The Female Athletic Triad: Disordered Eating, Amenorrhea, and Osteoporosis

12

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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

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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

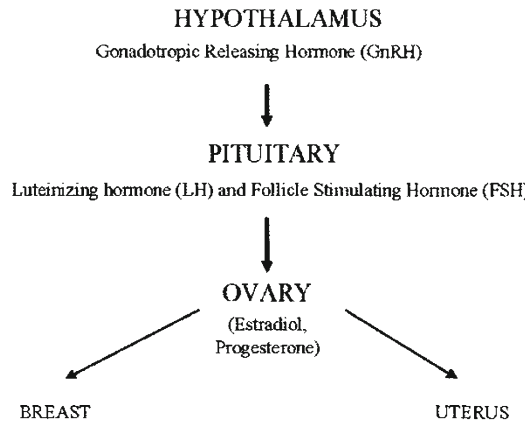

Fig. 12.1 This figure shows the primary components of the female reproductive system and the hormones that communicate between the various organs. The hormones produced by each gland are shown in parentheses

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

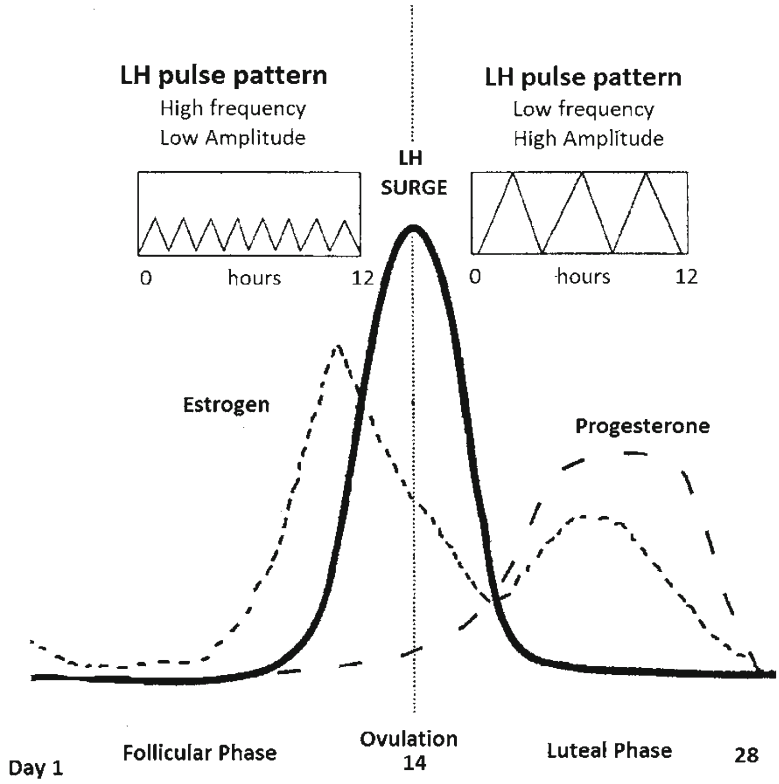

Fig. 12.2 An LH surge occurs at the time of ovulation and marks the division between the follicular phase (days 1–14) and the luteal phase (days 15–28). LH pulse pattern also changes across the menstrual cycle; pulse frequency decreases from the follicular phase (−65- to 80-min intervals) to the luteal 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.

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