Long term consequences of the female athlete triad

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A B S T R A C T

In the past 40 years, female sports participation, particularly at the high school level, has significantly increased. Physical activity in females has numerous positive benefits, including improved body image and overall health. Unfortunately, a select population of exercising females may experience symptoms related to the female athlete triad, which refers to the interrelatedness of energy availability, menstrual function, and bone mineral density. Clinically, these conditions can manifest as disordered eating behaviors, menstrual irregularity, and stress fractures. Triad symptoms are distributed along a spectrum between optimal health and disease; all of the components of the triad may not be affected simultaneously.

The female athlete triad was first identified in 1992. Since that time, a vast amount of research related to the identification, management and prevention of this condition has been published. More recently, research related to the long term effects of triad components has come into light. Women who were diagnosed with female athlete triad syndrome as adolescents and young adults in the 1990s are now in their 30s and 40s; negative long term effects of the female athlete triad, such as low bone mineral density, are now starting to manifest.

Women of all ages should be assessed for triad components during routine annual physical examinations; appropriate measures to treat any current triad components should be implemented. In addition, women in their 30s, 40s and early 50s should be screened for a history of the female athlete triad. Multidisciplinary management of these conditions is strongly recommended.

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1. Introduction

Since the passage of Title IX in 1972, the United States has seen girls’ participation in sports dramatically increase. Prior to its implementation, girls comprised 7% of high school athletes. Currently, girls comprise 42% of high school athletes [1].

Numerous benefits to female sports participation have been observed; overall, the benefits of exercise far outweigh the risks [2]. However, in 1992, an association between disordered eating, amenorrhea, and osteoporosis in athletes was identified; the condition was coined the female athlete triad (triad). The American College of Sports Medicine (ACSM) published a Position Stand on the
condition in 1997 [3] and updated the Position Stand in 2007 [2]. One of the 2007 revisions was a change in terminology. Clinicians and researchers soon realized that the terms disordered eating, amenorrhea, and osteoporosis were too restrictive; females with less severe conditions were not recognized as having the triad. Therefore, the 2007 Position Stand boasts broader definitions along a spectrum of energy availability, menstrual function, and bone mineral density. With these new definitions, more athletes are now recognized as suffering from triad components [2] (Fig. 1).

A significant amount of research on the triad at the high school level [4–9] and in the collegiate and elite female athlete populations [10–12] exists. Women first identified as having triad-related conditions in the mid–1990s are now presenting with negative long term consequences. Although no literature to date has examined females for the long term consequences of the triad in its entirety, research related to the long term effects of the individual components of the triad exists. Therefore, the purpose of this paper is to review the latest research on the long term consequences of low energy availability, menstrual irregularity, and low bone mass in female athletes.

2. Components of the female athlete triad energy availability

a. Energy availability

Energy availability is energy intake minus energy expenditure. The post-exercise energy is utilized for the regulation of bodily functions, including tissue healing, cardiovascular functioning and menstruation [2,11]. Energy availability ranges from optimal energy availability to low energy availability; low energy availability may occur intentionally or unintentionally [2] (Fig. 1). For example, some athletes may intentionally restrict caloric intake while others may fall into a negative energy balance because they are unaware of their caloric needs [13,14]. Intentional energy reduction occurs through excessive exercise, by dramatically decreasing caloric intake, or by combining both methods. Intentional caloric restriction often manifests as disordered eating (DE), which may involve restrictive eating, fasting, skipping meals, or use of diet pills, laxatives, diuretics, or binge-eating followed by purging [2,11,13,15]. Specific sub-categories of DE include anorexia nervosa (AN), bulimia nervosa (BN), and eating disorders not otherwise specified (EDNOS). These eating disorders are all characterized by a significant preoccupation with weight and shape [16]. Research suggests that, regardless of when unhealthy eating attitudes and behaviors manifest, the behaviors are formed during adolescence [17–19].

Anorexia nervosa is a serious psychiatric illness; criteria for the diagnosis of AN includes refusal to maintain a minimal body weight (less than 85% of expected), intense fear of weight gain and distorted body image [16]. Unfortunately, the outcome is poor; only 46% of patients fully recover from AN; 20% remain chronically ill [21]. The prevalence of AN is listed in Table 1.

Bulimia nervosa is characterized by periods of fasting and dieting interrupted by binge eating and a feeling of a loss of control [16]. Binging is compensated for by self-induced vomiting, laxative, or diuretic abuse. Criteria for BN requires a minimum frequency of 2 binges per week. Unlike AN, BN criteria involving binge duration and food consumption are questionable. The prevalence of BN is listed in Table 1.

Eating disorder not otherwise specified (EDNOS) is a catchall diagnosis for patients with significant features of eating disorders that do not meet the criteria for AN or BN. Although this category is considered a “residual” category, it is the most common eating disorder diagnosed. The behaviors which lead to the diagnosis of EDNOS are oftentimes difficult to determine as they are typically assessed via self-report. The prevalence of DE among various populations, determined by the use of surveys or food diaries, is listed in Table 1.

b. Menstrual function

According to the 2007 Position Stand, menstrual function occurs along a spectrum, ranging from eumenorrhea, or normal menses, to amenorrhea [2] (Fig. 1). Menstrual dysfunction, often called menstrual irregularity (MI), includes primary amenorrhea, secondary amenorrhea, and oligomenorrhea [2]. Primary amenorrhea is a delay in menarche; the defining age for primary amenorrhea is 15
Secondary amenorrhea is a cessation of menstruation for 3 consecutive months in the post-menarche female. Oligomenorrhea is menstrual cycles occurring greater than 35 days apart [2].

Determining the prevalence of MI in the adolescent population comes with challenges; MI immediately post-menarche is common. However, normalization of menstruation occurs within 2 years of menarche in 90% of females [24]. The prevalence of MI in girls >2 years post-menarche has been reported to range from 18.8 to 54% [6,7,9,22,23]. Lean build athletes and aesthetic athletes (cheerleading, pom-pon squad, dance team, diving and gymnastics) have a significantly higher prevalence of MI-26.7% and 28.2%, respectively, when compared to general high school athletes [9,22]. The prevalence of secondary amenorrhea has been reported to be as high as 65% in elite long-distance runners compared to 2–5% in the general population [25]. Although primary amenorrhea prevalence is less than 1% in the general population, the prevalence in collegiate cheerleading, diving and gymnastics athletes has been reported to be as high as 22% [14,26,27].

c. Bone mineral density
Bone mineral density (BMD) occurs across a spectrum, ranging from optimal bone health to osteoporosis [2] (Fig. 1). Osteopenia and osteoporosis are well-defined terms in post-menopausal women; osteopenia is a bone mineral density (BMD) T-score between 0.0 and -2.5, and osteoporosis is a T-score of less than -2.5 [28]. However, T-scores scores are not used in adolescent and pre-menopausal females. The International Society for Clinical Densitometry (ISCD) recommends that BMD in this population be expressed as Z-scores, as these scores compare individuals to age and sex-matched controls. Any Z-score lower than -2.0 is defined as “low bone density for chronological age” in adolescent and pre-menopausal females [28].

An athlete’s BMD reflects numerous variables, including energy availability, menstrual status, genetic composition and environmental factors. Therefore, a one-time “snapshot” of BMD in the adolescent or pre-menopausal female may not provide as much information as does changes over time [2,29]. As such, studies related to BMD in the young female population are limited [6,7]. The prevalence of low bone mass in 170 high school females was determined to be 21.8% [7]. In 80 high school female athletes, 13% had a Z-score between -0.0 and -1.9, and 3% had a Z-score of ≤ -2.0 [6]. In addition, 19% reported a history of a stress fracture. These findings are concerning; athletes participating in weightbearing sports should have BMD 12–15% higher than sedentary controls; this was not the case in this study [6].

### Table 1

Prevalence of disordered eating behaviors in various populations.

<table>
<thead>
<tr>
<th></th>
<th>Anorexia nervosa</th>
<th>Bulimia nervosa</th>
<th>Eating disorder not otherwise specified</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adolescent/teenage females</td>
<td>0.2–1.0%</td>
<td>0.3%</td>
<td>18.2–36.0%</td>
</tr>
<tr>
<td>Adult females</td>
<td>1.2–2.2%</td>
<td>1.0–2.0%</td>
<td>15.2–32.4%</td>
</tr>
<tr>
<td>Collegiate or adult elite female athletes</td>
<td>0.2–1.0%</td>
<td>0.3%</td>
<td>18.2–36.0%</td>
</tr>
</tbody>
</table>

is one of the most important factors in determining osteoporosis and fracture risk later in life [30]. These important events, unfortunately, coincide with the peak time for the development of eating disorders [20].

Adolescent eating disorders have a high rate of medical complications [16]. Adolescents with AN are reported to have lower Z-scores when compared to their healthy peers; the prevalence of Z-scores of < −1 at the spine and hip was 50% and 30%, respectively [31]. When adolescent females with AN are followed over a year, their bone mass accrual plateaued as compared to their healthy peers, who show a continued increase [32]. Females with AN who increased their body mass index (BMI) by 10% and resumed menses over a 12-month period showed improved BMD, although it remained less than controls [31,33].

When adult women with AN were examined for bone loss, those with an onset of amenorrhea before age 18 had lower BMD than those who developed amenorrhea after age 18, even after controlling for duration of amenorrhea [34]. These findings confirm the importance of adolescent bone mass accrual.

Unfortunately, bone density may not improve with weight gain and resumption of menses. In a study of 19 women with adolescent-onset AN who had completely recovered from AN for 21 years, bone density at the femur was lower than in controls; lumbar spine BMD was no different than controls [35]. These findings mirror the site-specific acquisition of bone; peak bone mass for the femur occurs during adolescence whereas lumbar spine peak bone mass is acquired later [30].

Another potential reason for site-specific gains in BMD in women who have recovered from AN is related to bone type. Because of the association between hypogonadism and decreased trabecular bone density, resumption of menses may improve bone density at trabecular-rich sites, such as the spine [34,36]. Cortical bone density is reliant upon BMI and lean body mass, both of which may remain low, even if the female has recovered from AN [35,37]. This offers a second explanation as to why cortical bone density changes are less remarkable in patients with AN, even after they have recovered [34,35,38].

When females with BN are of normal weight and have no history of AN or amenorrhea, their bone density is similar to that of controls [39]. Exercising bulimic women may have higher BMD than those who do not [40]. Other studies have refuted these findings and determined that women with BN and EDNOS have significantly lower BMD than controls [41]. In patients with AN, BN and EDNOS, all 3 subgroups had decreased BMD; the findings were worst in the EDNOS subgroup [42].

Looking more specifically at the EDNOS subgroup, adolescent female competitive runners completed questionnaires regarding eating behaviors and menstrual history and were assessed for BMD [43]. Runners with "elevated dietary restraint" – a sub-category of the eating questionnaire – had a significantly greater incidence of low BMD than runners without elevated restraint. These findings suggest that dietary restraint may be the DE behavior most associated with negative bone health in adolescent female runners [43].

In a study of 91 female distance runners between the ages of 18 and 26, Cobb et al. examined the relationship between DE, MI and BMD [10]. The authors determined that DE was associated with low BMD, even in the absence of menstrual irregularity [10].

3. Long term affects of the female athlete triad

Adolescence is a period of growth, development and maturation of numerous systems, especially the reproductive and skeletal systems. Approximately 50% of peak bone mass is acquired during adolescence; the greatest increase is between 11 and 14 years [20], Women attain >95% of their total BMD by age 18 [30]. Peak bone mass attainment is site specific; peak bone mass accrual at the femoral neck occurs by age 16, but in the lumbar spine, bone mass continues to increase into the third decade of life [30]. Bone mass acquisition involves the interaction of numerous hormones and environmental factors, including dietary habits and physical activity [30]. The amount of skeletal mass acquired during adolescence...
These findings – suboptimal Z-scores and low BMD in adolescent and young females with DE behaviors – are alarming. The majority of bone mass is accumulated in adolescence; eating disorders result in low energy availability and interfere with normal bone acquisition, which can lead to permanent deficits [16]. Adolescents with DE are at greater risk for the development of osteopenia or osteoporosis; this is associated with fracture risk in adulthood 2–7x higher than the adolescent without an eating disorder, especially in non-spine fractures [16,44]. This would correlate with previous data suggesting cortical bone recovery loses than trabecular bone [34,35,38].

Numerous factors contribute to the reduction in bone mass seen in adolescents with DE and are summarized in Table 2. The most important factor counteracting bone loss in adolescents with DE is normalization of body weight [16].

The rate of suicide and suicide attempts is increased in individuals with eating disorders. Approximately 10–20% of patients with AN report a suicide attempt; approximately 25–35% of patients with BN report attempted suicide [16]. Approximately 5–6% of women with AN suffer a premature death [16]. The mortality ratios for women with AN, BN, and EDNOS are 5.86, 1.93, and 1.92, respectively [21].

Menstrual function, the second triad component, has been examined for its long-term effects. Women with previous secondary amenorrhea had significantly lower BMD in the pelvis and lumbar spine than those with previous oligomenorrhea. The most important counteraction at follow-up high physical activity and BMI ≥ 22 [45]. Of 577 adolescent females aged 14–19 years who were examined, those with oligomenorrhea at initial presentation were almost 3 times more likely to have oligomenorrhea at follow-up [46].

As previously reported, a big concern related to adolescent low bone mass is the risk these individuals run in attaining peak BMD in adulthood. Barrack et al. [47] examined 40 female adolescent runners with low bone mass without an eating disorder; subjects were followed over a 3-year period. The authors discovered that approximately 90% of girls with low bone mass at baseline also had low bone mass at the 3-yr follow-up. Factors including higher training levels, older age and older age at menarche were negatively associated with the change in BMD. The authors concluded that “catching up” bone mass may be difficult. This study highlights factors other than DE and menstruation as contributing to low BMD [47].

In a similar study, bone mass accrual among a cross-sectional sample of 93 female adolescent runners and 90 girls participating in a non-endurance running sport was determined [5]. Although bone mass rose at all sites in the non-runners between the ages of 13–14 years and 17–18 years, no such increase was noted in the runners. Runners exhibited significantly lower Z-scores among the older (16–18 years) but not younger (13–15 years) age groups. These findings suggest that the runners, in contrast to the non-runners, exhibited a suppressed bone accrual pattern. This supports the notion that female adolescent endurance runners may be at risk for inadequate bone mass gains and thus a low peak BMD [5].

Because of negative long term consequences of sustaining a musculoskeletal injury at a younger age, and the increased prevalence of injuries in females as compared to males, research has examined the relationship between triad components and injury [8]. Of the 3 components, menstrual dysfunction exhibited the strongest relationship to injury [8]. In a study of 249 high school female athletes, those who reported MI sustained a higher percentage of severe injuries than did athletes who reported normal menses [48]. The relationship among DE, MI, low BMD and musculoskeletal injury among 163 female high school athletes was assessed; all three triad components were associated with musculoskeletal injuries [8]. These studies highlight the association of triad components and musculoskeletal injury. The long-term effects of sustaining an injury can be devastating; it may require the athlete to modify activity or cease exercising altogether. This can lead to negative systemic effects such as obesity, hypertension and diabetes.

4. Management of the long term affects of the female athlete triad

All of the components of the female athlete triad-energy availability, menstrual function, and bone density–appear to have a negative long-term effect on bone. Negative energy balance and poor diet cause a decrease in calcium and vitamin D, both essential for bone acquisition. With a lack of energy, numerous bodily systems, including the reproductive system, go into “shut-down” mode. Adolescent secondary amenorrhea and oligomenorrhea with or without an eating disorder-causes hypogonadism, or low estrogen levels, which leads to decreased bone acquisition. Decreased bone acquisition during the critical years of adolescence precludes the female from filling her “bone bank”; once “withdraws” from the bone bank start in the mid- to late-20s, low bone mass for chronological age will begin to manifest in the pre-menopausal female; the condition will only worsen postmenopause.

As such, it becomes critical that all healthcare professionals perform a thorough history when working with female athletes of all ages. A thorough discussion of past eating behaviors, menstrual history and previous DXA scans are critical steps in determining the appropriate course of care. It is imperative that the questioning go back as far as adolescence, as research has clearly demonstrated how critical this time is in the development of eating behaviors, menstrual function and bone acquisition. Any female who is identified as having current signs and symptoms of one female athlete triad component should be examined for the other components and treated appropriately. Any female, regardless of age, who reports altered eating behaviors from adolescence to present day, reports menstrual irregularity anytime throughout her reproductive lifetime, or reports stress fracture(s) or prolonged musculoskeletal injury, should undergo a more detailed examination. A 3-day food inventory, looking for adequate calcium and vitamin D intake, should be performed. A complete pelvic examination should also be performed; any pathologic findings should be addressed. Lastly, any women reporting a history of disordered eating behaviors, menstrual irregularity (currently or in the past) or >1 stress fracture should have their BMD assessed via DXA scan. Treatment strategies, such as diet modification, addressing menstrual dysfunction, promotion of impact and weightbearing exercise should be implemented. Most importantly, changes in bone occur slowly; as such, repeat assessments of BMD should be repeated every 6-12 months. Further changes in the treatment regimen should reflect findings in the changes in bone.

5. Conclusions

The female athlete triad exists in all ages. Since the triad was first identified in 1992, women are now seeing the negative
long term consequences of suffering from triad components. Research suggests that disordered eating behaviors cause low energy availability, resulting in shut down of the reproductive and skeletal systems. Menstrual irregularity is associated with hypoestrogenism, which affects bone mass accrual. Lack of bone mass accrual during the critical time of adolescence is typically not reversed; pre-menopausal and post-menopausal women will “withdraw” bone from an already-depleted bone bank. The impact of these conditions is medically significant, as a lack of bone mass accrual has devastating effects. Any female who reports a history of altered eating behaviors, menstrual irregularity, stress fracture(s) or prolonged musculoskeletal injury should undergo a more detailed examination. Treatment strategies include nutrition counseling, diet modification, addressing menstrual dysfunction and promotion of weightbearing exercise. Changes in bone occur slowly; therefore, repeat assessments of BMD are warranted.

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