestations have not been well described, it is generally recommended that an additional 300 kcal and 10 g of protein per fetus beyond singleton are standard [5, 56].

### 31.3.4 Additional Energy Requirements for the Exercising Woman During Pregnancy and Lactation

Women who are active during their pregnancy need extra calories for exercise. Recent studies have found that between 41 and 61 % of women engage in regular leisure physical activity during pregnancy [57]. At the same time, the percentage of women within the work force has increased, and more women engage in physically demanding lines of work (such as police officers, fire fighters, military personnel). Furthermore, there has been an increase in the interest of adult women to join fitness clubs and participate in exercise programs as part of a healthy lifestyle corresponding with a rise in the incidence of overweight and obese women worldwide.

This additional energy should come from added servings of carbohydrate because carbohydrate intake meets the growth needs of the fetus and provides energy for exercise [58]. Carbohydrates are the main fuel both for fetus and muscles so, while doing exercise, there can

**Table 31.2** Doubly labeled water lactation studies

| References | *n* | Activity | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Stage of lactation (months) | Total energy expenditure (kcal/day) | Total energy expenditure (kcal/kg/day) | Physical activity level | Energy expenditure (kcal/day) | Milk energy output (kcal/day) | Energy mobilization (kcal/day) | Energy requirement (kcal/day) |
| [48] | 10 | 1 | 2,109 | 35.8 | 1.50 | 703 | 536 | Gained fat | 2,646 |
| | | 2 | 2,171 | 36.9 | 1.55 | 774 | 532 | Mass | 2,702 |
| | | 3 | 2,138 | 36.5 | 1.59 | 793 | 530 | | 2,667 |
| [49] | 23 | 2 | 2,532 | 39.3 | 1.82 | 1,123 | 502 | 72 | 2,962 |
| | | 6 | 2,580 | 41.0 | 1.79 | 1,123 | | | |
| [53] | 9 | 3–6 | 2,413 | 37.2 | 1.75 | 1,037 | 538 | 287 | 2,663 |
| [51] | 10 | 1 | 2,146 | – | 1.62 | – | – | – | – |
| [52] | 24 | 3 | 2,391 | 38.1 | 1.79 | 1,061 | 483 | 155 | 2,719 |

*Source*: Adapted from the Institute of Medicine (US), Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for energy carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. Washington, DC: National Academy Press, 2002

be a competition for blood glucose that can lead to hypoglycemia periods which can be dangerous both for the mother and the offspring.

The RDA recommended by the IOM of 1,731 g/day of CH [18] may not be sufficient to cover the necessities of being an active pregnant woman. The ingestion of a carbohydrate-rich snack before any practice of sportive activity is advised [57]. The combination of energy demands for childbearing necessities, and the additional energy needed for an active lifestyle may require personal and customized dietary advice for active pregnant women. Micronutrient status is another potential problem for an active pregnancy. For example, athletes usually have lower iron tissue stores before pregnancy and so are more likely to have iron deficiencies. Vitamin D and calcium status can also be a problem [57, 59, 60].

### 31.3.5 Weight Gain During Pregnancy

Weight gain during pregnancy comprises the products of conception (fetus, placenta, amniotic fluid), the growth of various maternal tissues (uterus, breasts), and the increase in blood, extracellular fluid, and maternal fat stores. An inadequate weight gain is associated with intrauterine growth retardation, preterm birth, preeclampsia, eclampsia, and postpartum hemorrhage and later obesity of the offspring and mother [61–63].

The optimal amount of prenatal weight gain is modified by a woman's prepregnancy weight for height (body mass index [BMI]). Total weight gain varies widely among women. Well-nourished women should gain 10–14 kg during pregnancy, with an average of 12 kg, in order to increase the probability of delivering full-term infants with an average birth weight of 3.3 kg and to reduce the risk of fetal and maternal complications. The mean rate of weight gain is 1.6 kg in the first trimester and 0.44 kg/week in the second and third trimesters. For underweight women, the mean rate of weight gain is 2.3 kg in the first trimester and 0.49 kg/week in the second and third trimesters. For overweight women, the mean rate of weight gain is 0.9 kg in the first trimester and 0.30 kg/week in the second and third trimesters [64].

Following the National Academy of Sciences, Institute of Medicine (IOM), recommendations [64] total weight gain ranges are 11–15 kg (25–35 lb) for normal-weight women (19.5<BMI>25.9), 12.7–18 kg (28–40 lb) for underweight women (BMI<19.8), and 7–11 kg (15–25 lb) for an overweight woman (BMI>26). This translates to 0.4 kg/week for normal-weight women, 0.31 kg/week for underweight women, and 0.3 kg/week for overweight women. Women with a BMI>29.0 (obesity) should be advised to gain at a rate that does not exceed 11.4 kg (25 lb) throughout total pregnancy and have a minimum gain of 7 kg (15 lb) [7]. The impact of maternal weight gain in the second trimester is most important for fetal development and is protective of fetal growth even if the overall weight gain is poor [5].

In the USA excessive weight gain during gestation remains of predominant concern, as 60 % of obese women gain more than recommended and, also, approximately 40 % of normal-weight women gain more than recommended [65]. Pregnancy-related obesity can lead to a lifetime of unhealthy weight for the mother and her offspring and to health problems as gestational diabetes or hypertension. Therefore, many women want or need to remain active during pregnancy, but little is known about the impact of exercise during pregnancy on nutritional requirements, nutrient intakes, and maternal weight gain, and whether these impact birth outcomes in physically active, pregnant women [66, 67].

## 31.4 Contemporary Understanding of the Issues

The overall quality of a woman's diet affects her needs for supplementation. However, it has been found that iron supplementation during pregnancy increases maternal iron status and stores and improves pregnancy outcome, when the mother is anemic or from a population in which anemia prevalence is high [68, 69]. That is the main reason why the IOM recommends a daily supplement containing 16–20 mg of iron during pregnancy for healthy women with a mixed diet [70]. The supplement should also

include vitamin B12 and 400 mcg (0.4 mg) of folic acid per daily dose [71].

According to the Department of Health and Human Services and the Department of Agriculture [72], a healthy eating pattern focuses on nutrient-dense foods—vegetables, fruits, whole grains, fat-free or low-fat milk and milk products, lean meats and poultry, seafood, eggs, beans and peas, and nuts and seeds that are prepared without added solid fats, sugars, starches, and sodium. Combined into an eating pattern, these foods can provide the full range of essential nutrients and fiber, without excessive calories. The oils contained in seafood, nuts and seeds, and vegetable oils added to foods also contribute essential nutrients.

The nutritional value of seafood is of particular importance during fetal growth and development, as well as in early infancy and childhood. Moderate evidence indicates that intake of omega-3 fatty acids, in particular DHA, from at least 8 oz of seafood per week for women who are pregnant or breastfeeding is associated with improved infant health outcomes, such as visual and cognitive development. Due to their methyl mercury content, limit white (albacore) tuna to 6 oz per week and do not eat the following four types of fish: tilefish, shark, swordfish, and king mackerel.

The Dietary Guidelines for Americans 2010 (http://www.cnpp.usda.gov/DGAs2010-PolicyDocument.htm) include the following key recommendations for all population groups [73]:

- Follow a healthy eating pattern while staying within calorie needs.
- Increase vegetable and fruit intake.
- Eat a variety of vegetables, especially dark-green and red and orange vegetables and beans and peas.
- Consume at least half of all grains as whole grains. Increase whole-grain intake by replacing refined grains with whole grains.
- Increase intake of fat-free or low-fat milk and milk products, such as milk, yogurt, cheese, or fortified soy beverages.
- Choose a variety of protein foods, which include seafood, lean meat and poultry, eggs, beans and peas, soy products, and unsalted nuts and seeds.
- Increase the amount and variety of seafood consumed by choosing seafood in place of some meat and poultry.
- Replace protein foods that are higher in solid fats with choices that are lower in solid fats and calories and/or are sources of oils.
- Use oils to replace solid fats where possible.
- Choose foods that provide more potassium, dietary fiber, calcium, and vitamin D, which are nutrients of concern in American diets. These foods include vegetables, fruits, whole grains, and milk and milk products.
- Women capable of becoming pregnant should choose foods that supply heme iron, which is more readily absorbed by the body. They should consume additional iron sources as well as enhancers of iron absorption such as vitamin C-rich foods.
- Women capable of becoming pregnant should consume 400 micrograms (mcg) per day of synthetic folic acid (from fortified foods and/or supplements) in addition to food forms of folate from a varied diet. The major dietary sources of folates are fresh and frozen green leafy vegetables, citrus fruits and juices, liver, wheat bread, and legumes.
- Women who are pregnant or breastfeeding should consume 8–12 oz of seafood per week from a variety of seafood types.
- If pregnant, take an iron supplement as recommended by an obstetrician or other health-care provider [74, 75].

## 31.5 Future Directions

It is thought that prepregnancy iron reserves can be achieved by an adequate diet, food fortification, or preventive iron supplementation (low daily doses or weekly doses). Ideally, the last two types of intervention should include folate and, if needed, other nutrients such as vitamin A and zinc [2].

Not all countries and all people have the necessary financial resources to provide nutrient-dense foods, healthy environments, and healthy lifestyles that contribute to or provide the essential vitamins and minerals needed for a robust

pregnancy. For example, results from a review of vitamin D deficiency in pregnant women indicated that 331 of the 76 studies reviewed reported that the mean or median maternal concentrations of 231(OH)D levels were 331 nmol/L or less that represents the lower limits of normal. Low concentrations were reported among different ethnic groups in many regions including North America, Europe, the UK, Africa, the Middle East, and Asia. In the USA the degree of deficiency is greatest in those with darker pigmentation, i.e., African-American women, but deficiency exists among Hispanic and Caucasian women who have limited access to sunlight, either through limited activity outdoors, type of clothing, cultural practices, or thorough use of sunscreen when outdoors [74, 75].

From a global public health perspective, food fortification with folic acid and some other B vitamins (B12 and B6) may be the most effective method to prevent NTDs in unplanned pregnancies [18]. In Hungary [76], three vitamins (folic acid [160 mcg], vitamins B12 [0.8 mcg], and vitamin B6 [0.864 mcg]) were added to 100 g of flour [77].

The concept of weekly iron–folic acid supplementation as a public health approach to prevent iron anemia was presented at the 1993 World Health Organization/United Nations University meeting [78]. It appears that small daily doses as recommended by the Food and Drug Administration [79] and the Food and Nutrition Board and IOM [81] as well as weekly dosing starting early in pregnancy are safer and essentially as efficacious as daily iron in preventing iron deficiency and improving iron nutrition when adherence is satisfactory [2] for females with iron deficiency anemia; therapy consists of 60–120 mg of ferrous iron in divided doses throughout the day [79]. The therapeutic dose depends on the hemoglobin level of the pregnant woman. For more complete guidelines, please refer to the guidelines for the assessment of iron deficiency in women of childbearing age and the IOM recommended guidelines for the prevention, detection, and management among US children and women of childbearing age for iron deficiency anemia [79, 80]. Even for nonpregnant women, long-term weekly supplementation with iron and folic acid can bring benefits in terms of the prevention of neural tube defects and hyperhomocysteinemia early in pregnancy [81].

However, there is not a universal consensus regarding the type of supplementation and the dose of folic acid. Even though the mandatory fortification of standardized enriched cereal grain products in the USA in 1998 resulted in a substantial increase in blood folate concentrations and a concomitant decrease in 36 % of NTD prevalence, mandatory fortifications have raised concerns about the consequences of excessive intakes.

Excessively high intakes of folic acid may cause harmful effects, including progression of nerve damage in B12-deficient persons, excess intake in children, accumulation of unmetabolized folic acid, blunting of antifolate therapy (methotrexate and phenytoin), accelerated cognitive decline in the elderly, epigenetic hypermethylation, and cancer promotion [82]. These findings are mainly attributed to an excess of folate intake and to the presence of no metabolized folic acid in plasma of people receiving very high doses of folic acid [24]. More data are needed to find the right equilibrium between fortification and supplementation while accomplishing the needs of the most nutrition vulnerable populations.

## 31.6 Concluding Remarks

The overall quality of a woman's diet affects her needs for supplementation and nutritional counseling advice. Excess or deficiency of any nutrient, vitamin, or mineral is a concern during pregnancy. Optimal pregnancy outcomes occur when women maintain a balanced diet prior to conception. Therefore, all women of childbearing age should be encouraged to consume a variety of nutrient-dense foods and beverages within and among the basic food groups while choosing foods that limit the intake of saturated and trans fats, cholesterol, added sugars, salt, and alcohol. Specifically, women of childbearing age who may become pregnant should eat foods high in heme iron and/or consume iron-rich

plant foods or iron-fortified foods with an enhancer of iron absorption, such as vitamin C-rich foods. Women of childbearing age who may become pregnant and those in the first trimester of pregnancy should consume adequate synthetic folic acid daily (from fortified foods or supplements) in addition to food forms of folate from a varied diet.

During pregnancy, women should consume an additional 300 kcal and 10 g of protein per fetus. It is suggested that the additional calories needed for pregnancy come from adding one protein and one dairy serving [9]. Women who are active during their pregnancy may need extra calories for exercise above the additional protein and dairy servings. Ideally, this additional energy should come from added servings of carbohydrate because carbohydrate intake meets the growth needs of the fetus and provides energy for exercise [58].

Lastly, it is recommended that women who are most vulnerable to congenital malformations be counseled preconceptionally. There is a fourfold increased risk of congenital malformations related to poor control of diabetes during embryogenesis. Also, girls under the age of 17 are at increased risk for preterm delivery, perinatal mortality, and low body weight. Specifically, girls within 2 years of menarche may require additional energy, protein, and calcium, to meet their nutritional needs for maternal and fetal growth. The erratic use of vitamin supplementation, poor nutrition, and body image issues for this specific population suggests that additional counseling may be warranted [5].

Women of childbearing age who may become pregnant and those in the first trimester of pregnancy should consume adequate synthetic folic acid daily (from fortified foods or supplements) in addition to food forms of folate from a varied diet.

## Appendix 1 Physical Activity Level Categories and Walking Equivalence

| | | | Walking equivalence (mi/day at 3–4 mph) | | |
|---|---|---|---|---|---|
| PAL category | PAL range | PAL | Lightweight individual (44 kg) | Middleweight individual (70 kg) | Heavyweight individual (120 kg) |
| Sedentary | 1.0–1.39 | 1.25 | ~0 | ~0 | ~ 0 |
| Low active | 1.4–1.59 | | | | |
| Mean | | 1.5 | 2.9 | 2.2 | 1.5 |
| Active | 1.6–1.89 | | | | |
| Minimum | | 1.6 | 5.8 | 4.4 | 3.0 |
| Mean | | 1.75 | 9.9 | 7.3 | 5.3 |
| Very active | 1.9–2.49 | | | | |
| Minimum | | 1.9 | 14.0 | 10.3 | 17.5 |
| Mean | | 2.2 | 22.5 | 16.7 | 12.3 |
| Maximum | | 2.5 | 31.0 | 23.0 | 17.0 |

Adapted from the National Research Council. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (macronutrients). Washington, DC: The National Academies Press, 2005
In addition to energy spent for the generally unscheduled activities that are part of a normal daily life, the low, middle, and high miles/day values apply to relatively heavyweight (120 kg), midweight (70 kg), and lightweight (44 kg) individuals, respectively

## Appendix 2 Estimated Energy Expenditure* Prediction Equations at Four Physical Activity Levels

*EER for infants and young children 0–3 years*

TEE (kcal/day) = 89 (±3 [standard error]) × weight of the child (kg) − 100 (±56 [standard error])
EER = TEE + energy deposition
0–3 months (89 × weight of infant [kg] − 100) + 175 (kcal for energy deposition)
4–6 months (89 × weight of infant [kg] − 100) + 56 (kcal for energy deposition)
7–12 months (89 × weight of infant [kg] − 100) + 22 (kcal for energy deposition)
13–36 months (89 × weight of child [kg] − 100) + 20 (kcal for energy deposition)
Where PA = physical activity coefficient:
PA = 1.0 if PAL is estimated to be ≥ 1.0 < 1.4 (sedentary)
PA = 1.13 if PAL is estimated to be ≥ 1.4 < 1.6 (low active)
PA = 1.26 if PAL is estimated to be ≥ 1.6 < 1.9 (active)
PA = 1.42 if PAL is estimated to be ≥ 1.9 < 2.5 (very active)

*EER for girls 3–8 years*

TEE = see doubly labeled water data used to predict energy expenditure for standard error in the dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (macronutrients) at http://www.nap.edu/openbook.php?isbn=0309085373
EER = TEE + energy deposition
EER = 135.3 − (30.8 × Age [years]) + PA × (10 × weight [kg] + 934 × height [m]) + 20 (kcal for energy deposition)
Where PA = physical activity coefficient:
PA = 1.0 if PAL is estimated to be ≥ 1.0 < 1.4 (sedentary)
PA = 1.16 if PAL is estimated to be ≥ 1.4 < 1.6 (low active)
PA = 1.31 if PAL is estimated to be ≥ 1.6 < 1.9 (active)
PA = 1.56 if PAL is estimated to be ≥ 1.9 < 2.5 (very active)

*EER for girls 9–18 years*

TEE = see doubly labeled water data used to predict energy expenditure for standard error in the dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (macronutrients) at http://www.nap.edu/openbook.php?isbn=0309085373
EER = TEE + energy depositions
EER = 135.3 − (30.8 × age [years]) + PA × (19 × weight [kg] + 934c height [m]) + 25 (kcal for energy deposition)
Where PA = physical activity coefficient:
PA = 1.0 if PAL is estimated to be ≥ 1.0 < 1.4 (sedentary)
PA = 1.16 if PAL is estimated to be ≥ 1.4 < 1.6 (low active)
PA = 1.31 if PAL is estimated to be ≥ 1.6 < 1.9 (active)
PA = 1.56 if PAL is estimated to be ≥ 1.9 < 2.5 (very active)

*EER for women 19 years and older*

EER = 354 − (6.91 × age [years]) + PA × (9.36 × weight [kg] + 726 × height [m])
Where PA = physical activity coefficient:
PA = 1.0 if PAL is estimated to be ≥ 1.0 < 1.4 (sedentary)
PA = 1.12 if PAL is estimated to be ≥ 1.4 < 1.6 (low active)
PA = 1.27 if PAL is estimated to be ≥ 1.6 < 1.9 (active)
PA = 1.45 if PAL is estimated to be ≥ 1.9 < 2.5 (very active)

*EER for pregnant women (14–18 years)*

$EER_{pregnant}$ = adolescent $EER_{nonpregnant}$ + additional energy expended during pregnancy + energy deposition
First trimester = adolescent EER + 0 + 0
Second trimester = adolescent EER + 160 kcal (8 kcal/week × 20 week) + 180 kcal
Third trimester = adolescent EER + 272 kcal (8 kcal/week × 24 week) + 180 kcal

*EER for pregnant women (19–50 years)*

$EER_{pregnant}$ = adult $EER_{nonpregnant}$ + additional energy expanded during pregnancy + energy deposition
First trimester = adult EER + 0 + 0
Second trimester = adult EER + 160 kcal (8 kcal/week × 20 week) + 180 kcal
Third trimester = adult EER +272 kcal (8 kcal/week × 34 week) + 180 kcal

(continued)

(continued)

| |
|---|
| *EER for lactating women (14–18 years)* |
| $EER_{lactation}$ = adolescent $EER_{prepregnancy}$ + milk energy output − weight loss<br>First 6 months = adolescent EER + 500 − 170<br>Second 6 months = adolescent EER + 400 − 0 |
| *EER for lactating women (19–50 years)* |
| $EER_{lactation}$ = adult $EER_{prepregnancy}$ + milk energy output − weight loss<br>First 6 months = adult EER + 500 − 170<br>Second 6 months = adult EER + 400 − 0 |
| *Weight maintenance TEE in overweight girls 3–18 years or at risk of a high* |
| TEE = 389 − (41.2 × age [years]) + PA × (15.0 × weight [kg] + 701.6 × height [m])<br>Where PA = physical activity coefficient:<br>PA = 1.0 if PAL is estimated to be ≥ 1.0 < 1.4 (sedentary)<br>PA = 1.12 if PAL is estimated to be ≥ 1.4 < 1.6 (low active)<br>PA = 1.24 if PAL is estimated to be ≥ 1.6 < 1.9 (active)<br>PA = 1.45 if PAL is estimated to be ≥ 1.9 < 2.5 (very active) |
| *Overweight and obese women 19 years and older* |
| TEE = 448 − (7.95 × age [years]) + PA × (11.4 × weight [kg] + 619 × height [m])<br>Where PA is the physical activity coefficient:<br>PA = 1.0 if PAL is estimated to be ≥ 1.0 < 1.4 (sedentary)<br>PA = 1.16 if PAL is estimated to be ≥ 1.4 < 1.6 (low active)<br>PA = 1.27 if PAL is estimated to be ≥ 1.6 < 1.9 (active)<br>PA = 1.44 if PAL is estimated to be ≥ 1.9 < 2.5 (very active) |
| *Normal and overweight or obese women 19 years and older* |
| TEE = 387 − (7.31 × age [years]) + PA × (10.9 × weight [kg] + 600.7 × height [m])<br>Where PA is the physical activity coefficient:<br>PA = 1.0 if PAL is estimated to be ≥ 1.0 < 1.4 (sedentary)<br>PA = 1.14 if PAL is estimated to be ≥ 1.4 < 1.6 (low active)<br>PA = 1.27 if PAL is estimated to be ≥ 1.6 < 1.9 (active)<br>PA = 1.45 if PAL is estimated to be ≥ 1.9 < 2.5 (very active) |

Adapted from the National Research Council. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. Washington, DC: The National Academies Press, 200. See http://www.nap.edu/openbook.php?isbn=0309085373

*Estimated energy expenditure (EER) is the average dietary energy intake that is predicted to maintain energy balance in a healthy adult of a defined age, gender, weight, height, and level of physical activity consistent with good health. In children and pregnant and lactating women, the EER includes the needs associated with the deposition of tissues or the secretion of milk at rates consistent with good health

Physical activity level (PAL) is the physical activity level that is the ratio of the total energy expenditure to the basal energy expenditure

Total energy expenditure (TEE) is the sum of the resting energy expenditure, energy expended in physical activity, and the thermic effect of food

Body mass index (BMI) is determined by dividing the weight (in kilograms) by the square of the height (in meters)

## References


1. Georgieff MK, Innin S. Controversial nutrients that potentially affect preterm neurodevelopment: essential fatty acids and iron. Pediatr Res. 2005;57(5 Pt 2):99R–103.
2. Viteri F, Berger J. Importance of pre-pregnancy and pregnancy iron status: can long-term weekly preventive iron and folic acid supplementation achieve desirable and safe status? Nutr Rev. 2005;63(12 Pt 2):S65–76.
3. Cornel MC, de Smit DJ, de Jong-van den Berg LTW. Folic acid—the scientific debate as a public health policy. Reprod Toxicol. 2005;20(3):411–5.
4. Smitasiri S, Solon F. Implementing preventive iron-folic acid supplementation among women of reproductive age in some western pacific countries: possibilities and challenges. Nutr Rev. 2005;63(12 Pt 2):S81–6.
5. Bader TJ. OB/GYN secrets, updated. 3rd ed. Philadelphia, PA: Elsevier Mosby; 2005.
6. Schroth RJ, Lavelle C, Moffatt M. A review of vitamin D deficiency during pregnancy: who is affected? Int J Circumpolar Health. 2005;64(2):112–20.

7. Position of the American Dietetic Association. Nutrition and lifestyle for a healthy pregnancy outcome. J Am Diet Assoc. 2002;102(10):1479–90.
8. National Research Council. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (macronutrients). Washington, DC: The National Academies Press; 2005.
9. Worthington-Roberts B, Williams SR. Nutrition in pregnancy and lactation. 6th ed. Dubuque, IA: Brown and Benchmark; 1997.
10. Pavord S, Myers B, Robinson S, Allard S, Strong J. Oppenheimer C on behalf of the British Committee for Standards in Haematology. UK guidelines on the management of iron deficiency in pregnancy. Br J Haematol. 2012;156:588–600.
11. Shabert JK. Nutrition during pregnancy and lactation. In: Mahan LK, Escott-Stump S, editors. Krause's food, nutrition, & diet therapy. 11th ed. Philadelphia, PA: Saunders; 2004. p. 182–209.
12. Fernandez-Ballart J. Iron metabolism during pregnancy. Clin Drug Investig. 2000;19 Suppl 1:9–19.
13. Steer PJ. Maternal hemoglobin concentration and birth weight. Am J Clin Nutr. 2000;71 Suppl 31:12831S–7.
14. Xiong X, Buekens P, Alexander S, et al. Anemia during pregnancy and birth outcome: a meta- analysis. Am J Perinatol. 2000;17:137–46.
15. McLean E, Cogswell M, Egli I, Wojdyla D, de Benoist B. Worldwide prevalence of anemia, WHO Vitamin and Mineral Nutrition Information System, 1993–2005. Public Health Nutr. 2008;12:444–54.
16. World Health Organization. Worldwide prevalence of anemia report 1993–2005: WHO global database on anemia. Geneva: World Health Organization. 2008. http://whqlibdoc.who.int/publications/2008/9789241596657_eng.pdf. Accessed 20 Mar 2013.
17. Polhamus B, Dalenius K, Mackintosh H, Smith B, Grummer-Strawn L. Pediatric nutrition surveillance 2008 report. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention; 2009.
18. Berger J, Thanh HTK, Cavalli-Sforza T, et al. Community mobilization and social marketing to promote weekly iron-folic acid supplementation in women of reproductive age in Vietnam: impact on anemia and iron status. Nutr Rev. 2005;63(12 Pt 2):S95–108.
19. Czeizel AE, Dudas I. Prevention of the first occurrence of neural-tube defects by periconceptional vitamin supplementation. N Engl J Med. 1992;327:1832–18331.
20. Czeizel AE, Dob'o MC, Vargha P. Hungarian two-cohort controlled study of periconceptional multivitamin supplementation to prevent certain congenital abnormalities. Birth Defects Res A Clin Mol Teratol. 2004;70:8313–861.
21. Nevin NC, Seller MJ. Prevention of neural tube defect recurrences. Lancet. 1990;1:178–9.
22. MRC Vitamin Study Research Group. Prevention of neural tube defects: results of the Medical Research Council Vitamin Study. Lancet. 1991;338:131–7.
23. Berry RJ, Li Z, Erickson JD, et al. Prevention of neural-tube defects with folic acid in China. China US Collaborative Project for Neural-Tube Defect Prevention. N Engl J Med. 1999;341:1485–90.
24. Lamerts Y. Folate recommendations for pregnancy. Lactation and infancy. Ann Nutr Metab. 2011;59:32–7.
25. Yates RW, Ferfuson-Smith MA, Shenkin A, et al. Is disordered folate metabolism the basis for genetic predisposition to neural tube defects? Clin Genet. 1987;31:279–87.
26. Abrams SA. Nutritional rickets: an old disease returns. Nutr Rev. 2002;60(4):111–1131.
27. Yu CKH, Sykes L, Sethit M, Teoh TG, Robinson S. Vitamin D deficiency and supplementation during pregnancy. Clin Endocrinol (Oxf). 2009;70:685–90.
28. Holick MF, Chen TC. Vitamin D deficiency: a worldwide problem with health consequences. Am J Clin Nutr. 2008;87:1080S–6.
29. Ross AC, Taylor CL, Yaktine AL, Del Valle HB. Editors. Committee to review dietary reference intakes for vitamin D and calcium; Institute of Medicine. Dietary reference intakes for calcium and vitamin D. 2011. The National Academies Press. http://nap.edu/napsearch.php?term=dietary+intakes+for+calcium. Accessed 30 Mar 2013.
30. Viljakainen HT, Saarnio E, Hytinantti T, et al. Maternal vitamin D status determines bone variables in the newborn. J Clin Endocrinol Metab. 2010;95:1749–57.
31. Mahon P, Harvey N, Crozier S, et al. Low maternal vitamin D status and fetal bone development: cohort study. J Bone Miner Res. 2009;25:14–9.
32. Pasco JA, Wark JD, Carlin JB, Ponsonby AL, Vuillermin PJ, Morley R. Maternal vitamin D in pregnancy may influence not only offspring bone mass but other aspects of musculoskeletal health and adiposity. Med Hypotheses. 2008;71:266–9.
33. Grover SR, Morley R. Vitamin D deficiency in veiled or dark-skinned pregnant women. Med J Aust. 2001;1731(31):2311–2.
34. Bodnar LM, Simhan HN, Powers RW, Frank MP, Cooperstein E, Roberts JM. Black and white pregnant women residing in the northern united states and their neonates. J Nutr. 2007;137:447–52.
35. Biesalski H. Vitamin D, recommendations beyond deficiency. Ann Nutr Metab. 2011;59:10–6.
36. Wagner CL, Taylor SN, Dawodu A, Johnson DD, Hollis BW. Vitamin D and its role during pregnancy in attaining optimal health of mother and fetus. Nutrients. 2012;4:208–30.
37. Walker V, Zhang X, Rastegar I, et al. Cord blood vitamin D status impacts innate immune responses. J Clin Endocrinol Metab. 2010;96:1835–43.
38. Utiger RD. The need for more vitamin D. N Engl J Med. 1998;338(12):828–9.

39. Zimmermann M, Delange F. Iodine supplementation of pregnant women in Europe: a review and recommendations. Eur J Clin Nutr. 2004;58:979–84.
40. Caldwell KL, Makhmudov A, Ely E, Jones RL, Wang RY. Iodine status of the U.S. population, National Health and Nutrition Examination Survey, 2005–2006 and 2007–2008. Thyroid. 2011;21(4):419–27.
41. World Health Organization. United Nations Children's Fund & International Council for the Control of Iodine Deficiency Disorders. Assessment of iodine deficiency disorders and monitoring their elimination. 2nd ed. Geneva, Switzerland: WHO; 2007.
42. Morreale G, Obregón MJ, Escobar F. Maternal thyroid hormones early in pregnancy and fetal brain development. Best Pract Res Clin Endocrinol Metab. 2004;18(2):225–48.
43. Ausó E, Lavado-Autric R, Cuevas E, Escobar F, Morreale G, Berbel P. A moderate and transient deficiency of maternal thyroid function at the beginning of fetal neocorticogenesis alters neuronal migration. Endocrinology. 2004;145(9):4037–47.
44. Zimmerman M. Iodine deficiency in pregnancy and the effects of maternal iodine supplementation on the offspring: a review. Am J Clin Nutr. 2009;89(Suppl):668S–72. doi:10.3945/ajcn.2008.26811C.
45. Glinoer D. Feto-maternal repercussions of iodine deficiency during pregnancy. Ann Endocrinol. 2003;64(1):37–44.
46. Murcia M, Rebagliato M, Iñiguez C, et al. effect of iodine supplementation during pregnancy on infant neurodevelopment at 1 year of age. Am J Epidemiol. 2011;173(7):804–12.
47. Forsum E, Löf M. Energy metabolism during human pregnancy. Annu Rev Nutr. 2007;27:277–92.
48. Goldberg GR, Prentice AM, Coward WA, et al. Longitudinal assessment of the components in energy balance in well-nourished lactating women. Am J Clin Nutr. 1991;314:778–98.
49. Forsum E, Kabir N, Sadurskis A, et al. Total energy expenditure of healthy Swedish women during pregnancy and lactation. Am J Clin Nutr. 1992;316:334–42.
50. Goldberg GR, Prentice AM, Coward WA, et al. Longitudinal assessment of energy expenditure in pregnancy using the doubly labeled water method. Am J Clin Nutr. 1993;317:494–31031.
51. Kopp-Hoolihan LE, Van Loan MD, Wong WW, et al. Longitudinal assessment of energy balance in well-nourished, pregnant women. Am J Clin Nutr. 1999;69:697–704.
52. Butte NF, Wong WW, Hopkinson JM. Energy requirements for lactating women derived from doubly labeled water and mild energy output. J Nutr. 2001;131:313–8.
53. Lovelady CA, Meredith CN, McCroy MA, et al. Energy expenditure in lactating women: a comparison of doubly labeled water and heart-rate-monitoring methods. Am J Clin Nutr. 1993;317:3112–8.
54. Kullick K, Dugan L. Exercise and pregnancy. In: Rosenbloom CA, editor. Sports nutrition. 3rd ed. Chicago, IL: American Dietetic Association; 2000. p. 463–76.
55. Luke B, Martin J. The rise in multiple births in the United States: who, what, when, where, and why. Clin Obstet Gynecol. 2004;47:118–33.
56. Rosello-Soberon ME, Fuentes-Chaparro L, Casanueva E. Twin pregnancies: eating for three: maternal nutrition update. Nutr Rev. 2005;63(9):295–302.
57. Giroux I, Inglis SD, Lander S, Gerrie S, Mottola MF. Dietary intake, weight gain, and birth outcomes of physically active pregnant women: a pilot study. Appl Physiol Nutr Metab. 2006;31:483–9.
58. Soultanakis HN, Artal R, Wiswell RA. Prolonged exercise in pregnancy: glucose homeostasis, ventilatory and cardiovascular responses. Semin Perinatol. 1996;20:3131–327.
59. Di Santolo M, Stel G, Banfi G, Gonano F, Cauci S. Anemia and iron status in young fertile non-professional female athletes. Eur J Appl Physiol. 2007;102(6):703–9.
60. Powers S, Nelson WB, Larson-Meyer E. Antioxidant and Vitamin D supplements for athletes: sense or nonsense? J Sports Sci. 2011;29 Suppl 1:S47–55.
61. FAO/WGO/ONU Expert consultation. Energy requirements of pregnancy. In: Human energy requirements. Food and Nutrition Technical Report, vol. 1. Geneva, Switzerland: Author; 2001. p. 20–34.
62. Zhang S, Rattanatray L, Morrison JL, Nicholas LM, Lie S, McMillen IC. Maternal obesity and the early origins of childhood obesity: weighing up the benefits and costs of maternal weight loss in the periconceptional period for the offspring. Exp Diabetes Res. 2011. doi:10.1155/2011/585749.
63. Herring SJ, Rose MZ, Skouteris H, Oken E. Optimizing weight gain in pregnancy to prevent obesity in women and children. Diabetes Obes Metab. 2012;14(3):195–203.
64. Institute of Medicine (IOM). Nutrition during pregnancy. Washington, DC: National Academies Press; 1990.
65. Chu SY, Callaghan WM, Bish CL, d'Angelo D. Gestational weight gain by body mass index among US women delivering live births, 2004–2005: fueling future obesity. Am J Obstet Gynecol. 2009;200(3):2711–7.
66. Gradmark A, Pomeroy J, Renström F, et al. Physical activity, sedentary behaviors, and estimated insulin sensitivity and secretion in pregnant and non-pregnant women. BMC Pregnancy Childbirth. 2011;11:44.
67. Phelan S, Hart C, Phipps M, et al. Maternal behaviors during pregnancy impact offspring obesity risk. Exp Diabetes Res. 2011;985139:1–9. doi:10.1155/2011/985139.
68. Milman N. Iron in pregnancy - how do we secure an appropriate iron status in the mother and child? Ann Nutr Metab. 2011;59:50–4.
69. Scholl T. Maternal iron status: relation to fetal growth, length of gestation, and iron endowment of the neonate. Nutr Rev. 2011;69 Suppl 1:S23–9.
70. Cockell K, Miller DC, Lowell H. Application of the dietary reference intakes in developing a recommen-

dation for pregnancy iron supplements in Canada. Am J Clin Nutr. 2009;90:1023–8.
71. Health Canada. Prenatal nutrition guidelines for health professionals - iron contributes to a healthy pregnancy. 2009. http://www.hc-sc.gc.ca/fn-an/pubs/nutrition/iron-fer-eng.php Accessed 20 Mar 2013.
72. US Department of Health and Human Services, US Department of Agriculture, Dietary Guidelines for Americans, Home and Garden Bulletin No. 232, 20031.
73. United States Department of Agriculture. Center for Nutrition Policy and Promotion Web Site. Dietary Guidelines for Americans. Last modified 14 Mar 2012. http://www.cnpp.usda.gov/DGAs2010-PolicyDocument.htm. Accessed 20 Mar 2013.
74. Johnson DD, Wagner CL, Hulsey TC, McNeil RB, Ebeling M, Hollis BW. Vitamin D deficiency and insufficiency is common during pregnancy. Am J Perinatol. 2011;28:7–12.
75. Hamilton S, McNeil R, Hollis B, et al. Profound Vitamin D deficiency in a diverse group of women during pregnancy living in a sun-rich environment at latitude 32°N. Int J Endocrinol. 2010;2010:917428. doi:10.1155/2010/917428.
76. Center for Disease Control. Spina bifida and anencephaly before and after folic acid mandate—United States, 1995–1996 and 1999–2000. MMWR Morb Mortal Wkly Rep. 2004;53(17):362–5.
77. Czeizel AE, Merhala Z. Bread fortification with folic acid, vitamin B12 and vitamin B6 in Hungary. Lancet. 1998;3312:12231.
78. WHO/UNICEF/UNU. Iron deficiency anemia. Assessment, prevention, and control. A guide for programme managers. Geneva: World Health Organization; 2001.
79. Anderson SA, editor. Guidelines for the assessment and management of iron deficiency in women of childbearing age. Bethesda, MD: Federation of American Societies for Experimental Biology; 1991.
80. Institute of Medicine. Iron-deficiency anemia: recommended guidelines for the prevention, detection, and management among U.S. children and women of childbearing age. Washington, DC: National Academies Press; 1993.
81. Adank C, Green TJ, Skeaff CM, et al. Weekly high-dose folic acid supplementation is effective in lowering serum homocysteine concentrations in women. Ann Nutr Metab. 2003;47:3131–319.
82. Smith AD, Kim YI, Refsum H. Is folic acid good for everyone? Am J Clin Nutr. 2008;87:517–33.

# Nutritional Guidelines, Energy Balance, and Weight Control: Issues for the Aging Active Female

32

Jacalyn J. Robert-McComb, Natalia E. Bustamante-Ara, and José E. Almaraz Marroquin

**Abstract**

During aging total energy expenditure (TEE) decreases by 6 % per decade in women, parallel to the reduction in physical activity. Resting metabolic rate (RMR) decreases 1–2 % per decade and increases from 50 years (3 % per decade). There is a change in body composition not associated with the reduction in RMR or loss of fat-free mass (FFM). This change in body composition produces an increase in fat mass, and it is higher in women than in men. The change in body composition does not always imply a change in body weight (or body mass index). As the caloric intake requirements decrease with aging, the right quality of food and adequate portions become more important. Energy imbalances complicate health and quality of life in both malnutrition and overweight. The 7th edition of the Dietary Guidelines for the USA published in 2010 and incorporating MyPlate in 2011 are available resources to advise people and help improve nutrition, serving as a guide for adults and older active women also. Adequate calorie intakes should be matched to physical activity level in each, providing the required amount of macronutrients, vitamins, and minerals, and possible food supplements for active women to achieve proper weight control, energy balance, and heath.

**Keywords**

Total energy expenditure • Physical activity • Women • Caloric intake

J.J. Robert-McComb, Ph.D., FACSM, ACSM P.D. (✉)
Department of Health, Exercise, and Sports Sciences,
Texas Tech University, Lubbock, TX, USA
e-mail: jacalyn.mccomb@ttu.edu

N.E. Bustamante-Ara, Ph.D.
Physical Activity and Sport, Department
of Physical Activity and Health,
Nengn Company, Maipú, Santiago, Chile
e-mail: natalia_eba@yahoo.es

J.E.A. Marroquin, M.D.
Department of Internal Medicine,
Hospital Universitario de Gran Canaria Dr. Negrín,
Las Palmas, Gran Canaria, Spain
e-mail: Dr_Emilio_Almaraz@hotmail.es

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2_32, 

## 32.1 Learning Objectives

After completing this chapter, you should have an understanding of:

- Energy balance and weight control.
- Dietary guidelines for the mature woman.
- Important nutrients, vitamins, and minerals for physically active women;

## 32.2 Introduction

The nutritional needs of an aging population require special attention. Energy expenditure declines with age; thus, to achieve energy balance, less energy needs to be consumed. The reduction in energy intake can have adverse effects on the nutritional status of older people unless high nutritional quality foods are eaten [1]. Worldwide, micronutrient status of women is inadequate for several micronutrients. The rationale for micronutrient adequacy in the individual woman has been well defined for many micronutrients such as iron, calcium, iodine, folate, and vitamins A and D. However, for older women, especially those living beyond their eighties, more research about nutritional requirements is needed. Important micronutrients for the aging are discussed in this chapter as well as the dietary guidelines for the mature woman.

The decline in energy intake associated with aging also increases risk frailty and mortality [2] in people with low body mass index (BMI). However, obesity is now common in older women. Obesity is also associated with an increase in the prevalence of disability. Conversely, weight loss in overweight older women has been associated with increases in quality of life. Issues in energy balance and weight control are also highlighted in this chapter.

The Food and Nutrition Information Center (FNIC) is a leader in online global nutrition information. It is located at the National Agricultural Library (NAL) of the United States Department of Agricultural (USDA). The FNIC Web site contains over 2,500 links to current and reliable nutrition information. The FNIC provides links to the Dietary Reference Intakes (DRI) tables and reports discussed in this chapter. These tables and reports have been developed by the Institute of Medicine's Food and Nutrition Board. Appendices 1 and 2 contain a list of the valuable information that you can assess at http://fnic.nal.usda.gov/dietary-guidance.

## 32.3 Research Findings

### 32.3.1 Energy Balance and Weight Control

Generally, positive energy balance leads to weight gain and negative balance to weight loss. Changes in body composition are due to alterations in energy balance; however, this is not as simple as it seems. The aging process brings about many changes in body composition, often without concomitant changes in body weight and BMI. In the aging, body fat percentage (BF%) increases and leans body mass (LBM) and bone mineral density (BMD) decrease. The increase in fat mass (FM) is distributed more specifically in the abdominal region, an area associated with cardiovascular disease and diabetes [3]. Additionally, there is a difference in FM and body fat distribution between the sexes. Women are more efficient in conserving and storing energy as fat. It is also known that postmenopausal women experience an increase in the waist-to-hip ratio [4]. Women lose less fat-free mass (FFM) compared with men with similar weight loss (27.3 % vs. 25.4 % men and women respectively). Supporting this notion is the recognition that women must reduce their dietary

intake by a higher proportion to achieve the same degree of weight loss as men [4].

Following the age of 40 years, total energy expenditure (TEE) begins to decline quite dramatically. Women 75 years old or more experience TEE levels similar to a 7–11 year old, despite having greater body mass. In order to fully understand TEE, some definitions need to be clarified using a single authoritative source [5]. In the research literature, terms are sometimes expressed with slight variations.

- The *basal metabolic rate* (*BMR*) describes the rate of energy expenditure that occurs in the postabsorptive state
- The BMR is commonly extrapolated to 24 h to be more meaningful, and it is then referred to as *basal energy expenditure* (*BEE*), expressed as kcal/24 h.
- *Resting metabolic rate* (*RMR*), energy expenditure under resting conditions, tends to be somewhat higher (10–20 %) than under basal conditions due to increases in energy expenditure caused by recent food intake (i.e., by the "thermic effect of food") or by the delayed effect of recently completed physical activity.
- *Resting energy expenditure* (*REE*) is RMR extrapolated to 24 h.
- *The thermic effect of food* (*TEF*) was originally known as the Specific

Dynamic Action (SDA) of food. The intensity and duration of meal-induced TEF is determined primarily by the amount and composition of the foods consumed and the associated metabolic costs.

- The *physical activity level of index* (*PAL*) is a way to express a person's daily physical activity as a number, and is used to estimate a person's TEE. The PAL is defined for a nonpregnant, non-lactating adult as that person's TEE in a 24-h period, divided by his or her BEE or PAL = TEE/BEE.
- The *physical activity coefficient* (*PA*) is used in the formula found in Table 32.3 to determine estimated energy requirements (EER) where PA for girls 3–18 years old is as follows:
  PA = 1.00 if PAL is estimated to be ≥1.0 < 1.4 (sedentary);
  PA = 1.16 if PAL is estimated to be ≥1.4 < 1.6 (low active);
  PA = 1.31 if PAL is estimated to be ≥1.6 < 1.9 (active);
  PA = 1.56 if PAL is estimated to be ≥1.9 < 2.5 (very active).
- The *physical activity coefficient* (*PA*) is used in the formula found in Table 32.3 to determine estimated energy requirements (EER) where PA for women 19+ years is as follows:
  PA = 1.00 if PAL is estimated to be ≥1.0 < 1.4 (sedentary)
  PA = 1.12 if PAL is estimated to be ≥1.4 < 1.6 (low active)
  PA = 1.27 if PAL is estimated to be ≥1.6 < 1.9 (active)
  PA = 1.45 if PAL is estimated to be ≥1.9 < 2.5 (very active)
- *TEE* is the sum of BEE, which includes a small component associated with arousal, as compared to sleeping, the TEF, physical activity, thermoregulation, and the energy expended in depositing new tissues and in producing milk.
- The *estimated energy requirement* (*EER*) is the average dietary energy intake that is predicted to maintain energy balance in a healthy adult of a defined age, gender, weight, height, and level of physical activity consistent with good health. In children and pregnant and lactating women, the EER is taken to include the needs associated with the deposition of tissues or the secretion of milk at rates consistent with good health.

Thus, it is important to distinguish between BMR and RMR. Because RMR is much easier to measure than BMR, RMR is frequently seen in the literature as a component of TEE. TEE is thus comprised of RMR, the TEF, and activity energy expenditure (AEE) [6]. Activity energy expenditure (AEE) is the modifiable component of TEE derived from all activities, both volitional and nonvolitional. Thus, TEE (kcal/day) = RMR + AEE + TEF [7].

With increasing age, TEE decreases for both RMR and AEE. This decrease in TEE with age is associated with reductions in body mass and FFM [6]. TEE decreases 6 % per decade for women, as a result of decreases in physical

activity energy expenditure [8]. TEE remains higher for men than for women. However, when adjusted for FMM, TEE is higher for women than for men [9].

RMR is one of the largest components of TEE, comprising 50–80 %, and it has previously been estimated to decline 1–2 % per decade after 20 year. This decline in RMR with age may not be linear, breakpoint decline quickly becomes apparent around 50 years in women; this may be due to an accelerated loss of FFM during menopause [10, 11]. Recently, longitudinal studies indicated 3 % reductions in RMR per decade in women respectively, and the rate of decline in RMR was faster at age 70–80 years than at age 40–50 years [8].

Aging is associated with a decrease in almost all components of the equation: ↓RMR (change in FM and FFM), AEE (change in activity level), as well as energy intake. Yet, it remains unclear what factor initiates the change. The TEF contributes <10 % to TEE and does not decline with aging per se [11]. In many circumstances, aging and diseases might contribute to a decrease or increase in RMR [6]. The lower RMR of older adults may be due in part to slowed organ metabolic rates, and this may contribute to changes in FM, FFM, and fat distribution [12]. This could also be due, for example, to morphological changes like infiltration of the organs with fat, edema, or cystic structures [13] and change in fat oxidation. However, the decline in RMR is not entirely due to changes in body composition [3].

Older women maintain lower levels of AEE than men (576 kcal/day vs. 769 kcal/day) until the seventh decade of their life (seventies). During their seventies older women have similar physical activity levels (PAL) as men [14]. Older women may ameliorate the age related decrease in RMR, through increased physical activity to preserve body composition. There is a strong association [10] between physical activity levels and FFM [6, 7].

The use of a measure or an estimate of TEE to validate instruments that measure food intake is dependent on the principle of energy balance. That is, in weight-stable adults, energy intake must equal TEE. In a balanced state, TEE corresponds to EER.

Recommendations for caloric intake to maintain weight will vary depending on a person's age, sex, size, and level of physical activity. Specific equations for estimating caloric needs are provided in the Dietary Reference Intakes for Energy Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids [5]. These reports can be found in the USDA's NAL and can be downloaded free of charge in a PDF file (http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes/dri-reports). Web pages change with time, if the page is no longer available, type in DRI reports in the search box at http://fnic.nal.usda.gov/.

The most accurate way to assess TEE is through the doubly labeled water technique (DLW). The DLW technique is considered the gold standard measure of free-living activity expenditure in conjunction with direct calorimetric for measurement of RMR. However, this method is expensive and impractical in many healthcare settings, and therefore predictive equations are used to estimate RMR in most clinical and inpatient care practices.

The Mifflin–St Jeor equation is more likely than other equations to estimate RMR within 10 % of the measured [15] and is estimated from weight, height, and age. Multiple-regression analyses were employed to drive relationships between RMR and weight, height, and age for both sexes ($R^2 = 0.71$) but separation by sex did not affect its predictive value.

RMR = 9.99 × weight (kg) + 6.25 × height (cm) − 4.92 × age (year) + 166 × sex (males, 1; females, 0) − 161 [16]. This equation has also been validated in obese population [17]. Table 32.1 details the process to estimate total caloric needs depending on activity level using the Mifflin–St Jeor multiple regression equation to estimate RMR. For example, if a person's RMR was 1,000 and they were doing heavy activity, you would multiply 1,000 times the percentage above rest 100 % or 1.00 and add that value to their estimated RMR, 1,000 (RMR) + 1,000(1,000 × 1 for additional calories above rest) = 2,000. This value would be their estimated energy expenditure in kcal/day using the method presented in Table 32.1.

**Table 32.1** An estimated energy expenditure prediction equation using the Mifflin–St Jeor equation to determine resting metabolic rate

| |
|---|
| *Step 1*: *Estimate resting metabolic rate* (*RMR*) *using the Mifflin–St Jeor equation* |
| RMR = 9.99 × weight (kg) + 6.25 × height (cm) − 4.92 × age (year) + 166 × sex (males, 1; females, 0) − 161. |
| *Step 2*: *Determine additional caloric requirements based on level of activity* |

| Physical activity level | Percentage above resting level |
|---|---|
| Bed rest | 10 |
| Quiet rest | 30 |
| Light activity | 40–60 |
| Moderate activity | 60–80 |
| Heavy activity | 100 |

| |
|---|
| Additional caloric requirements = RMR × Percentage above resting level |
| *Step 3*: *Determine predicted total energy expenditure* (*TEE*) |
| TEE = RMR + Additional caloric requirements based on activity |
| TEE = predicted energy expenditure in kcal/day |

Adapted from Physiology of Fitness (3rd ed.) (p. 359) by B. J. Sharkey, 1990, Champaign, IL: Human Kinetics

The PAL can be used as an indirect index of physical activity and is useful in recommending energy intakes based on RMR and a PAL value [18]. In this instance, PAL is calculated as TEE/RMR [14] The PAL value establishes the difference between a sedentary and a very active person (the greater the value, the greater level of activity) [14]. The PAL value establishes categories for PA coefficient defined as: sedentary = 1.0; low active = 1.12; active = 1.27; and 1.45 very active for adult women >19 years and older [5, 19].

The EER of mature persons using their PAL (as multiples of RMR), provides a convenient and practical way of controlling for age, sex, body weight and body composition, and the energy intake needs of a wide range of people in a quick way [20].

A Report of the Panel on Macronutrients, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference [5] provides equations for calculating EER based on sex, age, height, weight, and PA coefficient. The equation for adult women >19 years and older is as follows:

$$EER = 354 - (6.91 \times AGE) + PA \times (9.36 \times WT + 726 \times HT).$$

TEE measured from DLW and the equations in the referenced report [5] were highly correlated.

Tables 32.2 and 32.3 contain the information to calculate EER using PA coefficient, weight, sex, age, and height for female infants, girls and women. The equations were validated in terms of their intended use to estimate EER for healthy individuals but the equations were not validated for use in nutritional epidemiology or surveillance studies [9].

## 32.4 Contemporary Understanding of the Issues

### 32.4.1 Dietary Guidelines for the Mature Woman

The aim of food-based dietary guidelines is to reduce chronic malnutrition, micronutrient malnutrition, and diet-related communicable and non-communicable diseases. Food-based dietary guidelines allow the principles of nutrition education to be expressed, qualitatively and quantitatively [1]. The Dietary Guidelines of Americans is published jointly every 5 years by the Department of Health and Human Services (HHS) and the USDA. The 2010 Dietary Guidelines for Americans, 2010 [21] was released January 31, 2011 and can be downloaded at:

http://www.health.gov/dietaryguidelines/dga2010/DietaryGuidelines2010.pdf.

The Dietary Guidelines for Americans, 2010 focuses on three major goals for Americans.

- Balance your intake of calories with physical activity to manage weight.
- Increase your intake of certain foods and nutrients such as fruits, vegetables, whole grains, fat-free and low-fat dairy products, and seafood.
- Decrease your intake of foods with sodium (salt), saturated fats, *trans* fats, cholesterol, added sugars, and refined grains.

**Table 32.2** Physical activity level index (PAL) and physical activity coefficient (PA) used to derive estimated energy requirements (EER) for women

| | Sedentary | Low active | Active | Very active |
|---|---|---|---|---|
| PAL | (1.0–1.39) | (1.4–1.59) | (1.6–1.89) | (1.9–2.5) |
| | Typical daily living activities (e.g., household tasks, walking to the bus) | Typical daily living activities PLUS 30–60 min of daily moderate activities (e.g., walking at 5–7 km/h) | Typical daily living activities PLUS at least 60 min of daily moderate activities | Typical daily living activities PLUS at least 60 min of daily moderate activities an additional 60 min of vigorous activity or 120 min of moderate activity |
| PA | PA (level 1) | PA (level 2) | PA (level 3) | PA (level 4) |
| Girls 3–18 year | 1.00 | 1.16 | 1.31 | 1.56 |
| Women 19 year+ | 1.00 | 1.12 | 1.27 | 1.45 |

Adapted from A Report of the Panel on Macronutrients, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for energy carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (*macronutrients*). Washington DC: National Academy Press; 2005
Complete report can be viewed and downloaded at http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes/dri-reports
PAL = Physical activity level or physical activity index
PA = Physical activity coefficient

Furthermore, the Dietary Guidelines for Americans, 2010 include 23 key recommendations for the general population and six additional key recommendations for specific population groups [21]. According to research, the diet quality of Americans age 65 and older did not significantly improve in the past decade. To improve diet quality, the USDA suggests that older Americans need to increase their intakes of whole grains, dark green and orange vegetables, legumes, and milk. They also need to choose more nutrient-dense forms of foods. These changes, if made, would provide substantial health benefits [22]. The intention of the new dietary recommendations is to stimulate people to make more thoughtful choices: Choices that will reflect healthier foods and portion sizes that are appropriate for their caloric needs. It is also hoped that the physical activity of Americans is increased along with the healthier food choices, thereby reducing the risk of developing diet-related chronic disease [23].

The nutrition information found in the seventh edition of the Dietary Guidelines for Americans, 2010 (http://www.cnpp.usda.gov/)will help the aging woman choose nutrient dense foods for an adequate diet [21]. Appendix 3 displays the USDA food groups and recommended subgroups to select in your diet. Appendix 4 outlines the USDA's healthy choices for a desired caloric intake. The patterns of behaviors correlated with a healthy body weight are as follows:

- focus on the total number of calories consumed;
- monitor food intake;
- when eating out, choose smaller portions or lower-caloric options;
- prepare, serve, and consume smaller portions of foods and beverages, especially those high in calories;
- eat a nutrient-dense breakfast; and
- limit screen time

The total numbers of calories a person needs each day varies depending on a number of factors. These factors include: age, weight, gender, height, and level of physical activity. Generally one of the significant age-associated changes is that the need for energy decreases [24]. In order to maintain, lose or gain weight, the caloric needs of an individual should be known. However, even when caloric needs are known, many women find it impossible to lose weight or maintain a healthy weight after weight loss. A growing body of research has begun to describe overall

**Table 32.3** Equations to estimate energy requirement

| | |
|---|---|
| *Infants and young children* | |
| Estimated energy requirement (kcal/day) = Total energy expenditure + Energy deposition | |
| 0–3 months | EER = (89 × weight [kg] − 100) + 175 |
| 4–6 months | EER = (89 × weight [kg] − 100) + 56 |
| 7–12 months | EER = (89 × weight [kg] − 100) + 22 |
| 13–35 months | EER = (89 × weight [kg] − 100) + 20 |
| *Children and adolescents 3–18 years* | |
| Estimated energy requirement (kcal/day) = Total energy expenditure + Energy deposition | |
| Girls | |
| 3–8 years | |
| EER = 135.3 − (30.8 × age [year]) + PA × [(10.0 × weight [kg]) + (934 × height [m])] + 20 | |
| 9–18 years | |
| EER = 135.3 − (30.8 × age [year]) + PA × [(10.0 × weight [kg]) + (934 × height [m])] + 25 | |
| *Adults 19 years and older* | |
| Estimated energy requirement (kcal/day) = Total energy expenditure | |
| Women | |
| EER = 354 − (6.91 × age [year]) + PA × [(9.36 × weight [kg]) + (726 × height [m])] | |
| Pregnancy | |
| Estimated Energy Requirement (kcal/day) = Nonpregnant EER + Pregnancy Energy Deposition | |
| First trimester | EER = Nonpregnant EER + 0 |
| Second trimester | EER = Nonpregnant EER + 340 |
| Third trimester | EER = Nonpregnant EER + 452 |
| Lactation | |
| Estimated energy requirement (kcal/day) = Nonpregnant EER + Milk energy output − Weight loss | |
| 0–6 months postpartum | EER = Nonpregnant EER + 500 − 170 |
| 7–12 months postpartum | EER = Nonpregnant EER + 400 − 0 |

*Note*: These equations provide an estimate of energy requirement. Relative body weight (i.e., loss, stable, gain) is the preferred indicator of energy adequacy
*Note*: See Table 32.2 to find the appropriate PA value to use in these equations
Adapted from: A Report of the Panel on Macronutrients, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for energy carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (**macronutrients**). Washington DC: National Academy Press; 2005
Complete report can be viewed and downloaded at http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes/dri-reports
EER = Estimated Energy Requirement
PA = Physical Activity Coefficient

eating patterns that help promote caloric balance and weight management. One researched aspect of these eating patterns is the concept of energy density, or the amount of calories provided per unit of food weight [25].

Energy density (ED) is the amount of energy per weight of food or beverage (kilojoules/g [kJ/g] or kilocalories/gram [kcal/g]). Foods high in water and/or fiber are lower in ED, while foods high in fat are higher in ED, increasing calorie intakes. The 2010 Dietary Guidelines Advisory Committee (DGAC) indicated that there is strong and consistent evidence in adults that a decrease in dietary ED improves weight loss and weight maintenance. Choosing foods that have a lower ED may also be associated with a lower risk of type 2 diabetes in adults [25].

Weight gains following menopause may be an indicator of relapses in weight reduction. More than 50 % of women in the menopausal period

**Table 32.4** Nutritional goals for female groups and age, based on dietary reference intakes and dietary guidelines recommendations

| Nutrient (units) | Female 31–50 | Female 51+ | Source of goal[a] |
|---|---|---|---|
| Macronutrients | | | |
| Protein | 46 g | 46 g | RDA[b] |
| Calories | 10–35 % | 10–35 % | AMDR[c] |
| Carbohydrate | 130 g | 130 g | RDA |
| Calories | 45–65 % | 45–65 % | AMDR |
| Total fiber | 25 g | 22 g | IOM[d] |
| Total fat (calories) | 20–35 % | 20–35 % | AMDR |
| Saturated fat (calories) | <10 % | <10 % | DG[e] |
| Linoleic acid | 12 g | 11 g | AI[f] |
| Calories | 5–10 % | 5–10 % | AMDR |
| Alpha-Linolenic acid | 1.1 g | 1.1 g | AI |
| Calories | 0.6–1.2 % | 0.6–1.2 % | AMDR |
| Cholesterol | <300 mg | <300 mg | DG |

*Sources*: Britten P, Marcoe K, Yamini S, Davis C. Development of food intake patterns for the MyPyramid Food Guidance System. J Nutr Educ Behav 2006;38(6Suppl):S78–S92

IOM. Dietary Reference Intakes: The essential guide to nutrient requirements. Washington (DC): The National Academies Press; 2006

IOM. Dietary Reference Intakes for Calcium and Vitamin D. Washington (DC): The National Academies Press; 2010. Revised Page 76, 77 pdf found at http://www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/PolicyDoc/PolicyDoc.pdf

Complete report can be viewed and downloaded at http://www.cnpp.usda.gov

[a]Dietary guidelines recommendations are used when no quantitative Dietary Reference Intake value is available; apply to ages 2 years and older

[b]Recommended dietary allowance, IOM

[c]Acceptable macronutrient distribution range, IOM

[d]14 g per 1,000 cal, IOM

[e]Dietary guidelines recommendation

[f]Adequate intake, IOM

attempt to restrict their calories in an attempt to lose weight; however, they are unsuccessful at weight loss maintenance. Peak body weight gain is observed around age 50 [26].

From a clinical point of view, this fact is of great interest. Diets that are reduced in calories must also have macronutrient proportions that are within the ranges recommended in the Dietary References Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Proteins, and Amino Acids (protein: 10–35 %; carbohydrate: 45–65 %; fat: 20–35 %) [5]. Furthermore ED is also an important component of choosing the right food combinations that helps you lose weight and maintain the weight loss [25]. Nutritional goals for female 31–50 and +50 years based on dietary reference intakes and dietary guidelines recommendations are presented in Table 32.4.

Recently, in June 2011, MyPlate replaced MyPyramid, a new initiative designed to make healthy food choices more attractive to the consumer. MyPlate illustrates five food groups in their design with bold colors [23, 27]. The USDA provides a Web site in which a food plan can be customized, at (http://www.ChooseMyPlate.gov/). This Web site helps consumers choose a healthy personal eating plan. Physical activity is also emphasized based on the recommendations of the American College of Sports Medicine (ACSM) [28].

Topics such as *what is physical activity*, *why is physical activity important*, *how much physical activity do I need* can be answered on this site.

Solid fats are abundant in the diets of Americans and contribute significantly to excess caloric intake and weight gain. A fat intake recommendation for older women is: 20–35 % of

total daily calories. These ranges are associated with reduced risk of chronic diseases, such as cardiovascular disease, while providing for adequate intake of essential nutrients. The recommendation is to keep *trans* fatty acid consumption as low as possible, especially by limiting foods that contain synthetic sources of *trans* fats, such as partially hydrogenated oils, and by limiting other solid fats. All individuals, not just older women, should consume less than 10 % of their daily calories from saturated fatty acids. Saturated fatty acids should be replaced with monounsaturated and polyunsaturated fatty acids [21].

Among the significant age-associated changes in nutrient requirements is that the need for protein increases with age [24]. Protein requirements of older adults may be higher than the current recommended level of 0.8 g/kg/day. Despite an estimated increase in mean protein requirements for older adults, a recent study indicated that there is no difference between the young and old when the protein requirements are expressed per kilogram FFM. The recommended level is now 0.85 g/kg/day for both the young and old adult [29].

There are a wide variety of recommendations about the specific value of protein requirements; however, the data is non-conclusive with regard to the best recommendation. Nevertheless, dairy products digest quickly and have high biological levels of protein that contain essential amino acids (meaning that the human body cannot synthesize it, and it therefore must be ingested) such as leucine. Milk, a good source of high biological value protein, is particularly rich in essential and branched-chain amino acids. Protein metabolites, such as small peptides, have been shown to have bioactive properties [30]. Bioactive describes something that can have an effect on living tissue, such as the effect of the sun rays on the skin.

In the USA, total consumption of sugar has increased substantially in recent decades [31]. The obesity epidemic has focused attention on the relationship of sugars and sugar-sweetened beverages (SSB)—particularly glucose, sucrose, and fructose, e.g., as high-fructose corn syrup [32]. Higher consumption of carbohydrates have been associated with dyslipidemia, a lipid profile known to increase cardiovascular disease risk including lower HDL-C levels, higher triglyceride levels, and higher ratios of triglycerides to HDL-C. The consumption of large amounts of added sugars, a prominent source of low-nutrient calories, is a relatively new phenomenon. For mature women it is advised to limit their added sugars to fewer than 100 cal daily (approximately 5 % of total energy intake) [31].

Daily sodium intake should be reduced to less than 2,300 mg. Adults 51 years of age or more should further reduce their intake to 1,500 mg. Adults of any age who are African American or have hypertension, diabetes, or chronic kidney disease should also reduce their intake of sodium to 1,500 mg. The 1,500 mg recommendation applies to about half of the US population [21].

Since hypertension is a major public health problem affecting millions of adult women, the Dietary Approach to Stop Hypertension (DASH) diet has been created. This diet plan, DASH, is designed to: reduce the intake of saturated fat, total fat, sodium, and cholesterol; to increase the intake of fruits and vegetables; and to increase the consumption of potassium, calcium, magnesium, fiber, and protein. Adherence to DASH is a key component to controlling blood pressure [33]. Table 32.5 compares the usual US intake, the recommended DASH intake, and the USDA food patterns adjusted to a 2,000 cal level. Table 32.6 presents three different caloric intakes according to the DASH eating plan.

Adverse Blood Pressure (BP)—prevalent worldwide—is an independent major risk factor for cardiovascular diseases (CVD). Established modifiable risk factors for elevated BP are high sodium intake, inadequate potassium intake, high body mass index (BMI), and excessive alcohol intake [32]. Sodium and potassium have opposing effects on arterial vasodilation [30].

### 32.4.2 Important Nutrients, Vitamins, and Minerals For Physically Active Women

The micronutrient and macronutrient needs of individuals, men and women alike, who are physically active, has always been a subject of

**Table 32.5** Pattern comparison: Usual US intake, DASH, and USDA food Patterns, average daily intake at or adjusted to a 2,000 cal level

| Pattern | Usual US intake adults[a] | DASH[b] | USDA food pattern |
|---|---|---|---|
| Food groups | | | |
| Vegetables | 1.6 cups | 2.1 cups | 2.5 cups |
| Fruit and juices | 1.0 cups | 2.5 cups | 2.0 cups |
| Grains | 6.4 oz | 7.3 oz | 6.0 oz |
| Whole grains | 0.6 oz | 3.9 oz | ≥ 3.0 oz |
| Milk and milk products (dairy products) | 1.5 cups | 2.6 cups | 3.0 cups |
| Protein foods | | | |
| Meat | 2.5 oz | 1.4 oz | 1.8 oz |
| Poultry | 1.2 oz | 1.7 oz | 1.5 oz |
| Fish/seafood | 0.5 oz | 1.4 oz | 1.2 oz |
| Nuts, seeds, and soy products | 0.5 oz | 0.9 oz | 0.6 oz |
| Oils | 18 g | 25 g | 27 g |
| Solid fats | 43 g | nd | 16 g[c] |
| Added sugars | 79 g | 12 g | 32 g[c] |
| Alcohol | 9.9 g | nd[d] | nd[e] |

Complete report can be viewed and downloaded at http://www.cnpp.usda.gov/

[a]*Source*: US Department of Agriculture, Agricultural Research Service and US Department of Health and Human Services, Centers for Disease Control and Prevention. What We Eat In America, NHANES 2001–2004, 1 day mean intakes for adult males and females, adjusted to 2,000 cal and averaged

[b]See the DGAC report for additional information and references at www.dietaryguidelines.gov

[c]Amounts of solid fats and added sugars are examples only of how calories from solid fats and added sugars in the USDA Food Patterns could be divided

[d]nd = Not determined

[e]In the USDA Food Patterns, some of the calories assigned to limits for solid fats and added sugars may be used for alcohol consumption instead

debate. The intensity, duration, and frequency of the physical activity as well as the overall nutrient intake of the individual have an impact on whether or not micronutrients and macronutrients are required in greater amounts. The Dietary Reference Intakes (DRIs) for macronutrients, vitamins, and minerals for females of all ages, regardless of level of physical activity, as established by the Food and Nutrition Board, Institute of Medicine, and National Academies can be found in the Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients) [5]. The report is referenced in Appendix 2 and can be downloaded at http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes/dri-reports. There is no charge for the PDF download.

Generally, the vitamin and minerals needs of active individuals are not greater than those who are not active, if the DRIs are being met. However, it has been shown that frequently women, of all ages, do not meet their nutrient needs through diet alone, and therefore supplementation may be necessary. Older adults are at even greater risk for nutritional deficiencies than are younger adults due to physiologic changes associated with aging. Therefore, it is recommended that individuals 50 years and older consume food fortified cereals, or dietary supplements [21]. Nonetheless, mega dosing with one vitamin and/or mineral can impair the functions of other vitamins and minerals.

The DRIs suggest dietary carbohydrate should be in the range of 45–65 % of total calories. Using the recommendations of 5–7 g/kg/day for general training needs, a 54.4 kg (120 lb) woman would need roughly 272–380 g of carbohydrate. The typical US diet provides 4–5 g/kg/day, and athletes who train daily and compete at high intensity

**Table 32.6** The DASH eating plan at various calorie levels. The number of daily servings in a food group varies depending on caloric needs[a]

| Calories | 1,600 | 1,800 | 2,000 | Serving sizes |
|---|---|---|---|---|
| Food groups[b] | | | | |
| Vegetables | 3–4 | 4–5 | 4–5 | 1 cup raw leafy vegetable, ½ cup cut-up raw or cooked vegetable, ½ cup vegetable juice |
| Fruit and juices | 4 | 4–5 | 4–5 | 1 medium fruit, ¼ cup dried fruit ½ cup fresh, frozen, or canned fruit, ½ cup fruit juice |
| Grains | 6 | 6 | 6–8 | 1 slice bread 1 oz dry cereal[c] ½ cup cooked rice, pasta, or cereal[c] |
| Fat-free or low-fat milk and milk products | 2–3 | 2–3 | 2–3 | 1 cup milk or yogurt, 1½oz cheese |
| Lean meats, poultry, and fish | 3–4 or less | 6 or less | 6 or less | 1 oz cooked meats, poultry, or fish, 1 egg |
| Nuts, seeds, and legumes | 3–4 per week | 4 per week | 4–5 per week | 1/3 cup or 1½oz nuts, 2 Tbsp peanut butter, 2 Tbsp or ½oz seeds, ½ cup cooked legumes (dried beans, peas) |
| Fats and oils | 2 | 2–3 | 2–3 | 1 tsp soft margarine, 1 tsp vegetable oil, 1 Tbsp mayonnaise, 1 Tbsp salad dressing |
| Sweets and added sugars | 3 or less per week | 5 or less per week | 5 or less per week | 1 Tbsp sugar, 1 Tbsp jelly or jam, ½ cup sorbet, gelatin dessert,1 cup lemonade |
| Maximum sodium limit[e] | 2,300 mg/day | 2,300 mg/day | 2,300 mg/day | |

*Source*: USDA and HHS, US Department of Agriculture and US Department of Health and Human Services. Dietary Guidelines for Americans, 2010. Washington, DC: Complete report can be viewed and downloaded at http://www.cnpp.usda.gov/

[a]The DASH eating Patterns from 1,600 to 3,100 cal meet the nutritional needs of children 9 years and older and adults

[b]Significance to DASH Eating Plan, selection notes, and examples of foods in each food group

*Grains*: Major sources of energy and fiber. Whole grains are recommended for most grain servings as a good source of fiber and nutrients. Examples: Whole-wheat bread and rolls; whole-wheat pasta, English muffin, pita bread, bagel, cereals; grits, oatmeal, brown rice; unsalted pretzels and popcorn

*Vegetables*: Rich sources of potassium, magnesium, and fiber. Examples: Broccoli, carrots, collards, green beans, green peas, kale, lima beans, potatoes, spinach, squash, sweet potatoes, tomatoes

*Fruits*: Important sources of potassium, magnesium, and fiber. Examples: Apples, apricots, bananas, dates, grapes, oranges, grapefruit, grapefruit juice, mangoes, melons, peaches, pineapples, raisins, strawberries, tangerines

*Fat-free or low-fat milk and milk products*: Major sources of calcium and protein. Examples: Fat-free milk or buttermilk; fat-free, low-fat, or reduced-fat cheese; fat-free/low-fat regular or frozen yogurt

*Lean meats*, *poultry*, *and fish*: Rich sources of protein and magnesium. Select only lean; trim away visible fats; broil, roast, or poach; remove skin from poultry. Since eggs are high in cholesterol, limit egg yolk intake to no more than 4 per week; 2 egg whites have the same protein content as 1 oz meat

*Nuts, seeds, and legumes*: Rich sources of energy, magnesium, protein, and fiber. Examples: Almonds, filberts, mixed nuts, peanuts, walnuts, sunflower seeds, peanut butter, kidney beans, lentils, split peas

*Fats and oils*: DASH study had 27 % of calories as fat, including fat in or added to foods. Fat content changes serving amount for fats and oils. For example, 1 Tbsp regular salad dressing=one serving; 2 Tbsp low-fat dressing=one serving; 1 Tbsp fat-free dressing=zero servings. Examples: Soft margarine, vegetable oil (canola, corn, olive, safflower), low-fat mayonnaise, light salad dressing

*Sweets and added sugars*: Sweets should be low in fat. Examples: Fruit-flavored gelatin, fruit punch, hard candy, jelly, maple syrup, sorbet and ices, sugar

[c]Serving sizes vary between ½ cup and 1¼ cups, depending on cereal type. Check product's Nutrition Facts label

[e]The DASH Eating Plan consists of patterns with a sodium limit of 2,300 and 1,500 mg/day

need more carbohydrate [34]. Therefore, women who train daily would need more carbohydrates than the typical US diet would provide.

## 32.5 Future Directions

As individuals are living longer than the lifespan expectancies of their parents for the most part, supplementation may be needed for the aging woman who wants to be vital and competitive in recreational sports and activities. Among the micronutrients, the significant ones that may be associated with deficiencies in mature active women include vitamin B-12, vitamin A, vitamin C, vitamin D, calcium, iron, zinc, and other trace minerals [35]. Since essential fatty acids help in the absorption of the fat-soluble vitamins A, D, E, and K, it is important that women intake healthy levels of fats ranging from 20 to 35 % of total daily calories (<10 % saturated).

Taking antioxidants including carotenoids, vitamin C, flavonoids, and other polyphenols through vegetables is important because of an associated beneficial decrease in CVD risk. Natural antioxidants are present in the human diet in many different chemical forms [36]. The need for energy adjusted antioxidant intake from diet increases with age and exercise level (except for flavonoids). Furthermore, the need for antioxidant intake is higher in older very active woman (supplementation may be necessary). Interestingly, low total serum carotenoid concentrations are associated with low walking speed and greater decline of walking speed [37]. Thus, fortified foods and supplements may be needed in order to meet the DRIs of these micronutrients in the older women.

Multi-nutrient supplementation may improve indices of inflammation and help exercise recovery in active older women. In addition to improving energy, supplements may prolong functionality and physiological performance with age. Thus supplements may allow older women to maintain an active lifestyle and promoting a cycle of anti-inflammatory and anti aging activity [38].

With regard to the effects of exercise on vitamin and minerals needs, irrespective of age, thiamin, vitamin C, E, calcium, and iron are discussed in more detail in this chapter under the category, Future Directions, since research has shown that the DRI for these micronutrients may be greater in exercising individuals. Vitamin $B_{12}$, folate, and vitamin A are also discussed in Future Directions because of the importance of these micronutrients from a health perspective for the older woman.

### 32.5.1 Vitamin $B_{12}$ and Folate

The benefits of vitamin $B_{12}$ and folic acid fortification/supplementation are not only applicable to women throughout the lifecycle but also to all sectors of the population due to vitamin $B_{12}$'s ability to lower homocysteine level [39]. Folic acid is also emerging as important in lowering the risk of certain types of cancers [40].

On average, Americans 50 years and older consume adequate levels of vitamin $B_{12}$. Nonetheless, a substantial proportion of individuals 50 years and older may have reduced ability to naturally absorb vitamin $B_{12}$. They are encouraged to include foods fortified with vitamin $B_{12}$, such as fortified cereals, or take dietary supplements [21]. Supplement $B_{12}$ is easily available, adequately absorbed, and well tolerated in older adults.

Even if adequate supplementation raises the level to an acceptable range, a physician should assess the individual with the deficiency because the deficiency may be caused by disease. Vitamin $B_{12}$ deficits are associated with impaired peripheral nerve function and the development of anemia. Nerve function impairment may lead to declines in physical function and disability in older adults [41].

### 32.5.2 Vitamin A

Vitamin A has many roles in the maintenance of health; it is important to maintain normal vision, for cell differentiation, efficient immune function, and genetic expression. Obtaining supplemental vitamin A in its precursor form β-carotene, appears to be considerably safer, more effective,

and has not been associated with adverse or unanticipated side effects.

Physical activity and total serum carotenoids are strong and independent predictors of survival in older women [42]. It is important for active women to maintain high intakes of fruit and vegetables. Consuming a diet rich in fruits and vegetables is a reasonable way to meet vitamin A needs in older adults as well as providing a good source of dietary fiber [35].

Together with robust levels of physical activity, high total serum carotenoid concentration offers women some added health protection [41].

### 32.5.3 Thiamin

Thiamin, also known as vitamin B1 and aneurin (a less common name for thiamin), functions as a coenzyme in the metabolism of carbohydrates and branched-chain amino acids [43]. The DRI for women age 19–70 is 1.1 mg/day. For women who are pregnant or following birth of their child, during lactation, the requirement is higher, 1.4 mg/day. However, those who engage in physically demanding occupations or who spend much time training for active sports may require increased levels of thiamin intake [44].

### 32.5.4 Vitamin C

Women tend to have higher blood levels of vitamin C than men of the same age, even when intake levels are the same, making the requirements for women lower than for men. However, pregnant women who smoke, abuse drugs or alcohol, or regularly take aspirin may have increased requirements for vitamin C. If an individual has adequate C status, supplementation with vitamin C does not enhance performance. However, strenuous and prolonged exercise has been shown to increase the need for vitamin C, physical performance can be compromised with marginal vitamin C status or deficiency. Athletes who participate in habitual prolonged, strenuous exercise should consume 100–1,000 mg of vitamin C [45]. Both of these values are greater than the established DRIs [43].

The major food sources of dietary vitamin C in the USA are citrus fruit juice, citrus fruits, fruitades, potatoes, tomatoes, and other vegetables. The dietary intake of vitamin C in the USA is less than in Europe. The difference can be explained by the fact that fruit and vegetable intake in Europe on average is higher compared with in the USA [36].

### 32.5.5 Vitamin E

Because endurance exercise results in increased oxygen consumption and thus increased oxidative stress, it seems logical that vitamin E supplementation might be beneficial for people who exercise. Although vitamin E has been shown to sequester free radicals in exercising individuals (by decreasing membrane disruption) [46], there have been no reports that vitamin E actually improves exercise performance. Nonetheless, vitamin E's role in the prevention of oxidative damage due to exercise may be noteworthy. However, more long-term research is needed to make solid claims about the role that vitamin E plays in decreasing oxidative stress.

### 32.5.6 Calcium

Presently, the DRIs for calcium for adult women is 1,200 mg/day calcium but there have been suggestions that daily intakes of 1,500 mg/day may by appropriate for postmenopausal women or women over age 65 [35].

Calcium recommendations may be achieved by consuming recommended levels of fat-free or low-fat milk and milk products and/or consuming alternative calcium sources [21].

Individuals should avoid calcium supplements containing bone meal, oyster shell, and shark cartilage due to the increased lead content in these supplements, which can result in toxic effects in the body.

Calcium supplements are best absorbed if taken in 500 mg or less between meals. For older women, calcium citrate is the best supplement [40, 47]. It is advisable for any calcium intake to

be accompanied by Vitamin D supplementation in order to increase absorption rates [43], especially in postmenopausal women. Fortunately, Vitamin D levels can also be improved from moderate skin sun exposure, since it is produce from cholesterol molecules in skin cells.

### 32.5.7 Iron

Studies show that iron status is often marginal or inadequate in many individuals, particularly females, who engage in regular, intense physical activity. The requirement of these individuals may be as much as 30–70 % greater than those who do not participate in regular strenuous exercise.

There are two forms of dietary iron: heme and nonheme. Heme iron is derived from hemoglobin, the protein in red blood cells that delivers oxygen to cells. Heme iron is found in animal foods that originally contained hemoglobin, such as red meats, fish, and poultry. Iron in plant foods such as lentils and beans is arranged in a chemical structure called nonheme iron. This is the form of iron added to iron-enriched and iron-fortified foods. Heme iron is absorbed better than nonheme iron, but most dietary iron is nonheme iron.

Plant-based foods, such as vegetables, fruits, whole-grain breads, or whole-grain pasta contain 0.1–1.4 mg of nonheme iron per serving. Fortified products, including breads, cereals, and breakfast bars can contribute high amounts of nonheme iron to the diet [43]. Therefore, exercising women should consume high levels of food containing both heme and nonheme iron. Consuming iron-enriched and iron-fortified foods as well as supplementation may be necessary to achieve the level required for highly active individuals.

## 32.6 Concluding Remarks

A food-based approach is ideal for meeting macronutrient and micronutrients needs of women. However, with food-based approaches only, women are not attaining the intake level needed for optimal health and performance in the USA and worldwide [48]. Consideration must be given to fortified foods and/or supplements to meet the recommended daily allowances for optimal health and performance. Among the micronutrients, the significant ones that may be associated with deficiencies in the older women include vitamin B-12, vitamin A, vitamin C, calcium, iron, zinc, and other trace minerals [35].

## Appendix 1: Dietary Reference Intakes (DRIs)

The Food and Nutrition Information Center (FNIC) is a leader in online global nutrition information. Located at the National Agricultural Library (NAL) of USDA, the FNIC Web site contains over 2,500 links to current and reliable nutrition information.

FNIC provides links to the DRI Tables, developed by the Institute of Medicine's Food and Nutrition Board. To view these tables or download these tables in a PDF file, please go to: http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes/dri-tables

**Dietary Reference Intakes: Recommended Intakes for Individuals**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

Comprehensive DRI tables for vitamins, minerals, and macronutrients; organized by age and gender. Includes the 2010 updated recommendations for calcium and vitamin D.

**Dietary Reference Intakes: RDA and AI for Vitamins and Elements (PDF | 28 kB)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

DRI tables for recommended dietary allowances (RDA) and adequate intakes (AI) of vitamins and elements, including the 2010 updated recommendations for calcium and vitamin D.

**Dietary Reference Intakes: UL for Vitamins and Elements (PDF | 19 kB)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

DRI table for tolerable upper intake levels (UL) of vitamins and elements, including the

2010 updated recommendations for calcium and vitamin D.

**Dietary Reference Intakes: Macronutrients**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

DRI table for carbohydrate, fiber, fat, fatty acids, and protein.

**Dietary Reference Intakes: Estimated Average Requirements (PDF | 15 kB)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

DRI table for nutrients that have an estimated average requirement (EAR), the average daily nutrient intake level estimated to meet the requirements of half of the healthy individuals in a group.

**Dietary Reference Intakes: Electrolytes and Water**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

DRI table for sodium, chloride, potassium, inorganic sulfate, and water.

Note: You can access these tables at: http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes/dri-tables.

## Appendix 2: Dietary Reports

The Food and Nutrition Information Center (FNIC) is a leader in online global nutrition information. Located at the National Agricultural Library (NAL) of USDA, the FNIC Web site contains over 2,500 links to current and reliable nutrition information.

FNIC provides links and PDF downloads to the DRI reports, developed by the Institute of Medicine's Food and Nutrition Board. To distribute or reprint these reports, please visit The National Academies Press Web site to secure all necessary permissions.

http://fnic.nal.usda.gov/dietary-guidance/dietary-reference-intakes/dri-reports

UPDATED**—Dietary Reference Intakes for Calcium and Vitamin D (2010) (PDF | 355 kB)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

Report brief on new DRIs for calcium and vitamin D, revised in November 2010. Read the pre-publication report at the National Academies Press Web site.

**Dietary Reference Intakes: The Essential Guide to Nutrient Requirements**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

All eight volumes of the DRIs are summarized in one reference volume, organized by nutrient, which reviews function in the body, food sources, usual dietary intakes, and effects of deficiencies and excessive intakes.

**Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride (1997)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

For the 2010 updated recommendations for calcium and vitamin D, refer to the pre-publication report at the National Academies Press Web site.

**Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients) (2005)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline (1998)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc (2001)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes: Proposed Definition of Dietary Fiber (2001)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids (2000)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate (2004)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes: Guiding Principles for Nutrition Labeling and Fortification (2003)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes: Applications in Dietary Planning (2003)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes: Applications in Dietary Assessment (2000)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes Research Synthesis Workshop Summary (2006)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes: Proposed Definition and Plan for Review of Dietary Antioxidants and Related Compounds (1998)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

**Dietary Reference Intakes: A Risk Assessment Model for Establishing Upper Intake Levels for Nutrients (1998)**

*National Academy of Sciences. Institute of Medicine. Food and Nutrition Board.*

## Appendix 3: USDA Food Patterns—Food Groups and Subgroups

| Food group | Subgroups and examples |
|---|---|
| Vegetables | *Dark-green vegetables*: All fresh, frozen, and canned dark-green leafy vegetables and broccoli, cooked or raw: for example, broccoli; spinach; romaine; collard, turnip, and mustard greens. |
| | *Red and orange vegetables*: All fresh, frozen, and canned red and orange vegetables, cooked or raw: for example, tomatoes, red peppers, carrots, sweet potatoes, winter squash, and pumpkin. |
| | *Beans and peas*: All cooked and canned beans and peas: for example, kidney beans, lentils, chickpeas, and pinto beans. Does not include green beans or green peas. (See additional comment under protein foods group.) |
| | *Starchy vegetables*: All fresh, frozen, and canned starchy vegetables: for example, white potatoes, corn, and green peas. |
| | *Other vegetables*: All fresh, frozen, and canned other vegetables, cooked or raw: for example, iceberg lettuce, green beans, and onions. |
| Fruits | All fresh, frozen, canned, and dried fruits and fruit juices: for example, oranges and orange juice, apples and apple juice, bananas, grapes, melons, berries, and raisins. |
| Grains | *Whole grains*: All whole-grain products and whole grains used as ingredients: for example, whole-wheat bread, whole-grain cereals and crackers, oatmeal, and brown rice. |
| | *Enriched grains*: All enriched refined-grain products and enriched refined grains used as ingredients: for example, white breads, enriched grain cereals and crackers, enriched pasta, and white rice. |
| Dairy products | All milks, including lactose-free and lactose-reduced products and fortified soy beverages; yogurts; frozen yogurts; dairy desserts; and cheeses. Most choices should be fat-free or low-fat. Cream, sour cream, and cream cheese are not included due to their low calcium content. |
| Protein foods | All meat, poultry, seafood, eggs, nuts, seeds, and processed soy products. Meat and poultry should be lean or low-fat. Beans and peas are considered part of this group, as well as the vegetable group, but should be counted in one group only. |

*Source*: USDA and HHS, US Department of Agriculture and US Department of Health and Human Services. Dietary Guidelines for Americans, 2010. Washington, DC. Complete report can be viewed and downloaded at http://www.cnpp.usda.gov/

## Appendix 4: USDA Foods Patterns

For each food group or subgroup[a], recommended average daily intake amounts[b] at all calorie levels. Recommended intakes from vegetable and protein foods subgroups are per week.

| Calorie level of pattern[c] | 1,000 | 1,200 | 1,400 | 1,600 | 1,800 | 2,000 | 2,200 | 2,400 | 2,600 | 2,800 | 3,000 | 3,200 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Fruits | 1 c | 1 c | 1½ c | 1½ c | 1½ c | 2 c | 2 c | 2 c | 2 c | 2½ c | 2½ c | 2½ c |
| Vegetables[d] | 1 c | 1½ c | 1½ c | 2 c | 2½ c | 2½ c | 3 c | 3 c | 3½ c | 3½ c | 4 c | 4 c |
| Dark-green vegetables | ½ c/week | 1 c/week | 1 c/week | 1½ c/week | 1½ c/week | 1½ c/week | 2 c/week | 2 c/week | 2½ c/week | 2½ c/week | 2½ c/week | 2½ c/week |
| Red and orange vegetables | 2½ c/week | 3 c/week | 3 c/week | 4 c/week | 5½ c/week | 5½ c/week | 6 c/week | 6 c/week | 7 c/week | 7 c/week | 7½ c/week | 7½ c/week |
| Beans and peas (legumes) | ½ c/week | ½ c/week | ½ c/week | 1 c/week | 1½ c/week | 1½ c/week | 2 c/week | 2 c/week | 2½ c/week | 2½ c/week | 3 c/week | 3 c/week |
| Starchy vegetables | 2 c/week | 3½ c/week | 3½ c/week | 4 c/week | 5 c/week | 5 c/week | 6 c/week | 6 c/week | 7 c/week | 7 c/week | 8 c/week | 8 c/week |
| Other vegetables | 1½ c/week | 2½ c/week | 2½ c/week | 3½ c/week | 4 c/week | 4 c/week | 5 c/week | 5 c/week | 5½ c/week | 5½ c/week | 7 c/week | 7 c/week |
| Grains[e] | 3 oz-eq | 4 oz-eq | 5 oz-eq | 5 oz-eq | 6 oz-eq | 6 oz-eq | 7 oz-eq | 8 oz-eq | 9 oz-eq | 10 oz-eq | 10 oz-eq | 10 oz-eq |
| Whole grains | 1½ oz-eq | 2 oz-eq | 2½ oz-eq | 3 oz-eq | 3 oz-eq | 3 oz-eq | 3½ oz-eq | 4 oz-eq | 4½ oz-eq | 5 oz-eq | 5 oz-eq | 5 oz-eq |
| Enriched grains | 1½ oz-eq | 2 oz-eq | 2½ oz-eq | 2 oz-eq | 3 oz-eq | 3 oz-eq | 3½ oz-eq | 4 oz-eq | 4½ oz-eq | 5 oz-eq | 5 oz-eq | 5 oz-eq |
| Protein foods[d] | 2 oz-eq | 3 oz-eq | 4 oz-eq | 5 oz-eq | 5 oz-eq | 5½ oz-eq | 6 oz-eq | 6½ oz-eq | 6½ oz-eq | 7 oz-eq | 7 oz-eq | 7 oz-eq |
| Seafood | 3 oz/week | 5 oz/week | 6 oz/week | 8 oz/week | 8 oz/week | 8 oz/week | 9 oz/week | 10 oz/week | 10 oz/week | 11 oz/week | 11 oz/week | 11 oz/week |
| Meat, poultry, eggs | 10 oz/week | 14 oz/week | 19 oz/week | 24 oz/week | 24 oz/week | 26 oz/week | 29 oz/week | 31 oz/week | 31 oz/week | 34 oz/week | 34 oz/week | 34 oz/week |

(continued)

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Nuts, seeds, soy products | 1 oz/week | 2 oz/week | 3 oz/week | 4 oz/week | 4 oz/week | 4 oz/week | 4 oz/week | 5 oz/week | 5 oz/week | 5 oz/week | 5 oz/week | 5 oz/week |
| Dairy[f] | 2 c | 2½ c | 2½ c | 3 c | 3 c | 3 c | 3 c | 3 c | 3 c | 3 c | 3 c | 3 c |
| Oils[g] | 15 g | 17 g | 17 g | 22 g | 24 g | 27 g | 29 g | 31 g | 34 g | 36 g | 44 g | 51 g |
| Maximums of as[h] limit, calories (% of calories) | 137 (14 %) | 121 (10 %) | 121 (9 %) | 121 (8 %) | 161 (9 %) | 258 (13 %) | 266 (12 %) | 330 (14 %) | 362 (14 %) | 395 (14 %) | 459 (15 %) | 596 (19 %) |

*Source*: USDA and HHS, US Department of Agriculture and US Department of Health and Human Services. Dietary Guidelines for Americans, 2010. Washington, DC. Complete report can be viewed and downloaded at http://www.cnpp.usda.gov/

[a]All foods are assumed to be in nutrient-dense forms, lean or low-fat, and prepared without added fats, sugars, or salt. Solid fats and added sugar may be included up to the daily maximum limit identified in the table. For food items in each group and subgroup see Appendix 3

[b]Food group amounts are shown in cup (c) or ounce-equivalents (oz-eq). Oils are shown in grams (g). Quantity equivalents for each food group are:

*Grains*, 1 oz-equivalent is: 1-oz slice bread; 1 oz uncooked pasta or rice; ½ cup cooked rice, pasta, or cereal; 1 tortilla (6″ diameter); 1 pancake (5″ diameter); 1 oz ready-to-eat cereal (about 1 cup cereal flakes)

*Vegetables and fruits*, 1 cup equivalent is: 1 cup raw or cooked vegetable or fruit; ½ cup dried vegetable or fruit; 1 cup vegetable or fruit juice; 2 cups leafy salad greens

*Protein foods*, 1 oz-equivalent is: 1 oz lean meat, poultry, seafood; 1 egg; 1 Tbsp peanut butter; ½ oz nuts or seeds. Also, ¼ cup cooked beans or peas may also be counted as 1 oz-equivalent

*Dairy*, 1 cup equivalent is: 1 cup milk, fortified soy beverage, or yogurt; 1½ oz natural cheese (e.g., cheddar); 2 oz of processed cheese (e.g., American)

[c]Estimated calorie needs per day by age, gender, and physical activity level. Patterns from 1,600 to 3,200 cal meet the nutritional needs of children ages 9 years and older and adults

[d]Vegetable and protein foods subgroup amounts are shown in this table as weekly amounts, because it would be difficult for consumers to select foods from all subgroups daily

[e]Whole-grain subgroup amounts shown in this table are minimums. More whole grains up to all of the grains recommended may be selected, with offsetting decreases in the amounts of enriched refined grains

[f]The amount of dairy foods in the 1,200 and 1,400 cal patterns have increased to reflect new RDAs for calcium that are higher than previous recommendations for children ages 4–8 years

[g]Oils and soft margarines include vegetable, nut, and fish oils and soft vegetable oil table spreads that have no *trans* fats

[h]SoFAS are calories from solid fats and added sugars. The limit for SoFAS is the remaining amount of calories in each food pattern after selecting the specified amounts in each food group in nutrient-dense forms (forms that are fat-free or low-fat and with no added sugars). The number of SoFAS is lower in the 1,200, 1,400, and 1,600 cal patterns than in the 1,000 cal pattern. The nutrient goals for the 1,200–1,600 cal patterns are higher and require that more calories be used for nutrient-dense foods from the food groups

## References


1. WHO. Keep fit for life Meeting the nutritional needs of older persons. Geneva: WHO; 2002. p. 76–121.
2. Visvanathan R, Chapman IM. Undernutrition and anorexia in the older person. Gastroenterol Clin North Am. 2009;38:393–409.
3. St-Onge MP, Gallagher D. Body composition changes with aging: the cause or the result of alterations in metabolic rate and macronutrient oxidation? Nutrition. 2010;26:152–5.
4. Wu BN, O'Sullivan AJ. Sex differences in energy metabolism need to be considered with lifestyle modifications in humans. J Nutr Metab. 2011;2011:391809.
5. A Report of the panel on macronutrients, subcommittees on upper reference levels of nutrients and interpretation and uses of dietary reference intakes, standing committee on the scientific evaluation of dietary reference intakes. Front matter. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (macronutrients). Washington, DC: The National Academies Press; 2005.
6. Manini TM, Everhart JE, Anton SD, et al. Activity energy expenditure and change in body composition in late life. Am J Clin Nutr. 2009;90:1336–42.
7. Manini TM, Everhart JE, Patel KV, et al. Activity energy expenditure and mobility limitation in older adults: differential associations by sex. Am J Epidemiol. 2009;169:1507–16.
8. Luhrmann PM, Bender R, Edelmann-Schafer B, Neuhauser-Berthold M. Longitudinal changes in energy expenditure in an elderly German population: a 12-year follow-up. Eur J Clin Nutr. 2009;63:986–92.
9. Tooze JA, Schoeller DA, Subar AF, Kipnis V, Schatzkin A, Troiano RP. Total daily energy expenditure among middle-aged men and women: the OPEN Study. Am J Clin Nutr. 2007;86:382–7.
10. Manini TM. Energy expenditure and aging. Ageing Res Rev. 2010;9:1–11.
11. Roberts SB, Dallal GE. Energy requirements and aging. Public Health Nutr. 2005;8:1028–36.
12. Hughes VA, Roubenoff R, Wood M, Frontera WR, Evans WJ, Fiatarone Singh MA. Anthropometric assessment of 10-y changes in body composition in the elderly. Am J Clin Nutr. 2004;80:475–82.
13. Krems C, Luhrmann PM, Strassburg A, Hartmann B, Neuhauser-Berthold M. Lower resting metabolic rate in the elderly may not be entirely due to changes in body composition. Eur J Clin Nutr. 2005;59:255–62.
14. Manini TM, Everhart JE, Patel KV, et al. Daily activity energy expenditure and mortality among older adults. JAMA. 2006;296:171–9.
15. Frankenfield D, Roth-Yousey L, Compher C. Comparison of predictive equations for resting metabolic rate in healthy nonobese and obese adults: a systematic review. J Am Diet Assoc. 2005;105:775–89.
16. Mifflin MD, St Jeor ST, Hill LA, Scott BJ, Daugherty SA, Koh YO. A new predictive equation for resting energy expenditure in healthy individuals. Am J Clin Nutr. 1990;51:241–7.
17. Weijs PJ, Kruizenga HM, van Dijk AE, et al. Validation of predictive equations for resting energy expenditure in adult outpatients and inpatients. Clin Nutr. 2008;27:150–7.
18. Blanc S, Schoeller DA, Bauer D, et al. Energy requirements in the eighth decade of life. Am J Clin Nutr. 2004;79:303–10.
19. Britten P, Marcoe K, Yamini S, Davis C. Development of food intake patterns for the MyPyramid food guidance system. J Nutr Educ Behav. 2006;38:S78–92.
20. Shetty P. Energy requirements of adults. Public Health Nutr. 2005;8:994–1009.
21. McGuire S. U.S. Department of Agriculture and U.S. Department of Health and Human Services, Dietary Guidelines for Americans, 2010. 7th Edition, Washington, DC: U.S. Government Printing Office, January 2011. Adv Nutr. 2011;2:293–4.
22. USDA, United States Department of Agriculture. Diet quality of older Americans in 1994-96 and 2001-02 as measured by the healthy eating index-2005. 2008.
23. USDA, United Stated Department of Agriculture. USDA and HHS announce new dietary guidelines to help Americans make healthier food choices and confront obesity epidemic. Washington, DC: USDA; 2011.
24. FAO/WHO/UNU. Human energy requirements. Report of a joint FAO/WHO/UNU expert consultation. Rome: FAO; 2004.
25. USDA, United Stated Department of Agriculture. Dietary energy density and body weight: a review of the evidence. Washington, Dc: USDA; 2012.
26. Senechal M, Arguin H, Bouchard DR, et al. Weight gain since menopause and its associations with weight loss maintenance in obese postmenopausal women. Clin Interv Aging. 2011;6:221–5.
27. ODPHP, Office of disease prevention and health promotion. Eat Healthy Be Active Community Workshops. 2010.
28. American College of Sports Medicine, Chodzko-Zajko WJ, Proctor DN, et al. American College of Sports Medicine position stand. Exercise and physical activity for older adults. Med Sci Sports Exerc. 2009;41:1510–30.
29. Campbell WW, Johnson CA, McCabe GP, Carnell NS. Dietary protein requirements of younger and older adults. Am J Clin Nutr. 2008;88:1322–9.
30. McGrane MM, Essery E, Obbagy J, et al. Dairy consumption, blood pressure, and risk of hypertension: an evidence-based review of recent literature. Curr Cardiovasc Risk Rep. 2011;5:287–98.
31. Welsh JA, Sharma A, Abramson JL, Vaccarino V, Gillespie C, Vos MB. Caloric sweetener consumption and dyslipidemia among US adults. JAMA. 2010;303:1490–7.
32. Brown IJ, Stamler J, Van Horn L, et al. Sugar-sweetened beverage, sugar intake of individuals, and

their blood pressure: international study of macro/micronutrients and blood pressure. Hypertension. 2011;57:695–701.
33. Racine E, Troyer JL, Warren-Findlow J, McAuley WJ. The effect of medical nutrition therapy on changes in dietary knowledge and DASH diet adherence in older adults with cardiovascular disease. J Nutr Health Aging. 2011;15:868–76.
34. Rosenbloom CA, Dunaway A. Nutrition recommendations for Masters Athletes. Clin Sports Med. 2007;26:91–100.
35. Chernoff R. Micronutrient requirements in older women. Am J Clin Nutr. 2005;81:1240S–5.
36. Chun OK, Floegel A, Chung SJ, Chung CE, Song WO, Koo SI. Estimation of antioxidant intakes from diet and supplements in U.S. adults. J Nutr. 2010;140:317–24.
37. Alipanah N, Varadhan R, Sun K, Ferrucci L, Fried LP, Semba RD. Low serum carotenoids are associated with a decline in walking speed in older women. J Nutr Health Aging. 2009;13:170–5.
38. Dunn-Lewis C, Kraemer WJ, Kupchak BR, et al. A multi-nutrient supplement reduced markers of inflammation and improved physical performance in active individuals of middle to older age: a randomized, double-blind, placebo-controlled study. Nutr J. 2011;10:90.
39. Stover PJ. Physiology of folate and vitamin B12 in health and disease. Nutr Rev. 2004;62:S3–12. discussion S13.
40. Kim YI. Role of folate in colon cancer development and progression. J Nutr. 2003;133:3731S–9.
41. Leishear K, Boudreau RM, Studenski SA, et al. Relationship between vitamin B12 and sensory and motor peripheral nerve function in older adults. J Am Geriatr Soc. 2012;60:1057–63.
42. Nicklas TA. Nutrient profiling: the new environment. J Am Coll Nutr. 2009;28:416S–20.
43. Jennifer J. Otten, Jennifer Pitzi Hellwig, Linda D. Meyers. National Research Council. Dietary reference intakes: the essential guide to nutrient requirements. Washington, DC: The National Academies Press, 2006; Available from: http://www.nap.edu/catalog/11537.html
44. Manore MM. Effect of physical activity on thiamine, riboflavin, and vitamin B-6 requirements. Am J Clin Nutr. 2000;72:598S–606.
45. American Dietetic A, Dietitians of C, American College of Sports M, Rodriguez NR, Di Marco NM, Langley S. American College of Sports Medicine position stand. Nutrition and athletic performance. Med Sci Sports Exerc. 2009;41:709–31.
46. McBride JM, Kraemer WJ, Triplett-McBride T, Sebastianelli W. Effect of resistance exercise on free radical production. Med Sci Sports Exerc. 1998;30:67–72.
47. Dawson-Hughes B, Dallal GE, Krall EA, Sadowski L, Sahyoun N, Tannenbaum S. A controlled trial of the effect of calcium supplementation on bone density in postmenopausal women. N Engl J Med. 1990;323:878–83.
48. Bartley KA, Underwood BA, Deckelbaum RJ. A life cycle micronutrient perspective for women's health. Am J Clin Nutr. 2005;81:1188S–93.

# Questions

## Chapter 1 Questions

1. A disruption within the evaluative component of body image is usually referred to as
   (a) Body dissatisfaction*
   (b) Body dysphoria
   (c) Body image disorder
   (d) Body image distortion

2. According to the Tripartite Influence Model, which of the following mediate(s) the relationships between sociocultural influences and body image and eating concerns?
   (a) Appearance comparison
   (b) Internalization of the thin idea
   (c) Self-esteem
   (d) Both a and b*

3. Which authors stated that girls' identification with media models makes them particularly vulnerable to experiencing negative body image?
   (a) Bell and Dittmar*
   (b) Dohnt and Tiggeman
   (c) Jones and Crawford
   (d) Thompson and Stice

4. Researchers have shown that the psychological processes related to body dissatisfaction are already well established by the age of
   (a) 9*
   (b) 11
   (c) 12
   (d) 13

5. Warning signs of a possible eating disorder that may be seen in individuals with type 1 diabetes include all of the following *except*
   (a) Atypical exercise habits
   (b) Unexplained decreases in hemoglobin A1c*
   (c) Recurring issues with diabetic ketoacidosis
   (d) Symptoms of depressed or anxious mood

6. All of the following factors appear to contribute to different body weight ideals across the globe *except*
   (a) Degree of Westernization influence
   (b) Marital status*
   (c) Socioeconomic status
   (d) The role of women in society

7. Which researcher(s) outlined tips for enhancing body image assessment in clinical and research settings?
   (a) Cash and colleagues
   (b) Gardner and colleagues
   (c) Neumark-Sztainer
   (d) Thompson*

8. Which type of prevention program is designed to target high-risk individuals who may be exhibiting early signs of disordered eating?
   (a) Indicated*
   (b) Integrated
   (c) Selective
   (d) Universal

J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2, © Springer Science+Business Media New York 2014

9. The interaction of females, dieting behaviors, and sociocultural influences promoting the thin ideal refers to the theoretical concept of
   (a) Observational learning
   (b) Outcome expectations
   (c) Reciprocal determinism*
   (d) Self-regulation
10. All of the following are elements within the social marketing mix *except*:
   (a) Place
   (b) Price
   (c) Product
   (d) Program*

## Chapter 2 Questions

1. What is the average age of menarche in the United States?
   (a) 8 years
   (b) 10.5 years
   (c) 12.5 years*
   (d) 15 years
2. What was the legislative act that prevents sex discrimination in any educational program or activity in institutions from elementary school through college that receives federal funding?
   (a) Equal rights amendment
   (b) Title IX act*
   (c) Civil rights act
   (d) Second amendment to the constitution
3. The hormone from the hypothalamus that stimulates pituitary LH release is
   (a) Estrogen
   (b) ACTH
   (c) Progesterone
   (d) GnRH*
4. The hormones primarily responsible for breast development at puberty in girls are
   (a) Estrogen and progesterone*
   (b) LH and FSH
   (c) Cortisol and thyroxine
   (d) GnRH and TRH
5. Menopause is caused by
   (a) Decreased GnRH release from the brain
   (b) Ovary depleted of follicles*
   (c) Pituitary malfunction
   (d) Lack of responsiveness of the uterus
6. The average age at menopause is
   (a) 13 years
   (b) 25 years
   (c) 51 years*
   (d) 82 years
7. The growth of axillary and pubic hair is stimulated by
   (a) Estrogen
   (b) Progesterone
   (c) FSH
   (d) Androgen*
8. The most debilitating effect of low estrogen levels after menopause is
   (a) Alzheimer's disease
   (b) Cardiovascular disease
   (c) Osteoporosis*
   (d) Stroke
9. One of the primary benefits of estrogen replacement therapy after menopause is
   (a) Continued menstruation
   (b) Protection against bone loss*
   (c) Protection against stroke
   (d) Continued breast development
10. At puberty there is a growth spurt in both males and females after which growth ceases because the epiphyseal plate in long bones fuses. The agent responsible for the fusion of the epiphyseal plate is
   (a) Estrogen*
   (b) Growth hormone
   (c) Calcitonin
   (d) Progesterone

## Chapter 3 Questions

1. Which two bones appear to be the best predictors of sex?
   (a) Calcaneus and talus
   (b) Femur and tibia*
   (c) Humerus and pelvis
   (d) Pelvis and femur

2. In general, women have which one of the following cartilage characteristics compared to men?
   (a) Greater cartilage surface area
   (b) Greater cartilage thickness
   (c) Less cartilage degradation in older age
   (d) Slower cartilage accrual rate in youth*

3. In general, women have which one of the following bone characteristics compared to men?
   (a) Faster bone turnover in adulthood
   (b) Greater compressive and bending strength
   (c) Less peak bone mass in adulthood*
   (d) Less risk of bone fracture

4. All of the following are considered predilection sites for a woman's tendinosis of the lateral elbow extensor group, EXCEPT FOR
   (a) Brachioradialis insertion at the supracondylar ridge
   (b) Extensor carpi radialis brevis insertion at the lateral epicondyle
   (c) Extensor carpi ulnaris insertion at the lateral epicondyle*
   (d) Extensor digitorum communis insertion at the lateral epicondyle

5. Which of the following characterizes a woman's distinctive clinical symptom profile associated with carpal tunnel syndrome?
   (a) Autonomic nervous system disturbances
   (b) Decreased inter-tunnel pressure from movement*
   (c) Median nerve motor amplitude test outcomes
   (d) Median nerve sensory latency test outcomes

6. Compared to men, which one of the following appears to increase the woman's risk for macrotraumatic fracture at the hip?
   (a) Increased estrogen production
   (b) Decreased femoral neck strength*
   (c) Deeper acetabular width
   (d) Narrower coxadiaphyseal angle

7. Compared to men, which one of the following appears to specifically increase the woman's risk for labral afflictions at the hip?
   (a) Decreased femoral neck thickening
   (b) Decreased labral sensory nerve supply
   (c) Decreased labral tissue tensile strength*
   (d) Decreased coxadiaphyseal (CD) angulation

8. Which one of the following factors is LEAST likely going to predispose women to a symptom-producing condition at the cervical spine?
   (a) Cartilage coverage of the cervical facet articular surfaces
   (b) Segmental anterior-posterior translation during whiplash
   (c) Spinal canal narrowing that occurs with the whiplash event
   (d) Vertebral canal-to-body diameter ratio changes*

9. Which one of the following influences the development and progression of adolescent idiopathic scoliosis?
   (a) Changes in the premenarchal kyphosis and lordosis
   (b) Curve magnitude measured after 15 years of age
   (c) Increased premenarchal skeletal growth rate*
   (d) Leg-length discrepancy measured after 15 years of age

10. Compared to men, which one of the following sacroiliac joint characteristics is more commonly expected in women?
    (a) Equal thickness found in the sacral cartilage
    (b) Increased incidence of periarticular osteophytes
    (c) Increased pliability of the periarticular soft tissues*
    (d) Reduced joint mobility by the fifth decade in life

## Chapter 4 Questions

1. The phase of the cycle when the lining of the uterus is growing is
   (a) Menstrual
   (b) Luteal
   (c) Follicular*
   (d) Secretory

2. The hormone that causes ovulation is
   (a) FSH
   (b) LH*
   (c) Prolactin
   (d) GnRH

3. Hormonal birth control pills work on the basis of what relationship between the ovarian and pituitary hormones?
   (a) Negative feedback*
   (b) Positive feedback

4. What hormone prepares the lining of the uterus for implantation of the early embryo?
   (a) LH
   (b) FSH
   (c) GnRH
   (d) Progesterone*

5. The most common cause of amenorrhea in women of child-bearing age is
   (a) Nursing
   (b) Low energy availability
   (c) Pregnancy*
   (d) Psychological stress

6. How many follicles normally mature and ovulate in the human?
   (a) One*
   (b) Two
   (c) Ten
   (d) Many

7. Which of the following are steroid hormones?
   (a) FSH and LH
   (b) Inhibin and activin
   (c) GnRH and prolactin
   (d) Estrogen and progesterone*

8. The hormone progesterone is primarily produced by the
   (a) Corpus luteum*
   (b) Follicle
   (c) Pituitary
   (d) Hypothalamus

9. The hormone measured to determine pregnancy is
   (a) Estrogen
   (b) Progesterone
   (c) hCG*
   (d) GnRH

10. The cell type that produces GnRH is
    (a) Granulosa
    (b) Luteal
    (c) Neurons*
    (d) Gonadotrophs

## Chapter 5 Questions

1. The term used to describe regular menstrual cycles of 25–38 days is
   (a) Amenorrhea
   (b) Oligomenorrhea
   (c) Eumenorrhea*
   (d) Dysmenorrheal

2. Primary amenorrhea is when a young woman
   (a) Has her first menstrual period
   (b) Has not had her first menstrual period by age 16*
   (c) Is infertile

3. Which of the following conditions can cause menstrual cycle disturbances?
   (a) PCOS
   (b) Hyperprolactinemia
   (c) Crohn's disease
   (d) All of the above*

4. The female athlete triad is a recognized set of symptoms first reported in
   (a) 1944
   (b) 1972
   (c) 1993*
   (d) 2002

5. The physiological deficit in functional hypothalamic amenorrhea (FHA) is
   (a) Decreased GnRH release from the hypothalamus*
   (b) Ovarian failure
   (c) Hyperprolactinemia
   (d) High LH and FSH levels

6. The female athlete triad includes which of the following three symptoms?
   (a) Menstrual irregularity, osteopenia, obesity
   (b) Menstrual irregularity, disordered eating, bulimia

(c) Menstrual irregularity, osteopenia, galactorrhea
(d) Menstrual irregularity, osteopenia, disordered eating*

7. Menstrual cycles can be disturbed by many internal and external factors. The factor thought to be the primary influence on cycles in female athletes is
(a) Too much exercise
(b) Low energy availability*
(c) Birth control pills
(d) Pituitary tumors

8. One of the obvious signs of polycystic ovarian disease (PCOS) is
(a) Blurred vision
(b) Headaches
(c) Increased hair growth on face*
(d) Normal menstrual cycles

9. A major health concern for young women experiencing prolonged amenorrhea is
(a) Premature osteoporosis*
(b) Hot flashes
(c) Poor athletic performance
(d) Early menopause

10. Eating disorders have their highest prevalence during
(a) Childhood
(b) The postmenopausal period
(c) The adolescent years*
(d) Fertile adult years

## Chapter 6 Questions

1. Which of the following are thought to inhibit reproductive function?
(a) Psychological stress
(b) Nutritional stress
(c) Physical stress
(d) All of the above*

2. Suppressed LH secretion can sometime be reversed with naloxone. What substance does this finding implicate in the inhibition of LH release?
(a) Dopamine
(b) Estrogen
(c) Endogenous opiates*
(d) GnRH

3. The individual that first recognized that stress might impair reproductive function was
(a) Seyle*
(b) Refeinstein
(c) Ferin
(d) Loucks

4. The long-term consequences of FHA include
(a) Pregnancy
(b) Osteoporosis*
(c) Menopause
(d) Normal bone density

5. The most effective treatment of the underlying cause of FHA would be
(a) Hormonal therapy
(b) Cognitive behavioral therapy*
(c) Chemotherapy
(d) Radiation therapy

6. The diagnosis of FHA requires
(a) Exclusion of all other causes of amenorrhea*
(b) Significant weight loss
(c) Disturbed eating
(d) Low gonadotropin levels

7. The primary deficit that causes FHA is the
(a) Direct suppression of ovarian function
(b) Suppression of GnRH release from the hypothalamus*
(c) Elevated cortisol level
(d) Pregnancy

8. In women who exercise excessively and become amenorrheic, the amenorrhea is most likely due to
(a) Exercise
(b) Psychological stress
(c) Low energy availability*
(d) Dysfunctional attitudes

9. Which of the following is a major stimulator of GnRH release?
(a) CRH
(b) β-endorphin
(c) Vasopressin
(d) Kisspeptin*

10. A peptide produced in the brain implicated in the suppression of GnRH release is
    (a) ACTH
    (b) Cortisol
    (c) CRH*
    (d) Ghrelin

## Chapter 7 Questions

1. What organs are involved with the female menstrual cycle?
    (a) Hypothalamus and pituitary
    (b) Uterus and ovaries
    (c) Breasts
    (d) a and b*
2. What are the phases of the menstrual cycle?
    (a) Follicular, ovulatory, and luteal
    (b) Proliferative and secretory
    (c) Proliferative, ovulatory, and secretory
    (d) a and c*
3. Which hormones play a role in bone development?
    (a) Estrogen and progesterone
    (b) GH and IGF-1
    (c) Vitamin D
    (d) All of the above*
4. What are the different components of bone?
    (a) Osteoblasts, osteoclasts, and elastin
    (b) Glycoproteins, collagen, and elastin
    (c) Osteoblasts, osteoclasts, glycoproteins, and collagens
    (d) None of the above*
5. What are the variable factors affecting peak bone mass?
    (a) Hereditary and endocrine
    (b) Mechanical and lifestyle
    (c) Physical activity and nutrition
    (d) a and b*
6. What are some of the risk factors contributing to dysmenorrhea in young females?
    (a) Excessive weight gain
    (b) Excessive exercise
    (c) Inadequate nutrition
    (d) b and c*
7. At what age peak bone is achieved?
    (a) 15–25 years old
    (b) 20–30 years old
    (c) 25–35 years old*
    (d) None of the above
8. Which sport positively affects the skeleton in terms of building bone the most?
    (a) Running
    (b) Gymnastics*
    (c) Swimming
    (d) a and b
9. What are the components of the female athlete triad?
    (a) Dysmenorrhea, eating disorder, osteopenia
    (b) Anemia, disordered eating, osteoporosis
    (c) Amenorrhea, eating disorder, osteopenia
    (d) Amenorrhea, disordered eating, osteoporosis*
10. What is the rate of bone loss in females with menstrual disorders?
    (a) 2 %*
    (b) 4 %
    (c) 3 %
    (d) 1 %

## Chapter 8 Questions

1. Athletes who desire pregnancy but are not ovulating can be treated with
    (a) Clomiphene citrate for ovulation induction
    (b) Pulsatile GnRH or injected gonadotropins
    (c) Endogenous opiates
    (d) Both a and b to induce ovulation*
2. Which of the following statements is *false* about oral contraceptive pills (OCP)?
    (a) The use of OCPs will not normalize the metabolic factors impairing bone function, health, and performance
    (b) OCPs are unlikely to fully reverse the low BMD
    (c) Estrogen replacement without nutritional rehabilitation will reverse bone loss*
    (d) All statements are true

3. Hormone therapy should not be used in adolescents younger than _____ years old.
   (a) 15
   (b) 16*
   (c) 17
   (d) 18

4. Which of the following statements are true with regard to appetite in the female athlete?
   (a) Appetite is an unreliable indicator of energy requirements*
   (b) Athletes should just eat when they are hungry and this will prevent low energy availability
   (c) Athletes should wait for hunger and then eat until satisfied in order to increase energy availability
   (d) All of the statements are true

5. Treating the cause of menstrual dysfunction can lead to ovulatory cycles within 12 months, but up to _____ of athletes may remain amenorrheic.
   (a) 50 %
   (b) 70 %
   (c) 40 %
   (d) 30 %*

6. _____ percent of bone mass accrual occurs in the _____ years surrounding menarche.
   (a) 45, 4
   (b) 35, 3
   (c) 30, 1
   (d) 25, 2*

7. In adolescent girls, about _____ of total body mineral content is accrued by 15½–18 years of age.
   (a) 60 %
   (b) 70 %
   (c) 80 %
   (d) 90 %*

8. Target groups for menstrual screening should include which group(s) of women?
   (a) A normal secondary sexual development but no menarche by 15 years of age
   (b) Failure of thelarche (breast development) by 13 years old
   (c) No menarche within 5 years after breast development that occurred less than 10 years old
   (d) All of the abovementioned groups should be targeted*

9. In 2006, National Collegiate Athletic Association Division I Schools adopted a standardized eating disorder and menstrual dysfunction screening tool to be used for all female athletes.
   (a) True
   (b) False*

10. Which of the following characteristic(s) is (are) true for an athlete with polycystic ovarian syndrome (PCOS)?
    (a) She will usually be at or above a normal body mass index (BMI)
    (b) She will likely be hirsute
    (c) She may show signs of insulin resistance
    (d) All of the above characteristics could be possible with PCOS*

## Chapter 9 Questions

1. The prevalence trades of eating disorders are higher
   (a) In adolescents
   (b) In the general young adult population
   (c) In males
   (d) In adolescents and young women*

2. Common characteristics that appear in people who develop eating disorders include
   (a) Difficulty in accepting themselves
   (b) Body dissatisfaction
   (c) Fear of losing control
   (d) All of the above*

3. The concept of "emotional eating" refers to
   (a) The difficulty in separating emotions from food
   (b) Confusion between hunger and satiety
   (c) The act of eating as a way to reach emotional satisfaction*
   (d) A psychological need to eat

4. Throughout the life cycle, "affection and feeding" develop in parallel because
   (a) It is the first communication relationship between the baby and the exterior (the feeder)
   (b) There is a close relationship between hunger and dissatisfaction
   (c) There is a relationship between affection and food
   (d) All of the above*
5. The concept of "alexithymia" is defined as
   (a) The inability to identify and express emotions*
   (b) The inability to relate to the surroundings
   (c) The inability to express oneself in an emotional manner
   (d) The inability to control the anxiety produced by the intake
6. The act of intake or the feeding process is produced by
   (a) Purely physiological needs
   (b) By organic and psychosocial needs*
   (c) The need to control external situations
   (d) The need to communicate/relate with the surrounding environment
7. Commonly known diagnostic profiles EDNOS are considered
   (a) Atypical disorders or incomplete symptomatology but high in frequency*
   (b) Atypical disorders or incomplete symptomatology but low in frequency
   (c) Atypical disorders of body image distortion
   (d) None of the above
8. "Bigorexia" is characterized by
   (a) A muscle dimorphic disorder
   (b) Development of a muscular body for fear of appearing weak
   (c) a and b*
   (d) None of the above
9. The new psychological intervention treatments work by
   (a) Focusing on positive psychology and resilience
   (b) Focusing on training the person
   (c) Focusing on developing emotional intelligence and communication
   (d) All of the above*
10. "Orthorexia" is characterized by
    (a) A muscle dimorphic disorder
    (b) Development of a muscular body for fear of appearing strong
    (c) An obsession for healthy food that is considered pathological*
    (d) All of the above

## Chapter 10 Questions

1. Warning signs of an eating disorder include obsessive thinking about all of the following except
   (a) Healthy diet*
   (b) Weight
   (c) Shape
   (d) Exercise
2. What percentages of girls and boys, respectively, engage in disordered eating behaviors?
   (a) 10 % of girls, 6 % of boys
   (b) 25 % of girls, 12 % of boys
   (c) 30 % of girls, 16 % of boys*
   (d) 32 % of girls, 18 % of boys
3. What determinant factor(s) affects (affect) healthy habits during childhood and adolescence?
   (a) School
   (b) Sociocultural groups
   (c) Family
   (d) All of the above*
4. Health literacy comprises all of the following competencies except
   (a) Effective communication
   (b) Self-directed learning
   (c) Critical thinking and problem solving
   (d) Healthy eating habits*
5. Who are considered the main teachers and socializing agents for a child's interaction with the larger environment?
   (a) Friends
   (b) Teachers

(c) Parents*
(d) None of the above

6. School-based prevention programs designed for middle school and high school students may be more effective if they _____
   (a) Include relational components
   (b) Are based on the lived experiences of the participants
   (c) Coincide with developmental issues
   (d) All of the above*

7. While peer groups are often considered risk factors for harmful health behaviors among children and adolescents, peer groups that model _____ can serve as crucial protective factors.
   (a) Obsessive dieting
   (b) Pro-social values*
   (c) Obsessive exercise
   (d) Body image disorders

8. For successful prevention of body image and eating disturbances among children, sociocultural change should include all of the following except
   (a) How females are portrayed in the media
   (b) Expectations regarding gender roles
   (c) Acceptance of a wide range of body weights and shapes
   (d) Promotion of pro-ana and pro-mia websites*

9. Which of the following is not one of the objectives of positive youth development programs?
   (a) To promote healthy bonding
   (b) To promote social, emotional, cognitive, behavioral, and moral competencies
   (c) To provide recognition for negative behavior*
   (d) To cultivate spirituality

10. Which of the following is not a prevention question that deserves future study?
    (a) Which protective factors should be targeted and enhanced to reduce the incidence of body image and eating disturbances among children and adolescents from different age and ethnic groups?
    (b) Which interventions are more effective—universal or targeted ones?
    (c) How can high-risk youth be reached, and what interventions are most effective with this group?
    (d) All of the above questions deserve future study*

## Chapter 11 Questions

1. Which of the following BEST describes an eating disorder?
   (a) General term that describes a variety of abnormal or atypical eating behaviors
   (b) A psychiatric condition that requires a physician's diagnosis*
   (c) Any condition in which one's eating behavior changes more than six times per year
   (d) Any behavior in which one attempts to reduce one's body weight below a healthy range

2. High neuroticism, perfectionism, obsessiveness, and low self-esteem describe the personality traits of _____
   (a) Anorexia nervosa
   (b) Obesity
   (c) Bulimia nervosa
   (d) Binge eating disorder
   (e) Both a and c*

3. Oftentimes, individuals with AN feel bloated and full because food can stay in the stomach for _____ hour (s) as opposed to the normal passage time of _____ hours (s).
   (a) 4–5, 1*
   (b) 6–7, 4–5
   (c) 2–3, 3–4
   (d) 1, 4–5

4. Which of the following increases one's risk of developing an eating disorder?
   (a) Having a sibling with an eating disorder*
   (b) Large family size
   (c) Holding the first order of birth
   (d) All of the above

5. A method of purging includes which of the following?
   (a) Excessive exercise
   (b) Regular use of laxatives
   (c) Vomiting
   (d) All of the above*

6. _____ is the most common medical cause of death in people with AN.
   (a) Peripheral neuropathy
   (b) Bradycardia
   (c) Osteoporosis
   (d) Heart disease*

7. Tooth decay, calluses on knuckles, and swelling in the cheek area are all symptoms of which eating disorder?
   (a) Bulimia nervosa*
   (b) Anorexia nervosa
   (c) Chronic dieters
   (d) Binge eating disorder

8. Individuals with BN have been found to have *higher* levels of _____ and *lower* levels of _____ in plasma.
   (a) Potassium, chloride, phosphate; prolactin, estradiol
   (b) Prolactin, estradiol; cortisol, testosterone
   (c) Cholesterol; potassium, chloride, phosphate*
   (d) Cortisol; cholesterol

9. Which of the following is known to stimulate hunger?
   (a) Leptin
   (b) Cholecystokinin
   (c) Ghrelin*
   (d) Peptide Y-Y

10. _____ is synthesized extensively in the human gastrointestinal tract and helps regulate appetite, sleep patterns, and mood.
    (a) Leptin
    (b) Serotonin*
    (c) Dopamine
    (d) Catechol-*O*-methyltransferase

## Chapter 12 Questions

1. The female athletic triad is a term which describes
   (a) Muscular endurance, cardiovascular endurance, and muscular strength
   (b) Amenorrhea, oligomenorrhea, and eumenorrhea
   (c) Speed, agility, and power
   (d) Disordered eating, amenorrhea, and osteoporosis*

2. Athletes at greatest risk for developing signs and symptoms associated with the triad are
   (a) Female basketball players under tremendous pressure to perform
   (b) Female hockey players at the peak of their season
   (c) Athletes competing in sports that emphasize leanness or a low body weight*
   (d) Female softball players, particularly pitchers

3. Which of the following statements is false with regard to athletic amenorrhea and bone loss?
   (a) Research has shown that in athletic amenorrhea, estrogen replacement alone normalized low bone density*
   (b) Mechanisms other than estrogen deficiency may account for low bone mineral density in women with amenorrhea
   (c) Women who consistently diet may slow their metabolic rates, which could affect bone metabolism through decreased leptin levels
   (d) Leptin receptors have been reported to be found in bone and may be important to osteoblastic function

4. In this particular eating disorder, even though the drive for thinness persists, the individual's body weight is typically normal throughout the disorder:
   (a) Anorexia nervosa
   (b) Bulimia nervosa*
   (c) Binge eating disorder
   (d) Compulsive overeating

5. Even though the profile of menstrual dysfunction varies considerably in athletes, there is a progression that develops ranging from the least severe form of menstrual dysfunction to the most severe form of menstrual dysfunction. The typical progression of menstrual dysfunction is:
   (a) 3, 6, 2, 1, 4, 5
   (b) 6, 5, 2, 1, 4, 3*
   (c) 5, 6, 1, 2, 3, 4
   (d) 1, 2, 4, 3, 6, 5
      1. Irregular cycles but still ovulating
      2. Regular cycles with failure to develop and release an egg (ovulation)
      3. Absence of menses and anovulation
      4. Irregular cycles and anovulation
      5. Regular cycles with inadequate progesterone production
      6. Regular cycles with a shortened luteal phase—progesterone production stops

6. Primary amenorrhea (delayed menarche) is defined as
   (a) The absence of menstruationby age 12 in a girl with secondary sex characteristics
   (b) The absence of menstruation by age 13 in a girl with secondary sex characteristics
   (c) The absence of menstruation by age 14 in a girl with secondary sex characteristics
   (d) The absence of menstruation by age 15 in a girl with secondary sex characteristics*

7. Secondary amenorrhea is
   (a) The absence of three or more consecutive menstrual cycles or a period of 6 months without menses
   (b) The absence of four or more consecutive menstrual cycles after cycles have been established
   (c) The absence of three or more consecutive menstrual cycles or a period of 3 months without menses after menarche or after cycles have been established*
   (d) A period of 6 months without menses after menarche or after cycles have been established

8. According to Loucks, energy availability is defined as
   (a) Dietary energy intake minus exercise energy expenditure (DEI – EEE)*
   (b) Dietary energy intake plus exercise energy expenditure (DEI + EEE)
   (c) Exercise energy expenditure minus dietary energy intake (EEE – DEI)
   (d) Dietary energy intake (DEI) minus total energy expenditure (heat from all cellular functions)

9. The International Society for Clinical Densitometry recommends that bone mineral density in premenopausal women and children be expressed as
   (a) Z-scores*
   (b) T-scores
   (c) Standard deviations for a 30-year-old adult
   (d) Ratios

10. Luteinizing hormone (LH) pulsatility is disrupted when energy availability is reduced below approximately
   (a) 30 kilocalories (kcal) per kilogram (kg) of fat-free mass (FFM) per day (kcal/kg FFM/day)*
   (b) 45 kilocalories (kcal) per kilogram (kg) of FFM per day (kcal/kg FFM/day)
   (c) 45 kilocalories (kcal) per kilogram (kg) of body weight (BW) per day (kcal/kg BW/day)
   (d) 30 kilocalories (kcal) per kilogram (kg) of body weight (BW) per day (kcal/kg BW/day)

## Chapter 13 Questions

1. The American College of Sports Medicine Position Stand on the Female Athlete Triad describes the interrelationships between
   (a) Energy availability
   (b) Menstrual function
   (c) Bone mineral density
   (d) All of the above*

2. Energy availability is the energy remaining for body functions after that used for exercise is added to total energy intake.
   (a) True
   (b) False*

3. One way an athlete's available energy may be reduced is
   (a) Decreased energy expenditure with reduced exercise
   (b) Increasing energy intake
   (c) Abnormal eating behaviors*
   (d) None of the above

4. Screening for disordered eating and eating disorders can occur at a common entry point to athletic participation.
   (a) True*
   (b) False

5. Once an athlete screens positively for possible low energy availability or if a concern exists, the athlete should
   (a) Celebrate their positive screening
   (b) Continue their normal training routine
   (c) Increase energy expenditure while decreasing energy intake
   (d) Be referred for further medical and psychological or psychiatric evaluation*

6. What makes a screening tool useful is
   (a) Functionality
   (b) Validity
   (c) Reliability
   (d) All of the above*
   (e) None of the above

7. A more formal screening evaluation for disordered eating/eating disorders should include
   (a) Detailed medical, nutritional, and reproductive history
   (b) Physical examination with lab evaluation
   (c) Referral to a psychologist
   (d) All of the above*
   (e) None of the above

8. The informal settings where athletes may be screened for disordered eating/eating disorders occur
   (a) While interacting with personal trainers, family, and friends*
   (b) When filling out PPEs
   (c) In clinical settings
   (d) None of the above

9. Physical complaints that can help diagnose an eating disorder include
   (a) Amenorrhea
   (b) Bradycardia
   (c) Skin changes
   (d) Low body mass index
   (e) All of the above*

10. The clinical tool that is recognized as the assessment of choice for diagnosing an eating disorder is
    (a) Eating disorders interview (EDI)
    (b) EAT-26
    (c) Eating disorders exam (EDE)*
    (d) Female athlete screening tool (FAST)
    (e) SCOFF

## Chapter 14 Questions

1. One recognized trait of those with disordered eating is
   (a) High self-efficacy
   (b) Low self-esteem*
   (c) Bipolar disorder
   (d) Early onset of menstruation

2. A second well-recognized trait of those with disordered eating is
   (a) Perfectionism*
   (b) Anger
   (c) High self-esteem
   (d) Optimism

3. One frequently used theoretical framework in health behavior change is
   (a) Process change model
   (b) Meta-change model
   (c) Transtheoretical model*
   (d) Decisional balance model

4. The TTM assumes that individuals
   (a) Have developmentally finite periods of motivation and readiness to change behaviors
   (b) Vary in motivation and readiness to change behaviors

(c) Realistically understand that relapse is normal with significant behavior change
(d) b and c*

5. Cognitive dissonance is a component of the
(a) *Athletes@Risk*® program to prevent the female athlete triad
(b) Dissonance-based eating disorder prevention program*

6. In treating patients with anorexia nervosa, especially those under the age of 18, research indicates which is effective?
(a) Concentrated individual therapy
(b) Family therapy*
(c) Psychodynamic therapy
(d) Group therapy

7. The usual treatment setting for a patient with bulimia nervosa is
(a) Inpatient
(b) Outpatient*

8. The recommended treatment setting for an individual with anorexia nervosa who weighs less than 75 % of average weight or has severe metabolic disturbances is
(a) Inpatient*
(b) Outpatient

9. The treatment of choice for those suffering with bulimia nervosa is
(a) Family therapy
(b) Psychodynamic therapy
(c) Rational emotive therapy
(d) Cognitive behavioral therapy*

10. Factors important to the treatment and recovery from disordered eating include
(a) Achieving and maintaining a normal weight
(b) The personality of the clinician
(c) The therapeutic relationship
(d) All listed above*

## Chapter 15 Questions

1. When is the final adult stature attained in females?
(a) 12–13
(b) 14–15
(c) 16–17*
(d) 18–19

2. Neuromuscular control and development most specifically target which anatomical structure in adolescent females, as compared to males as far as injury is concerned?
(a) Shoulder
(b) Knee*
(c) Wrist
(d) Hip

3. What anatomical structures are involved with the "miserable malalignment syndrome"?
(a) Pelvis and knee
(b) Knee and foot
(c) Hip and leg
(d) All of the above*

4. Name different ways to help prevent musculoskeletal injuries.
(a) Go as hard and fast as possible to build up strength and stamina
(b) Use other players' equipment, and play in bad weather
(c) Wear fancy name brand running shoes
(d) None of the above*

5. What are the best recommended first-aid measures for musculoskeletal injury management?
(a) Rest, ice, compression, elevation
(b) Rest, ice, compression, elevation, medication
(c) Protect, rest, ice, compression, elevation*
(d) ROM, rest, ice, compression, elevation

6. Which of the following intrinsic factors make a person more susceptible to injuries?
(a) Movement skills
(b) Intensity of activity
(c) Previous injury*
(d) Equipment

7. Which structure of tibia is affected by Osgood–Schlatter's disease?
(a) Tibial plateau
(b) Tibial tuberosity*
(c) Tibial plafond
(d) Tibial shaft

8. Dislocation of patella is most commonly seen in females aged
   (a) 6–10 years
   (b) 10–14 years
   (c) 14–18 years*
   (d) 18–22 years

9. Which is the most common ligament injured in lateral ankle sprains?
   (a) Deltoid ligament
   (b) Calcaneonavicular
   (c) Posterior talofibular
   (d) Anterior talofibular*

10. Spondylolysis in spine is defined as
    (a) A defect in the lamina between the superior and inferior articular facets*
    (b) Translational movements between vertebral bodies
    (c) Disc protrusion between two vertebral segments
    (d) Trabecular defect within the vertebral bone

## Chapter 16 Questions

1. What is the percentage of essential fat in males versus females?
   (a) 5 % and 10–15 %
   (b) 2–4 % and 10–12 %
   (c) 3 % and 9–12 %*
   (d) None of the above

2. Which sports tend to have a higher rate of musculoskeletal injuries in females?
   (a) Handball and lacrosse
   (b) Soccer and basketball
   (c) Gymnastics and volleyball
   (d) All of the above*

3. Which bones are more commonly involved as far as stress fractures in female athletes are concerned?
   (a) Tibia
   (b) Hip and pelvis
   (c) Ankle and foot
   (d) b and c*

4. Cite the incidence and specific anatomical structure which is more readily injured in females vs. males.
   (a) Five times higher, knee
   (b) 2–10 times higher, ACL*
   (c) 2–10 times higher, MCL
   (d) Five times higher, ACL

5. Athletic taping becomes ineffective for joint stability after what amount of time?
   (a) 20 min*
   (b) 10 min
   (c) 15 min
   (d) 30 min

6. Which is the most common mechanism of ACL injury in women athletes?
   (a) Hip external rotation and tibial internal rotation
   (b) Hip internal rotation and tibial external rotation*
   (c) Hip and tibial internal rotation
   (d) Hip and tibial external rotation

7. Miserable malalignment syndrome is associated with
   (a) Decreased Q-angle
   (b) Genu varum
   (c) Hypomobile patella
   (d) Increased femoral anteversion*

8. ITB syndrome is associated with
   (a) Weak gluteus medius muscle*
   (b) Excessive tibial external rotation
   (c) Genu recurvatum
   (d) Reduced foot pronation

9. The most common level of spinal segments affected by spondylolysis is
   (a) L1–L2
   (b) L2–L3
   (c) L3–L4
   (d) L4–L5*

10. Which of the following should not be adopted as a rehabilitation regimen after an acute injury?
    (a) Maintaining ROM
    (b) PRICE

(c) Plyometrics*
(d) Muscle strengthening

## Chapter 17 Questions

1. Name the pertinent anatomy involved in pregnancy
   (a) Uterus and ovaries
   (b) Fallopian tubes
   (c) Mammary glands
   (d) All of the above*
2. What are the hormones associated with the pregnant state?
   (a) HCG, relaxin, prolactin
   (b) Progesterone and estrogen
   (c) Insulin, corticol, thyroid, parathyroid
   (d) a and b*
3. Name the "core" group of muscles supporting the fetus in the pregnant woman
   (a) The pelvic walls/floor musculature
   (b) Iliolumbar ligaments, the iliopsoas tendon, the rectus abdominus
   (c) Internal/external obliques, the quadratus lumborum, the erector spinae
   (d) a, b, and c*
4. Compensating body structural changes involved with pregnancy include the following:
   (a) Increased cervical tilt
   (b) Increased lumbar lordosis and thoracic kyphosis
   (c) Increased posterior upper body tilt and sagittal pelvic tilt
   (d) b and c*
5. Common musculoskeletal injuries occurring with exercise during pregnancy include
   (a) Transient hip osteoporosis
   (b) Diastasis recti and symphyseal diastasis
   (c) Meralgia paresthetica and de Quervain's stenosing tenosynovitis
   (d) All of the above*
6. Which positions should one avoid when exercising while pregnant?
   (a) Prolonged standing or supine lying
   (b) Excessive sitting or side-lying
   (c) Repetitive Valsalva maneuver or isometric contraction
   (d) a and c*
7. Which body areas experience the most load during pregnancy and thus should be protected?
   (a) Lumbar spine, pelvis, and SI joints
   (b) Hip and knee joints*
   (c) Shoulder and ankle joints
   (d) None of the above
8. Relatively safe medications/modalities which can be used by a pregnant woman include
   (a) Anesthetic/steroid injections*
   (b) NSAIDs/ASA
   (c) Ultrasound
   (d) E-stim device
9. At what point should one be concerned about pubic symphysis widening?
   (a) 1–2 cm
   (b) 2–3 cm
   (c) 3–4 cm
   (d) 4–5 cm*
10. A pregnant woman should avoid performing which of the following activities?
    (a) Swimming, skating
    (b) Non-contact team sports
    (c) Skiing, diving, horseback riding*
    (d) All of the above

## Chapter 18 Questions

1. What are some of the musculoskeletal manifestations which occur over the years of aging?
   (a) Hypokinesia
   (b) Sacropenia
   (c) Osteoporosis
   (d) All of the above*
2. Age-related sarcopenia is associated with
   (a) Decreased metabolic rate
   (b) Decreased soft tissue flexibility
   (c) Increased body fat
   (d) All of the above*

3. Kyphosis is a ___ plane deformity.
   (a) Sagittal*
   (b) Coronal
   (c) Horizontal
   (d) None of the above

4. Osteoarthritis is first manifested in which of the following joint?
   (a) Tibiofemoral
   (b) Carpometacarpal
   (c) Interphalangeal
   (d) Metacarpophalangeal*

5. Name some common sites of osteoporotic fractures affecting postmenopausal women.
   (a) Spine and wrist
   (b) Wrist and hip
   (c) Spine, wrist, and hip*
   (d) None of the above

6. What are some indicators of potential osteoporotic fractures?
   (a) 2″ loss in height
   (b) Hip pain after falling from standing height
   (c) Low BMI
   (d) a and b*

7. Which types of exercise have been found to be beneficial in older, active females?
   (a) Bicycling, aquatic aerobics
   (b) Skiing, rock climbing
   (c) Dancing, jogging
   (d) a and c*

8. What treatment modalities have been shown to benefit musculoskeletal health in the master athlete?
   (a) Glucosamine/chondroitin sulfate
   (b) Fish oil supplements*
   (c) Calcium/vitamin D
   (d) Sports supplements

9. ACSM and CDC recommend ___ of moderate-intensity exercise 3–7 days/week.
   (a) 5–10 min
   (b) 20–60 min*
   (c) 70–90 min
   (d) 90–120 min

10. To maximize aerobic fitness, the target heart rate range should be ___ of maximal heart rate.
    (a) 20–30 %
    (b) 80–100 %
    (c) 40–80 %*
    (d) All of the above

## Chapter 19 Questions

1. The three most common fracture sites in patients with osteoporosis are
   (a) Humerus, ankle, thoracic spine
   (b) Rib cage, pelvic girdle, distal ulna
   (c) Lumbar vertebrae, distal radius, femoral neck*
   (d) Clavicle, proximal radius, tibia

2. The World Health Organization has created useful definitions of osteoporosis based upon bone density measurements. Which of the following statements for those definitions is correct?
   (a) The Z-score is the most useful measurement when determining the diagnosis of osteoporosis
   (b) The T-score is the number of standard deviations above or below age- and race-matched young controls*
   (c) The definition of osteoporosis is a Z-score of less than −2.5 SD
   (d) T-score compares the patient with a population adjusted for age, gender, and race

3. The minimum dose of conjugated equine estrogen that has been shown to prevent osteoporosis in patients who ingest at least 1,200 mg of calcium in their diet and/or via calcium supplementation is which of the following:
   (a) 0.3 mg*
   (b) 2.5 mg
   (c) 0.9 mg
   (d) 0.625 mg

4. Factors known to increase the risk of osteoporosis include all of the following *except*
   (a) High alcohol intake in diet
   (b) Early spontaneous menopause with hormone therapy

(c) Cigarette smoking
(d) Obesity*
(e) Sedentary lifestyle

5. Each of the following are listed as important side effects of the bisphosphonates about which patients should be counseled, *except*
   (a) Uveitis, esophagitis, scleritis
   (b) Osteonecrosis of the mandible (jawbone)
   (c) Hot flashes, leg cramps, and possible thromboembolic events*
   (d) Pain in back, hips or other joints, atypical fractures of the femur

6. The National Osteoporosis Foundation and the North American Menopause Society recommendations concerning DXA (bone density testing) include which of the following?
   (a) Baseline *DXA* at age 50, earlier DXA with family or personal risk factors
   (b) Baseline DXA at 55, if normal, repeat DXA every 1–2 years
   (c) Baseline DXA only if personal or family risks exist, regardless of age
   (d) Baseline DXA at age 65, or baseline at younger age if other personal or familial risk factors exist including personal history of fragility fracture*

7. Match the following medications with their associated benefits or risks:

| | |
|---|---|
| (a) Raloxifene | w. Available in both injectable and oral forms |
| (b) Teriparatide | x. Shown to help prevent breast cancers |
| (c) Ibandronate | y. Associated with the development of osteosarcomas in rats |
| (d) Estrogen | z. May increase the risk of stroke, heart disease, and breast cancer |

Question 7 answer: (a) = x, (b) = y, (c) = w, (d) = z

8. Supplementation of calcium of greater than 2,000 mg daily has been associated with which of the following:
   (a) Improved bone mineral density
   (b) Increased risk of cardiovascular disease*
   (c) Formation of gallstones
   (d) Increased risk of esophageal reflux and stomach ulcer formation

9. The data on the effectiveness of phytoestrogens, botanicals, and other herbal therapies for the treatment of osteoporosis remains scant and somewhat controversial. The best studied phytoestrogen, which also may have proven benefits, is which of the following?
   (a) Soy(bean) isoflavones*
   (b) Black cohosh
   (c) Wild yam
   (d) Ginkgo biloba

10. According to the WHI (Women's Health Initiative Study), estrogen did show beneficial effects on the bone mineral density of postmenopausal women. What other health benefits of estrogen were documented in that study?
    (a) Decreased risk of breast and ovarian cancer
    (b) Overall reduction in risk of stroke and thromboembolic events
    (c) Improved cardiovascular health
    (d) Reduction in risk of colon cancer*

## Chapter 20 Questions

1. Please read the scenario to answer this question. Three responses are required for this question: (a)What is the recommended load to develop power for novice- and intermediate-trained individuals? (b) How many repetitions are suggested per set? (c) What is the recommended rest period between sets when training for power?

   You are an athletic trainer for a high school women's cross-country track team. At your school, you do not have a strength and conditioning coach for your athletes. Ashley, one of your senior track athletes, came in to see because of a sports-related injury. You questioned her regarding the origin of her injury. She stated that she has been following the coaches training guidelines for school practice; however, she has begun a resis-

tance-training program with a friend at a local gym. She stated that she has been training for power in order to increase her speed. Her resistance-training program consists of 6–8 repetitions at 70–85 % of 1 RM with 1-min rest between multiple sets of three:
(a) (a) 20–50 % of 1 RM; (b) 1–5 repetitions; (c) 4–5 min of rest between sets
(b) (a) 60–90 % of 1 RM; (b) 3–10 repetitions; (c) 1–2 min of rest between sets
(c) (a) 30–60 % of 1 RM; (b) 3–6 repetitions; (c) 2–3 min of rest between sets*

2. What are the minimum days per week one should strength train?
   (a) None, you do not need to strength train
   (b) 7
   (c) 4
   (d) 2*

3. On average, compared to those who are unactive, people who are physically active …
   (a) Live longer*
   (b) Eat more fast food
   (c) Have more health problems
   (d) Are better singers

4. According to the ACSM, moderate exercise intensity is at which percentage of oxygen uptake reserve?
   (a) 20 to <40 %
   (b) 40 to <60 %*
   (c) 50 to <60 %
   (d) 50 to <70 %

5. According to the ACSM, vigorous exercise intensity is at which percentage of oxygen uptake reserve?
   (a) 40 to <60 %
   (b) 60 to <75 %
   (c) 60 to <90 %*
   (d) 75 to <90 %

6. Which of the following is NOT a health-related component of physical fitness?
   (a) Cardiovascular endurance
   (b) Muscular strength and endurance
   (c) Body composition
   (d) Mental toughness*

7. Which of the following principles are important for a resistance-training program?
   (a) Overload principle
   (b) Principle of progression
   (c) Consistency principle
   (d) Both a and b*

8. When beginning an exercise regimen, it is important to include which of the following aspects?
   (a) Flexibility
   (b) Cardiovascular fitness
   (c) Muscular strengthening
   (d) All of the above*

9. Hypertrophy refers to …
   (a) Muscle growth*
   (b) Muscle shrinking
   (c) Muscle endurance
   (d) Muscle strength

10. What is the minimum number of minutes per week one should exercise in order to lose weight?
    (a) >75
    (b) >150*
    (c) >300
    (d) >450

## Chapter 21 Questions

1. You notice that your 6-year-old daughter enjoys running very much. How often should you at least let her run and be active?
   (a) 30 min/day, 1 day/week
   (b) 60 min/day, 1 day/week
   (c) 60 min/day, 3 days/week*

2. What intensity level would be appropriate for your daughter in order to obtain the health benefits?
   (a) Moderate to vigorous intensity*
   (b) Low intensity
   (c) Very high intensity

3. A physical activity program for a child (in general) should focus on
   (a) Execution of skill with the proper form, enjoyment, and social interaction*

(b) Training at very high intensities in order to win the little league
(c) One and only one activity alone so that the child has the highest chance of winning

4. Why is the metabolic cost higher in children than adults?
   (a) Lower resting basal metabolic rate
   (b) Lower stride frequency
   (c) Locomotion is biomechanically inefficient*

5. Which of the following is a benefit of starting physical activity participation during childhood and adolescence rather than when the individual is older?
   (a) Children are likely to get less injured
   (b) Children are more likely to continue participation into adulthood*
   (c) You cannot learn new skills when you are older

6. Which of the following is true?
   (a) The $VO_{2max}$ of children is lower than adults
   (b) There is no difference in the performance of skills between girls and boys as they age
   (c) The $VE/VCO_2$ slope is normally higher in children than in adults*

7. When should your son's exercise program be supervised and designed by a professional trainer or coach?
   (a) When your 8-year-old son wants to participate in an organized sport or activity*
   (b) When your 8-year-old son wants to play for hours and hours at the park
   (c) When your 8-year-old son is too engrossed with video games

8. Your 8-year-old son is seriously interested in the different track and field events. Why should you hire a coach/trainer for your son?
   (a) To look cool in front of his friends
   (b) To ensure that the exercises and training are effective, age appropriate, and safe*
   (c) He does not need a coach/trainer because the exercise programs available on the Internet are certainly safe and effective

9. Which of the following is true?
   (a) A child should only exercise at an intensity of 1–2 METs
   (b) Peak work rate is greater in children than in adults
   (c) HR recovery in children is faster than in adults*

10. Children should be physically active in order to prevent
    (a) Certain health problems that may develop later on*
    (b) Social interaction
    (c) Laziness

## Chapter 22 Questions

1. Which is not listed as a component of physical fitness?
   (a) Strength
   (b) Speed
   (c) Coordination
   (d) Jumping*

2. Skeletal muscle mass accounts for what percentage of body weight at birth?
   (a) 23–25 %*
   (b) 30–35 %
   (c) 42–46 %
   (d) 28–30 %

3. What has been defined as the skeletal muscle mass loss associated with an impaired functioning affecting physical performance?
   (a) Osteopenia
   (b) Hypertrophy
   (c) Sarcopenia*
   (d) Osteoporosis

4. Resistance training was avoided in the past for children because of which of the following?
   (a) Damage to growth plates
   (b) Not able to receive benefits
   (c) Both a and b*
   (d) None of the above

5. For healthy children, the ratio of instructors to students should be
   (a) 1:20
   (b) 1:5
   (c) 1:15
   (d) 1:10*

6. When increases in muscular strength is the goal of a resistance-training program, the load should be _____ 1 RM for novice- and intermediate-level lifters.
   (a) 70–100 %
   (b) 30–60 %
   (c) 60–70 %*
   (d) 80–90 %

7. When the elderly restart resistance training after a period of cessation, what percentage should they start out with?
   (a) 70 % or less
   (b) 50 % or less*
   (c) 60 % or less
   (d) 80 % or less

8. Which resistance-training program is designed to have more repetitions at lighter loads with a short rest period?
   (a) Hypertrophy
   (b) Power
   (c) Muscular strength
   (d) Muscular endurance*

9. Which resistance-training program is designed to have fast lifting velocities?
   (a) Hypertrophy
   (b) Power*
   (c) Muscular strength
   (d) Muscular endurance

10. When an individual is ready to progress, they should increase _____ when they exceed the current workload on _____ consecutive sessions.
    (a) 5–15 %, 2
    (b) 2–10 %, 1
    (c) 5–15 %, 1
    (d) 2–10 %, 2*

## Chapter 23 Questions

1. Choose the correct order of these terms going from least severe to most severe:
   (a) Staleness, overtraining, overreaching, the overtraining syndrome
   (b) Overtraining, staleness, the overtraining syndrome, overreaching
   (c) Staleness, overreaching, overtraining, the overtraining syndrome*
   (d) The overtraining syndrome, overreaching, staleness, overtraining

2. True or false: Overtraining usually occurs when an athlete is exposed to high volumes of training with adequate periods of rest
   (a) True
   (b) False*

3. A sign and symptom of nonfunctional overreaching, or overtraining, is
   (a) An increase in thirst*
   (b) Mood changes
   (c) Loss of appetite
   (d) Slight fatigue

4. Which of the following conditions takes several months to years to recover from?
   (a) Functional overreaching
   (b) Overtraining
   (c) Nonfunctional overreaching
   (d) The overtraining syndrome*

5. Which of the following is NOT a preventive measure of overtraining?
   (a) Maintain proper hydration
   (b) Perform every exercise to maximum exhaustion*
   (c) Switch up training sessions to avoid monotony
   (d) Administer an interactive coaching style

6. Fill in the blank: The overtraining syndrome is a chronic condition lasting for _____ or months at a time.
   (a) Hours
   (b) Days

(c) Weeks*
(d) Years

7. True or false: The overtraining syndrome can sometimes be confused as depression.
   (a) True*
   (b) False

8. A sign and symptom of sympathetic overtraining is
   (a) Weight gain
   (b) A decreased metabolic rate
   (c) Palpitation*
   (d) Increased coordination

9. The hypo-arousal, or parasympathetic, state is much more prevalent and occurs in endurance or aerobically centered athletes such as
   (a) Sprinters
   (b) Swimmers*
   (c) Divers
   (d) Baseball pitchers

10. Chris is a college football player. He has been experiencing a loss of appetite and has recently noticed some mood changes and a decrease in his overall performance during games. His coach recently had Chris perform a $VO_{2max}$ test. His current $VO_{2max}$ from this test is 55 ml/kg/min. This is significantly lower than his last measured $VO_{2max}$ of 60 ml/kg/min of his last test done 6 months ago. Which of the following is Chris most likely experiencing?
    (a) Overreaching
    (b) Depression
    (c) Overtraining
    (d) The overtraining syndrome*

## Chapter 24 Questions

1. Which is the best indicator to determine immunocompetence as a whole for a clinical point of view?
   (a) Incidence of URTI*
   (b) Number of leukocytes
   (c) Function of NK cells
   (d) Stress level

2. The J curve was first proposed based on
   (a) Studies with moderate exercise intensity
   (b) Studies in nonathletic population
   (c) Studies in athletic population that performed high-intensity exercise*
   (d) Studies in rats

3. After moderate exercises, studies showed that lung macrophages
   (a) Improved antiviral resistance*
   (b) Decreased antiviral resistance
   (c) Decreased in numbers
   (d) Increased in numbers

4. During prolonged aerobic exercise
   (a) Only a sharp increase of leukocyte is observed
   (b) A biphasic increase in leukocyte is observed*
   (c) A decrease in leukocyte is observed
   (d) No changes in leukocytes are observed

5. Which is the most responsible cell to exercise stimulus?
   (a) Neutrophils
   (b) Lymphocyte T
   (c) Lymphocyte B
   (d) NK cells*

6. Why NK cells increase their cell number immediately after exercise?
   (a) Increased catecholamine production*
   (b) Decreased plasma volume
   (c) Increased Th2 lymphocyte numbers
   (d) Increased T lymphocytes

7. The only immune parameter recognized by researcher's consensus as responsible for immune suppression after intense exercise was
   (a) Decreased NK cell function
   (b) Decreased IgA-salivary production*
   (c) Increased lymphocyte B
   (d) Decreased cytokine production

8. Which supplementations are support in athletes by the researchers consensus performed in 2011?
   (a) Vitamin C
   (b) Vitamin D
   (c) Quercetin*
   (d) Glutamine

9. Which variable is less important to control in further studies to analyze the difference in the cytokine production among women and men?
   (a) Menstrual cycle phase
   (b) Oral conceptive taken
   (c) Fitness level
   (d) Vitamin supplementation*
10. During URTI episodes, the guidelines for exercise recommend do not exercise if we have
   (a) Runny nose
   (b) Fever*
   (c) Sore throat
   (d) Cough

## Chapter 25 Questions

1. Excessive exercise describes a quantity exercise performed _____ the physical healthy limits.
   (a) Before
   (b) Up to
   (c) Beyond*
   (d) None of the above
2. In primary exercise addiction physical activity
   (a) Is an end in itself*
   (b) Exercise is not the objective
   (c) Co-occurs with an eating disorder or other compulsive disorders
   (d) Weight loss is the objective
3. Coverley Veale et al. proposed a classification for exercise addiction (EA) depending on what causes it or which is the role of the exercise called:
   (a) Restrictive and negative addiction
   (b) Restrictive and compulsive addiction
   (c) Positive and negative addiction
   (d) Primary and secondary addiction*
4. Etiological theories based on physiological factors are
   (a) Endorphin hypothesis and general theory of addiction
   (b) Anorexia analogue and endorphin hypothesis
   (c) Endorphin and sympathetic arousal hypothesis*
   (d) Personality traits and sympathetic arousal hypothesis
5. Exercise intensity (performed above 60 % of the maximal oxygen uptake) and duration (sustained for at least 3 min) are related to
   (a) Increased plasma b-endorphins*
   (b) Decreased plasma b-endorphins
   (c) Changes in brain functioning
   (d) No changes in plasma b-endorphins
6. Sympathetic arousal hypothesis reports increased concentrations of catecholamine induced by intense physiological or psychological stress.
   (a) Researchers have reported 1.5 to >5 times greater than basal concentrations
   (b) Researchers have reported 1.5 to >10 times greater than basal concentrations
   (c) Researchers have reported 1.5 to >15 times greater than basal concentrations
   (d) Researchers have reported 1.5 to >20 times greater than basal concentrations*
7. The prevalence of EA is
   (a) Uncertain*
   (b) 20 %
   (c) 5 %
   (d) 0.5 %
8. A screening tool that is measuring minutes, intensity, or time spent exercising is
   (a) Qualitative
   (b) Quantitative and qualitative
   (c) Quantitative*
   (d) None of all
9. Which is not a qualitative screening tool?
   (a) Obligatory exercise questionnaire (OEQ)
   (b) International physical activity questionnaire (IPAQ)*
   (c) Exercise addiction inventory (EAI)
   (d) Exercise dependence scale revised (EDS-R)
10. For eating disorders patients more than 6 h of physical activity per week during at least ___ consecutive weeks indicate excessive exercise (EE) practice.
   (a) 1
   (b) 2
   (c) 3
   (d) 4*

## Chapter 26 Questions

1. Hormonal and physiological changes happening in pregnancy will affect the _____ system during exercise.
   (a) Musculoskeletal
   (b) Metabolic
   (c) Cardiovascular*
   (d) Gastrointestinal

2. _____ is a temporary condition which comes about the end of pregnancy as a result of the action of insulin and placental hormones.
   (a) Gestational diabetes*
   (b) Diabetes
   (c) Preeclampsia
   (d) Both b and c

3. What disorder is NOT related with high blood pressure during gestation?
   (a) Chronic hypertension
   (b) Gestational hypertension
   (c) Preeclampsia/eclampsia
   (d) Gestational hypotension*

4. _____ is a disorder related to hypertension.
   (a) Gestational diabetes
   (b) Preeclampsia*
   (c) Both a and b
   (d) None of the above

5. What is one of the most important reasons for maternal mortality?
   (a) Exercise
   (b) Diabetes
   (c) Preeclampsia*
   (d) All of the above

6. What reduces the duration of active stages of labor and diminishes the incidence of obstetric difficulties during labor?
   (a) Exercise*
   (b) Food
   (c) Company
   (d) Music

7. What is the main concern when planning physical activity for pregnant?
   (a) Chronic fatigue*
   (b) Weight loss
   (c) Back pain
   (d) Cramps

8. What is NOT listed as the most described events affecting the fetus as a consequence of excess or wrong exercise?
   (a) Acute hyperthermia
   (b) Hypotension*
   (c) Acute hypoxia
   (d) Risk of preterm delivery

9. The most important aspect of exercise prescription in pregnancy is
   (a) Duration
   (b) Frequency
   (c) Intensity*
   (d) None of the above

10. Post delivery the FIRST aim is to perform exercise focused on recovering strength of _____
    (a) Abdominal muscles
    (b) Perineal muscles*
    (c) Hamstrings
    (d) Quadriceps

## Chapter 27 Questions

1. Resting metabolic rate can be accurately estimated from
   (a) FFM and fat mass*
   (b) Thyroid hormones
   (c) Thermic effect of food
   (d) Total daily energy expenditure

2. Adaptive thermogenesis occurs when
   (a) After a period of intervention measured RMR is increased higher than predicted values from changes on body composition
   (b) After a period of intervention measured RMR is reduced lower than predicted values from changes on body composition*
   (c) After a period of intervention measured RMR is equally increased as predicted values from changes on body composition
   (d) After a period of intervention measured RMR is equally reduced as predicted values from changes on body composition

3. Physical activity level (PAL) is the ratio of
   (a) RMR over thermic effect of food
   (b) Total daily energy expenditure over thermic effect of food
   (c) NEAT over RMR
   (d) Total daily energy expenditure over RMR*

4. Metabolic equivalents or METs can be transformed to $VO_2$ using the classical constant of
   (a) 1 MET = 1 L/min
   (b) 3.5 METs = 1 L/min
   (c) 1 MET = 3.5 ml/kg/min*
   (d) 1 MET = 1 kcal/kg/h

5. IPA-Q is a/an
   (a) Indirect calorimeter
   (b) Questionnaire used to estimate energy expenditure
   (c) Implement to perform appealing questions
   (d) Questionnaire used to estimate total daily physical activity*

6. The knowledge of oxidation rate of energetic substrates is an important issue in order to highlight metabolic pathways involved during exercise. What are the techniques that can estimate substrate oxidation ratios?
   (a) Doubly labeled water
   (b) Indirect calorimetry*
   (c) Direct calorimetry
   (d) All of them

7. Which sentence about total energy expenditure (TEE) is correct?
   (a) TEE is composed of the sum of BEE, RMR, TEF, and EEPA
   (b) The most variable component of TEE is EEPA
   (c) The resting or the basal component of energy expenditure constitutes the largest portion (60–75 %) of the TEE*
   (d) The TEE depends only on the nutrient content of the foods ingested

8. What does it mean that the MET of a certain activity is 5?
   (a) The energy expenditure of that activity is 5 kcal/min
   (b) The TEE multiplies by 5 when this activity is included
   (c) The energy expenditure of that activity is 5 kcal/kg
   (d) The energy expenditure of that activity is five times above the resting rate*

9. About the methods for measuring energy expenditure and requirements:
   (a) The doubly labeled water method is the most accurate technique for measuring TEE*
   (b) The energy requirements can be accurately estimated measuring the energy intake
   (c) Indirect calorimetry is an expensive technique, impractical for routine use by clinicians
   (d) Most physical activity questionnaires are valid for estimating TEE

10. The doubly labeled water method
    (a) Is a potentially dangerous method since it uses radioactive H and $O_2$
    (b) Permits the calculation of TEE based on the turnover rate of $^{2}H$
    (c) Consists on the intravenous infusion of water with deuterium
    (d) Uses two stable isotopes of water, $^{2}H$ and $^{18}O$*

## Chapter 28 Questions

1. Approximately what percentage of Americans aged 2–19 years are overweight or obese?
   (a) 66 %
   (b) 13 %
   (c) 32 %*
   (d) 9 %

2. Dietary reference intake (DRI) offers recommendations based on all of the following EXCEPT
   (a) Gender
   (b) Life stage
   (c) Activity level
   (d) All are used to create DRIs*

3. According to the estimated calorie needs per day, how many calories should a 10-year-old active female consume?
   (a) 1,000
   (b) 1,400
   (c) 2,000*
   (d) 2,200

4. According to the new MyPlate, you should fill at least half of your plate with
   (a) Whole grains
   (b) Fruits and vegetables*
   (c) Lean proteins
   (d) Low-fat dairy products

5. Which of the following is an example of a recommended whole grain?
   (a) White bread
   (b) Brown rice*
   (c) White spaghetti noodles
   (d) Degermed cornmeal

6. Daily dairy recommendations for children 2–3 years old are
   (a) One cup
   (b) Two cups*
   (c) Three cups
   (d) Two and a half cups

7. For all age groups, carbohydrates should make up _____ of the daily recommended calorie intake.
   (a) <10 %
   (b) 10–35 %
   (c) 25–35 %
   (d) 45–65 %*

8. Which of the following are not a characteristic of protein?
   (a) Composed of amino acids
   (b) Proteins should include lean meats and poultry
   (c) Necessary for growth and repair of our body's tissue
   (d) Primary energy source*

9. It is recommended that the fats consumed should be
   (a) Saturated
   (b) Polyunsaturated*
   (c) Trans-fatty acids
   (d) Cholesterol

10. Which of the following is not a micronutrient?
    (a) Fats*
    (b) Fiber
    (c) Minerals
    (d) Vitamins

## Chapter 29 Questions

1. The adult female human brain oxidizes approximately _____ of glucose each day at a continuous rate, and this must be provided daily by dietary _____ because the brain's rate of energy expenditure can deplete liver glycogen stores in <1 day.
   (a) ~50 g, carbohydrate
   (b) ~30 g, protein
   (c) ~80 g, carbohydrate*
   (d) ~40 g, fat

2. According to Loucks et al. (2003, 1998), functional amenorrhea in young exercising women occurs primarily because of
   (a) Excessive exercise stress
   (b) Excessive psychological stress related to competition and performance
   (c) Dietary energy restriction or "energy drain"*
   (d) Protein availability

3. Reproductive function in women critically depends on the pulsatile release of _____ from neurons in the arcuate nucleus of the hypothalamus and on the consequent pulsatile release of _____ from the pituitary.
   (a) Follicle-stimulating hormone (FSH), progesterone
   (b) Gonadotropin-releasing hormone (GnRH), luteinizing hormone (LH)*
   (c) LH, GnRH
   (d) FSH, estrogen

4. A joint position statement by the American College of Sports Medicine, the American Dietetic Association, and the Dietitians of

Canada stated that a diet substantially different from that recommended in the Dietary Guidelines for Americans or the Nutrition Recommendations for Canadians is needed for athletes.
(a) True*
(b) False

5. Loucks and Thuma found that LH pulsatility was disrupted abruptly at a threshold of energy availability of less than _____ kcal/kg of lean body mass per day (LBM day).
(a) 45
(b) 60
(c) 20
(d) 30*

6. Research consistently shows that female athletes
(a) Tend to take in more calories than they need
(b) Eat a well-balanced diet
(c) Eat at fast food restaurants like McDonalds and Burger King for most of their meals
(d) Takes in fewer calories than they actually need*

7. Secondary amenorrhea is defined as
(a) The absence of three or more consecutive menstrual cycles or a period of 6 months without menses after menarche or after cycles have been established
(b) The absence of five or more consecutive menstrual cycles or a period of 3 months without menses after menarche or after cycles have been established
(c) The absence of three or more consecutive menstrual cycles or a period of 3 months without menses after menarche or after cycles have been established*

8. Primary amenorrhea is the absence of menstruation by age _____ in a girl with secondary sex characteristics.
(a) 12
(b) 13
(c) 15*

9. The term functional hypothalamic amenorrhea means
(a) The menstrual disorder is an anatomical disorder that is not functioning at the level of the hypothalamus
(b) There is an altered hormonal pattern that may be caused by a cyst or other obstruction and if removed the menstrual cycles may become functional again
(c) There is a functional problem, not an anatomical one, and it is reversible*

10. The most accurate term for the menstrual disorder referred to in the triad is
(a) Primary amenorrhea
(b) Secondary amenorrhea
(c) FHA*

## Chapter 30 Questions

1. Which of the following reasons have female athletes given for consuming protein supplements?
(a) Like the taste
(b) Provide more energy and meet nutritional needs
(c) Gain strength and enhance performance
(d) All of the above*

2. Which of the following protein supplements increase protein synthesis during exercise?
(a) Whey
(b) Casein*
(c) Soy
(d) They are all equal in protein synthesis rate during exercise

3. Regarding EAA supplementation, there is an "anabolic window" post exercise. Which of the following is NOT a benefit of supplementing EAAs during the anabolic window?
(a) Increased muscle protein synthesis
(b) Enhanced complex carbohydrate digestion rate*
(c) Increased glycogen restoration
(d) Enhanced recovery process

4. The central fatigue hypothesis states that low blood concentrations of BCAAs
   (a) Increase glycogen restoration
   (b) Increase production of 5-HTP*
   (c) Reduce production of 6-HTP
   (d) Decrease the amount of tryptophan entering the brain
5. Arginine is a conditional EAA synthesized from
   (a) Ornithine
   (b) Citrulline
   (c) Glutamine
   (d) Both a and b*
6. The recommended dosage of L-carnitine per day is
   (a) >4 g/day
   (b) 630 g/day
   (c) 2–3.5 g/day*
   (d) 15 g/day
7. Which of the following statements is true about ginseng?
   (a) All forms have been equally effective in improving performance
   (b) 8 weeks of supplementation with Chinese ginseng improved Wingate performance
   (c) Siberian ginseng is more effective at improving $VO_{2max}$
   (d) Results are equivocal regarding ginseng performance improvements*
8. Echinacea has been reported to
   (a) Improve immune system function
   (b) Enhance protein synthesis at rest
   (c) Enhance $VO_{2max}$
   (d) Both a and c*
9. Female athletes concerned about body composition may take which of the following supplements?
   (a) Ephedra*
   (b) Echinacea
   (c) Multivitamin
   (d) None of the above
10. Adverse effects of anabolic steroid use may include
    (a) Decreased HDL
    (b) Enlargement of the clitoris
    (c) Muscle hypertrophy
    (d) Both a and b*

## Chapter 31 Questions

1. By what percentage and caloric amount should basic energy needs in pregnancy be increased over the nonpregnant state in a normal woman?
   (a) 5 % Increase and an additional 300 kcal/day
   (b) 12 % Increase and an additional 200 kcal/day
   (c) 17 % Increase and an additional 300 kcal/day*
   (d) 20 % Increase and an additional 200 kcal/day
2. During pregnancy, protein/fat/carbohydrate consumption should consist of …
   (a) 10 % Protein/25 % fat/65 % carbohydrate
   (b) 25 % Protein/25 % fat/50 % carbohydrate
   (c) 30 % Protein/20 % fat/50 % carbohydrate
   (d) 20 % Protein/30 % fat/50 % carbohydrate*
3. For how many months prior to conception and post conception should a woman supplement with folic acid?
   (a) 3 months prior to and 1 month post conception
   (b) 3 months prior to and 3 months post conception*
   (c) 1 month prior to and 6 months post conception
   (d) 1 month prior to and 1 month post conception
4. Which of the following is not a good source of iron for pregnant women?
   (a) Oysters
   (b) Apples*
   (c) Spinach
   (d) Legumes
5. Ideal birth weight, delivery time, and maternal health occur when Hb at term is between …
   (a) 95 and 125 g/dL*
   (b) 75 and 85 g/dL

(c) 55 and 56 g/dL
(d) 40 and 45 g/dL

6. Vitamin D deficiencies in pregnant women can lead to all of the following in children except …
   (a) Rickets
   (b) Hypocalcemia
   (c) Proper enamel formation*
   (d) Delayed ossification

7. A fetus is sensitive and vulnerable to neural tube defects (NTD) during which of the following weeks?
   (a) Third post-conception week to the eighth conception week*
   (b) First post-conception week to the third conception week
   (c) Eighth post-conception week to the tenth conception week
   (d) Third post-conception week to the fifth conception week

8. Food fortification with which two nutrients has been shown to reduce the incidence of NTD?
   (a) Vitamin A and vitamin C
   (b) Folic acid and B vitamins*
   (c) Vitamin K and magnesium
   (d) Potassium and vitamin E

9. In diabetic women who become pregnant, how many times greater is the risk of congenital malformations related to poor control of diabetes?
   (a) Four times
   (b) Three times
   (c) Two times
   (d) None of the above*

10. How many mg of folate per day is recommended to reduce the incidence of NTD during pregnancy?
    (a) 44.0 mg
    (b) 4.0 mg
    (c) 0.4 mg*
    (d) 0.04 mg

## Chapter 32 Questions

1. Aging is associated with a change in body composition, which can be related to
   (a) Decreased fat mass
   (b) Increased FFM
   (c) Increased and a redistribution of fat mass*

2. Which factors determine the caloric intake?
   (a) Weight, height, and gender
   (b) Height and age
   (c) Weight, height, age, gender, and PAL*

3. The TEE decreases with increasing age. Causes of the decline are
   (a) Decreased physical activity and fat mass
   (b) Decrease in FFM, physical activity, and basal metabolic rate*
   (c) Reduction in thermic effect of food

4. Using the equation of Mifflin–St Jeor for RMR:
   $RMR = 9.99 \times \text{weight (kg)} + 6.25 \times \text{height (cm)} - 4.92 \times \text{age (year)} + 166 \times \text{sex (males, 1; females, 0)} - 161.$

| Age = 75 years | Weight = 72 kg | Height = 1.74 m |
|---|---|---|

   Additional caloric requirements = 80 % (moderate activity)
   TEE for older women:
   (a) 2,300 kcal/day
   (b) 2,296 kcal/day
   (c) 1,957 kcal/day
   (d) 2,588 kcal/day*

5. Using the equation $EER = 354 - (6.91 \times \text{age [year]}) + PA \times [(9.36 \times \text{weight [kg]}) + (726 \times \text{height [m]})]$
   Older women:

| Age = 75 years | Weight = 72 kg |
|---|---|
| Height = 1.74 m | PA = 1.27 |

   Estimate total caloric needs:
   (a) 1,856 kcal/day
   (b) 2,296 kcal/day
   (c) 2,657 kcal/day
   (d) 2,585 kcal/day*

6. What does the USDA suggest to improve diet quality in older people?
   (a) Decrease the physical activity
   (b) Make healthy cooking classes for older women
   (c) Increase their intakes of whole grains, dark green and orange vegetables, legumes, and milk*
7. Nutritional recommendations for older people NOT considered:
   (a) Decrease the intake of potassium to 1,500 mg
   (b) Decrease the consumption of trans-fatty acid
   (c) Increase consumption from saturated fatty acids*
8. In older people, vitamin B12 deficiency is influenced by
   (a) Difficulty in absorption of vitamin*
   (b) Low sun exposure
   (c) Non-consumption of foods containing vitamin
9. Taking antioxidants in carotenoids, vitamin C, flavonoids, and other polyphenols is important for
   (a) Decreased CVD risk*
   (b) Decreased cancer risk
   (c) Increased mortality
10. There are two forms of dietary iron: heme and non-heme. Heme iron is found in
    (a) Fortified products, including breads, cereals, and breakfast to the diet
    (b) Fruits, whole-grain breads, or whole-grain pasta
    (c) Red meats, fish, and poultry*

# Index



J.J. Robert-McComb et al. (eds.), *The Active Female: Health Issues Throughout the Lifespan*,
DOI 10.1007/978-1-4614-8884-2, © Springer Science+Business Media New York 2014