

Chapter 11

The Female Athlete Triad



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Introduction

The female athlete triad (Triad) is comprised of three interrelated conditions: low energy availability (with or without disordered eating), menstrual dysfunction, and low bone mineral density. Low energy availability contributes to both menstrual dysfunction and low bone mineral density, and menstrual dysfunction also contributes to low bone mineral density. As illustrated in Fig. 11.1, these three conditions occur on a spectrum ranging from optimal health (adequate energy availability, eumenorrhea, and normal bone mineral density) to the end-stage that includes eating disorder, amenorrhea, and osteoporosis. Athletes with the female athlete triad can present with these three conditions in different stages at different periods of time, and diagnosis of the Triad does not require simultaneous clinical manifestations of all three conditions [1, 2].

It is crucial that sports medicine physicians are comfortable diagnosing and treating the female athlete triad. If untreated, the consequences of the Triad can be devastating, and include compromised reproductive health, increased rates of bone stress injuries, and other soft tissue musculoskeletal injuries, osteoporosis, and eating disorders. Early recognition and prompt treatment may help patients avoid the end-stage outcomes of the Triad.

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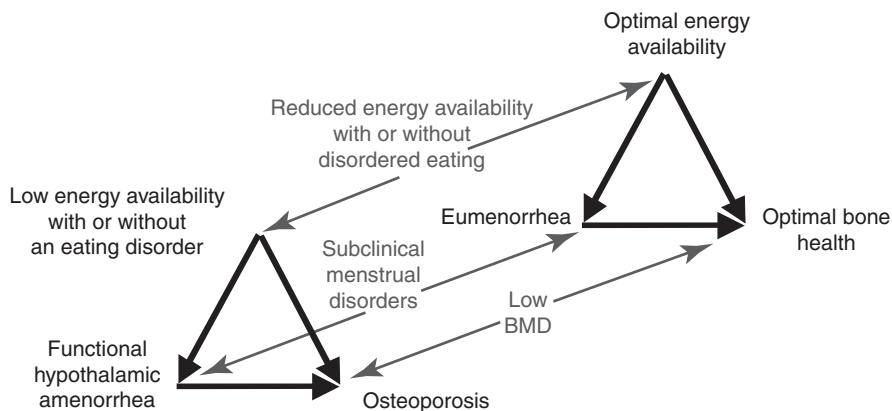


Fig. 11.1 The spectrum of the female athlete triad. The three inter-related components of the Triad are energy availability, menstrual status, and bone health. Energy availability affects menstrual status, and both energy availability and menstrual status affect bone health. (Reprinted with permission from Wolters Kluwer: *Medicine & Science in Sport & Exercise* [1])

Terminology

In 1992 the American College of Sports Medicine (ACSM) convened a panel of experts, which resulted in the initial description of the Triad [3] and subsequently in the 1997 ACSM Position Stand on the Female Athlete Triad [4]. They defined the Triad as a syndrome affecting active girls and women, comprised of the interrelated conditions of disordered eating, amenorrhea, and osteoporosis. Since this time, there has been a significant body of research dedicated to understanding the female athlete triad, its diagnosis, its management, and its consequences. This has resulted in two major updates. In 2007, the ACSM published an updated Position Stand on the Female Athlete Triad, re-defining the Triad as low energy availability, menstrual dysfunction, and decreased bone mineral density (BMD) [1]. An important feature of this update was the recognition that each component of the Triad may exist on a spectrum ranging from healthy, to subclinical or mild disturbances, to end-stage consequences including eating disorders, amenorrhea, and osteoporosis. The 2007 Position Stand illustrated the Triad as a prism (see Fig. 11.1), where athletes could move along the different axes of the prism at different rates, highlighting the importance of early recognition and treatment. In 2014, The Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad was published to provide further guidance on clinical management of the Triad and return to play decision making [2]. As our understanding of the female athlete triad expands, we have also observed the existence of a similar condition in males [5, 6] – most recently termed the male athlete triad [7]. This is comprised of low energy availability, hypogonadism, and low bone mineral density. The male athlete triad remains a promising area for future research and is further detailed below in section “Return-to-Play Guidelines.”

In 2014, the International Olympic Committee (IOC) published a paper on Relative Energy Deficiency in Sport (RED-S) [5], which was followed by an update in 2018 [8]. These papers are based upon research on the female athlete triad, and are similar in that they describe the impacts of low energy availability (EA) on athlete well-being. The RED-S model depicts low EA as the “hub” at the center of a wheel. Each “spoke” of the wheel illustrates the downstream effects of low EA on multiple organ systems, including those involved in the Triad (menstrual function and bone health), as well as several others (immunologic, cardiovascular, etc.). Although research does agree that low EA is the causative mechanism behind the menstrual dysfunction and impaired bone health seen in the Triad, there is not yet sufficient scientific evidence to directly link low EA to each of the other “spokes” of the RED-S model. As such, we will continue to use the more researched term “the female athlete triad.” Ongoing research on the health and performance effects of low EA is welcomed and encouraged.

Epidemiology

Unfortunately, the female athlete triad is very common. Because individual athletes may present at different points on each of the three spectra (low EA, menstrual dysfunction, and low BMD), it may be easier to quantify the prevalence of each of the individual components of the Triad. In one study of 425 female collegiate athletes at seven US universities, 3.3% reported a diagnosis of anorexia and 2.3% reported a diagnosis of bulimia nervosa; however, the Eating Attitudes Test (EAT-26) identified “at-risk” behaviors in 15.2% of athletes, and the Eating Disorder Inventory Body Dissatisfaction Subscale (EDI-BD) identified 32.4% of athletes as having “at risk” behaviors for eating disorders [9]. Eating disorders are also more common in athletes than in the general population. In one large study of 1620 athletes and 1696 controls, 20% of female athletes met criteria for an eating disorder compared to 9% of controls [10]. Even in the absence of a formal eating disorder, athletes also demonstrate a high frequency of disordered eating behaviors [11, 12]. Studies have shown that in weight class sports, as many as 70% of athletes may rely on dieting and disordered eating behaviors to make weight [13].

Menstrual irregularities have been reported in 31% of athletes not using oral contraceptives [9]. Other studies have found that the prevalence of secondary amenorrhea may be as high as 65% in elite distance runners [14], and 79% in elite dancers [15]. A Croatian study found rates of secondary amenorrhea were three times higher in athletes when compared to a non-athlete control group [14].

Prevalence of low bone mass ranges from 22% to 50%, and prevalence of osteoporosis ranges from 0% to 13% in athletes [16]. However, it should be noted that the World Health Organization definitions of low bone mass (osteopenia) and osteoporosis in postmenopausal women were used at the time of this study, instead of the current definitions for premenopausal women. Therefore T-scores were used in young women instead of Z-scores. One review found that while 16–60% of athletes

had at least one component of the Triad, 2.7–27% had two components, and 0–15.9% of athletes exhibited all three components of the Triad [17].

The incidence of the Triad remains highest in “lean sports” which include aesthetic sports (synchronized swimming, figure skating), sports with weight classes (wrestling, lightweight rowing), and endurance sports (distance running, cycling). In a population of high school athletes, the prevalence of both disordered eating and menstrual irregularity was 15.4% in aesthetic sports, 10.1% in endurance sports, and 7.6% in team/anaerobic sports [18]. A Norwegian study found similar trends – the prevalence of eating disorders in aesthetic sports was 42%, in endurance sports it was 24%, in technical sports it was 17%, and in ballgame sports it was 16% [10]. In a similar study on elite German athletes, the prevalence of eating disorder was 17% in aesthetic sports, 2% in ball sports, and 2% in non-athletes [19].

Although the Triad can occur in all races, the majority of research to date has been performed in Caucasian populations, and further research is needed on the influence of race on the Triad.

Low Energy Availability

Low energy availability (EA) occurs when an individual’s nutritional intake is not sufficient to meet their body’s energy expenditure, including the energy expended during exercise. This is defined as: energy intake (kcal) minus energy expenditure (kcal) divided by the kg of fat free mass. Research has suggested that optimal physiologic function occurs at an energy availability of 45 kcal/kg FFM. Energy availabilities of 30 kcal/kg FFM and less have been associated with abnormal metabolic markers and Triad symptoms in female athletes.

Low EA affects GnRH, which decreases LH pulsatility and estrogen levels [20]. The hypoestrogenic state contributes to menstrual dysfunction and decreases in BMD. In addition, low EA can also decrease resting energy expenditure, decrease total T3, increase Ghrelin, increase PYY, decrease leptin, decrease insulin-like growth factor (ILGF-1), and increase cortisol [21, 20]. Even in athletes who have adequate EA when averaged overall, periods of low EA throughout the day can contribute to some of the above hormonal changes [20].

Although low EA is easily defined, it can be challenging to calculate in clinical settings. Energy intake can be assessed via a food log or food frequency questionnaire. Energy expenditure can be estimated via metabolic equivalents of a task, heart rate monitoring, or accelerometer. Lastly, fat free mass can be calculated via dual-energy x-ray absorptiometry (DXA), air displacement plethysmography, bioelectrical impedance, or skin fold caliper measurement [22]. Many clinicians will use low body mass index (BMI) as an indicator of low energy availability. BMI is defined as a patient’s weight (in kg) divided by their height (in meters) squared. Although normal ranges for BMI can vary considerably due to differences in body composition and varying sport demands, a BMI of less than 18.5 is generally regarded as low. Caution should be used when relying solely on BMI as an indicator of energy

availability however, since evidence suggests that the body responds to chronic low EA by decreasing the resting metabolic rate [20]. Although not a direct measurement of EA, screening for restrictive eating patterns may identify red flags which suggest low EA. These behaviors might include highly restrictive diets, avoiding certain food groups completely, or skipping meals. In some patients who have developed an eating disorder, physical exam findings may be present, such as lanugo, Russell's sign (callus on finger from self-induced emesis), dental erosion, or large parotid glands. Given the challenges of identifying low energy availability in the clinical setting, it is very helpful to work with a sports dietitian whenever possible.

It is also important to note that low energy availability may be intentional or unintentional. Unintentional low energy availability usually results from a lack of awareness about the body's nutritional needs, or from a recent increase in training volume without a compensatory increase in dietary intake. A classic example can be seen in the freshman collegiate athlete who transitions to a more intense training schedule without making the necessary dietary changes their body needs in order to compensate for their higher level of training.

Intentional low energy availability may occur due to disordered eating (DE) behaviors, or if more severe, may meet criteria for an eating disorder (ED). According to the DSM-V criteria, anorexia nervosa is defined as persistent restriction of energy intake leading to significantly low body weight ("less than minimally normal in adults or less than expected weight in children and adolescents"), fear of gaining weight or persistent behavior that interferes with weight gain despite being at a low weight, and a disturbance in the way one's body weight/shape is experienced or a persistent lack of awareness of the seriousness of the low body weight [23]. Changes from the DSM-IV include removing the diagnostic criteria of having amenorrhea, and also adding that clinicians may infer fear of gaining weight or of body image disturbance from patient behaviors, even if the patient does not admit to these thoughts. Bulimia nervosa is defined as binge episodes and compensatory behaviors which occur on average at least once per week for 3 months [23]. This DSM-V diagnosis is also a change from the prior edition, as it reduces the minimum frequency of bingeing episodes required to meet diagnostic criteria. Research demonstrates that eating disorders are more common in athletes than in non-athletes [24]. Unfortunately eating disorders have one of the highest mortality rates of all mental health conditions, and also have a wide range of severe of health complications which can affect nearly every organ system in the body [24]. For this reason, it is important to diagnose eating disorders as soon as possible in order to facilitate prompt access to treatment.

Many athletes exhibit concerning behaviors or thought patterns around food, without meeting full diagnostic criteria for one of the above eating disorders. There are numerous disordered eating behaviors which fall into this category, including over-exercising, making meals contingent on exercise, purging behaviors (such as self-induced vomiting, or the use of laxatives and diuretics), avoiding certain food groups completely, intentionally restricting intake, etc. Because patients with eating disorders often deny or try to hide their condition, and eating disorders are associated with such significant health consequences, clinicians who identify disordered

eating behaviors should screen athletes for eating disorders, and should have a low threshold to refer them for further work-up or treatment as indicated.

DE and ED tend to be more common in “lean sports,” and part of this may be propagated by the belief that a lower body weight will improve athletic success. However, one large study which relied on self-report via online questionnaire found that athletes with low EA were more likely to report decreased training response, decreased coordination, decreased concentration, impaired judgment, irritability, depression, and decreased endurance performance [25]. Another study on ten elite junior swimmers found decreased performance results associated with ovarian suppression due to low EA [26]. A population of elite level rowers who increased their training volume without increasing their energy intake demonstrated reductions in body mass and fat mass but also showed decreased performance in timed 5K racing [27]. Similarly, in a study on elite endurance athletes, Tornberg et al. found decreased neuromuscular performance in amenorrheic athletes when compared to eumenorrheic athletes [28].

As will be detailed below, low EA predisposes athletes to bony injuries such as stress fractures, which further impair training and performance. However, evidence also suggests that high school athletes who reported some disordered eating were twice as likely to sustain a musculoskeletal injury in general [18]. In addition, low EA is often associated with low iron levels, which can further reduce endurance performance [29, 22].

Menstrual Dysfunction

Athletes should be regularly screened for menstrual dysfunction, because this is associated with significant short-term and long-term consequences for bone health. Menstrual dysfunction may take several different forms, including delayed menarche, oligomenorrhea, or amenorrhea. Delayed menarche is generally defined as starting one’s period at age 15 or older. Oligomenorrhea refers to menstrual cycles of 35 days or longer, or less than 9 menses per year. Secondary amenorrhea is diagnosed when it has been greater than 3 months since a patient’s last menstrual cycle. Primary amenorrhea refers to a patient who has not yet undergone menarche. All of these forms of menstrual dysfunction have been associated with increased incidence of bone stress injuries (BSI) and low BMD [30]. Furthermore, athletes who reported menstrual irregularities (oligomenorrhea/amenorrhea) may be up to three times as likely to sustain a musculoskeletal injury [31]. Even in patients who do have regular menstrual cycles, there may be subclinical menstrual disturbances such as luteal phase defects and anovulatory cycles, which may only be appreciated on detailed hormonal panels [20]. One study in recreational runners found the prevalence of luteal phase defects to be as high as 79% over a 3-month period [32].

Patients with functional hypothalamic amenorrhea resulting from low EA demonstrate decreased LH pulsatility, decreased FSH, decreased estrogen, and decreased progesterone [20]. Estrogen plays a key role in balancing bone formation and bone resorption and also inhibits bone turnover.

Table 11.1 Suggested studies for the work-up of patients with secondary amenorrhea

Suggested studies for the work-up of secondary amenorrhea
Luteinizing hormone (LH)
Follicle stimulating hormone (FSH)
Estradiol
Thyroid stimulating hormone (TSH)
Free T4
Total T3
Dehydroepiandrosterone (DHEA)
Testosterone (free and total)
Pregnancy test
Prolactin

All female athletes should be screened for menstrual dysfunction at their participation physical exams, and more frequently as clinically indicated. When screening for menstrual irregularities, it is important to inquire about any form of contraception that the athlete is using, as hormonal forms of contraception may mask or artificially induce menses. For example, an athlete with a history of amenorrhea may have regular periods if taking an oral contraceptive pill; however, this does not mean that she would be cycling regularly on her own, or that her bone mineral density is adequate.

If amenorrhea is identified, then further work-up is indicated in order to identify the cause of amenorrhea. While one of the most common causes of amenorrhea in the athlete is functional hypothalamic amenorrhea due to low EA, it is important to rule out other potential causes of amenorrhea including polycystic ovarian syndrome (PCOS), thyroid disease, prolactinemia, premature ovarian failure, or pregnancy. A suggested list of initial diagnostic studies for the work-up of secondary amenorrhea can be found in Table 11.1 and may need to be modified based on individual clinical presentations. Young female athletes with primary amenorrhea may require additional work-up including imaging to rule out structural abnormalities [2].

Low Bone Mineral Density

Low EA has been associated with low BMD (defined by low Z-scores) and also with abnormal bone turnover markers [20]. Triad-related effects on bone health are primarily due to two mechanisms: estrogen-dependent and estrogen-independent. In the estrogen-dependent mechanism, hypoestrogenism associated with amenorrhea causes upregulation of bone resorption and promotes bone loss by inducing osteoclastogenesis. The estrogen-independent mechanism is energy-dependent and involves metabolic hormone adaptations to low EA such as IGF-1, leptin, and T3. IGF-1 usually stimulates osteoblastogenesis and bone formation but is impaired in low EA states. Similarly, leptin is also impaired in low EA states, and it normally plays a role in osteoblast proliferation. T3 (also low in low EA states) normally stimulates osteoblast proliferation and differentiation and promotes bone formation [20].

Estrogen acts on osteoblasts and osteoclasts via a direct receptor-mediated fashion and has indirect effects on calcitonin, parathyroid hormone, cytokines, and growth factors [33].

Low bone mineral density (BMD) is very concerning in athletes and is associated with an increased incidence of bone stress injuries. Even excluding BSI, high school athletes with a BMD Z-score < -1.0 were 3.6 times more likely to incur a musculoskeletal injury [31]. Furthermore, low BMD predisposes athletes to BSIs in more high-risk areas, including those in trabecular bone, which are associated with prolonged recovery and complications such as non-union. A study in collegiate runners found that lower BMD was associated with prolonged return-to-play times for athletes with a BSI and also that athletes with multiple triad risk factors were more likely to have a BSI in a high-risk location like the sacrum or femoral neck [34]. Similarly, Barrack et al. found that patients with multiple Triad risk factors had increasing incidence of BSIs [35].

In addition to decreased BMD, newer modalities such as HR-pQCT suggest that menstrual status affects the microstructure of bone. Ackerman et al. found greater strength parameters (greater stiffness and failure load) at the tibia in eumenorrheic athletes, but amenorrheic athletes lost this effect when compared to non-athletes. Furthermore, the bone strength parameters seen in the amenorrheic athletes at non-weight-bearing sites such as the distal radius were even lower than those seen in non-athletes [36].

Adequate BMD is also of great concern because many patients with the Triad present during periods of peak bone accrual. If an individual is expected to reach their maximal BMD by age 30, then compromised bone health in one's 20s may significantly increase the risk of osteoporosis. Osteoporosis is an important health concern in the United States, where 52% of adults over age 50 have low bone mass at the femoral neck or lumbar spine and 1.5 million people sustain an osteoporotic fracture each year [33].

In order to identify athletes with low BMD, it may be helpful to obtain a DXA scan. DXA scans should be obtained on any athlete with one or more "high-risk" Triad risk factors, as defined by the 2014 Triad Consensus Statement: history of an eating disorder; BMI < 17.5 , $< 85\%$ of estimated weight, or weight loss $> 10\%$ in 1 month; menarche at age 16 or older; current or history of less than 6 menses within 12 months; two prior BSI, one high risk BSI, or a low-energy non-traumatic fracture; or prior Z-score < -2.0 . In addition, athletes with two or more "moderate-risk" Triad risk factors should also have a DXA. "Moderate-risk" factors include current or history of disordered eating for > 6 months; BMI between 17.5 and 18.5, $< 90\%$ estimated weight, or recent weight loss of 5–10% in 1 month; menarche between age 15–16; current or history of 6–8 menses over 12 months; one prior BSI; and prior Z-score between -1.0 and -2.0 . In addition, clinicians should consider screening athletes with one or more traumatic fractures in the setting of other Triad risk factors, or athletes on medications which may affect bone health (such as Depo-Provera or oral prednisone). In athletes found to have low BMD, DXA scans should be repeated every 1–2 years to monitor response to treatment. For premenopausal women, Z-scores should be used, as these reflect a comparison to age-matched

controls [2]. The American College of Sports Medicine (ACSM) defines low BMD or BMC to be a Z-score of <-1.0 in females engaged in weight-bearing sports [1].

Screening

All athletes should be screened for the Triad during their preparticipation physical examination (PPE). Screening questions for the Triad are included in the PPE Monograph [37], and additional questions are suggested in the 2014 Female Athlete Triad Consensus Statement [2]. Furthermore, the menstrual cycle can be used as a vital sign at the PPE, in order to help identify athletes with the Triad [38]. If screening identifies any one component of the Triad, thorough screening for the other two components is strongly recommended [20]. The 2014 Female Athlete Triad Consensus Statement also provided a cumulative risk assessment (CRA) tool intended to help physicians quantify a female athlete’s risk level for bone stress injury and poor bone health (see Fig. 11.2) [2]. This is discussed further in section “Follow Up.” The CRA may be used at the time of the PPE, or when making return-to-play decisions [39, 40].

Risk factors	Magnitude of risk		
	Low risk = 0 points each	Moderate risk = 1 point each	High risk = 2 points each
<i>Low EA with or without DE/ED</i>	<input type="checkbox"/> No dietary restriction	<input type="checkbox"/> Some dietary restriction‡; current/past history of DE;	<input type="checkbox"/> Meets DSM-V criteria for ED*
<i>Low BMI</i>	<input type="checkbox"/> BMI ≥ 18.5 or ≥ 90% EW** or weight stable	<input type="checkbox"/> BMI 17.5 < 18.5 or < 90% EW or 5 to < 10% weight loss/month	<input type="checkbox"/> BMI ≤ 17.5 or < 85% EW or ≥ 10% weight loss/month
<i>Delayed menarche</i>	<input type="checkbox"/> Menarche < 15 years	<input type="checkbox"/> Menarche 15 to < 16 years	<input type="checkbox"/> Menarche ≥ 16 years
<i>Oligomenorrhea and/or Amenorrhea</i>	<input type="checkbox"/> > 9 menses in 12 months*	<input type="checkbox"/> 6-9 menses in 12 months*	<input type="checkbox"/> < 6 menses in 12 months*
<i>Low BMD</i>	<input type="checkbox"/> Z-score ≥ -1.0	<input type="checkbox"/> Z-score -1.0*** < -2.0	<input type="checkbox"/> Z-score ≤ -2.0
<i>Stress reaction/fracture</i>	<input type="checkbox"/> None	<input type="checkbox"/> 1	<input type="checkbox"/> ≥ 2; ≥ 1 high risk or of trabecular bone sites†
Cumulative Risk (total each column, then add for total score)	___ points +	___ points +	___ points = ___ Total Score

Fig. 11.2 The female athlete triad cumulative risk assessment tool. The cumulative risk assessment provides an objective method of determining an athlete’s risk using risk stratification and evidence-based risk factors for the Female Athlete Triad. This assessment is then used to determine an athlete’s clearance for sport participation (see Fig. 11.3). ‡Some dietary restriction as evidenced by self-report or low/inadequate energy intake on diet logs; *current or past history; ** ≥90% EW; absolute BMI cut-offs should not be used for adolescents. ***Weight-bearing sport; † high-risk skeletal sites associated with low BMD and delay in return to play in athletes with one or more components of the Triad include stress reaction/fracture of trabecular sites (femoral neck, sacrum, pelvis). BMD bone mineral density, BMI body mass index, DE disordered eating, EA energy availability, EW expected weight, ED eating disorder. (Reprinted with permission from BJSM [2])

For many athletes with the Triad, their initial presenting symptom is a bone stress injury, so all athletes with stress reactions and stress fractures should also be thoroughly screened for the Triad.

Treatment

Treatment of the female athlete triad is best accomplished with a multidisciplinary team that includes the physician, sports dietitian, athletic trainer, and mental health provider (as indicated), and otherwise as available resources may dictate. In many cases, patients may require additional consultants based on their individual presentations and complications. Good communication between members of the treatment team is essential to success. In cases involving disordered eating or eating disorders, the athlete often exhibits reluctance to “buy in” to the treatment plan. In such situations, a medical contract may be very helpful. This serves to clarify expectations for the athlete and to describe the conditions necessary for ongoing sport participation. The use of contracts is highly individual and must be tailored to the unique circumstances of each patient. An example contract is provided in the 2014 Female Athlete Triad Consensus Statement [2].

Since the Triad is the result of low EA, treatment of low EA is crucial to success. This is accomplished either through an increase in nutritional intake, a decrease in training volume, or a combination of both. As mentioned above, this is best done with the assistance of a registered sports dietitian, who can provide tailored guidance to the athlete, and also continue to provide an assessment of energy availability. There is robust evidence that restoration or normalization of body weight is associated with resumption of menses and improvements in BMD [2]. Some athletes may need to increase caloric intake, but other athletes may benefit from adjusting the timing of meals and snacks, or altering the content of meals rather than the volume. If an athlete has been diagnosed with an eating disorder, their treatment team should include a mental health provider, as this is essential to treatment. For patients with eating disorders, providing nutritional advice in isolation is not likely to result in any clinical improvement. Most athletes with disordered eating behaviors would also benefit from seeing a mental health provider, in order to help treat dysfunctional attitudes towards food and body image concerns.

For patients with menstrual dysfunction, the treatment goal is to regain normal menses. As mentioned above, all patients with amenorrhea should undergo work-up to rule out alternative causes of amenorrhea (such as thyroid dysfunction), which would require different treatment approaches. For patients who have a laboratory work-up consistent with functional hypothalamic amenorrhea, the first-line treatment is to improve energy availability (see above). Although oral contraceptive pills may be indicated for patients who require protection against pregnancy, they are not indicated to treat menstrual dysfunction. Taking oral contraceptive pills may result in resumption of menses, but this does not correlate with the return of spontaneous menses or improvements in BMD, and may provide a false sense of reassurance

[41]. The lack of benefit on BMD has been associated with first-pass effects of hepatic metabolism on IGF-1 [2]. In cases of hypothalamic amenorrhea, which persists for more than 1 year, despite non-pharmacologic treatment, treatment with transdermal estrogen (which bypasses hepatic metabolism) and cyclic oral progesterone may be considered [2, 33]. It is important to note, however, that transdermal estrogen and cyclic progesterone have not been proven to prevent pregnancy, so patients should be counseled that this is not adequate for contraception.

The treatment of low BMD should also focus on improving EA. Monitoring for the spontaneous return of regular menses can serve as a positive clinical indication that factors which influence bone health are improving.

If low BMD is identified, patients should optimize their intake of calcium and vitamin D in order to provide the important building blocks for bone formation and remodeling. One study of female navy recruits found that the incidence of BSI was reduced by as much as 20% with calcium and vitamin D supplementation [42]. Another study found that higher intakes of calcium, skim milk, and dairy products were all associated with lower rates of stress fractures in active women. For every additional cup of skim milk consumed per day, there was a 62% reduction in stress fracture incidence [43]. Kelsey et al. also found that stress fractures were more common in female runners with low dietary calcium intake [44]. Per Institute of Medicine (IOM) guidelines, the Recommended Dietary Allowance (RDA) of calcium ranges from 1000 mg to 1300 mg per day for adults, with higher values recommended for adolescents and the elderly [45]. It is recommended that whenever possible, the majority of a patient's calcium intake should be from their diet, rather than supplements. This is particularly important since some studies have suggested increased incidence of kidney stones and cardiovascular disease in patients with high doses of oral calcium supplementation [46, 47].

Per IOM guidelines, the RDA for vitamin D is 600 international units (IU) per day for adolescents, pregnant or lactating women, and adults up to age 70. Adults older than age 70 have an RDA of 800 IU per day [45]. In patients who have low BMD or a strong history of bone stress injuries, it is recommended to check a vitamin D-25(OH) level. If vitamin D deficiency or insufficiency is identified, then patients may require higher levels of vitamin D supplementation. One study of professional football players found increased incidence of fractures in patients with vitamin D deficiency, and possibly decreased performance [48]. Another study in competitive distance runners found that even among runners with normal vitamin D levels, a higher vitamin D level in those with a history of bone stress injury was associated with less time off from running [49]. However, it is worth noting that at very high levels of vitamin D (such as may result from taking daily doses of 10,000 to 50,000 IU for extended periods of time), vitamin D intoxication may be seen [45]. Vitamin D intoxication may include hypercalcemia, hypercalciuria, vascular and tissue calcifications, renal complications, and heart arrhythmias [45]. One study examined the effects of a single annual high dose of vitamin D (500,000 IU) and actually found increased rates of falls and fractures in the elderly [50].

The specific sport played also affects BMD. Sports with multi-directional loading and higher impact, such as basketball, volleyball, or soccer, have been

associated with improved bone health. One study demonstrated that runners who played a ball sport in their childhood had a 50% lower risk of stress fracture [51]. In another study, athletes in high-impact sports had a 3–22% higher BMD compared to athletes in low-impact and moderate-impact sports [52]. Some low-impact sports, such as swimming or cycling, may result in decreased BMD, while running (repetitive impact but without multidirectional loading) seems to have a relatively neutral effect on BMD. This evidence supports the health benefits of being a multi-sport athlete, and demonstrates some of the potential dangers of early sports specialization.

Pharmacologic measures should be considered as a second-line option only when there is a lack of response to non-pharmacologic treatment for at least a year, and if new fractures occur during non-pharmacologic management [2]. As mentioned above, pharmacologic treatment may include transdermal estrogen with cyclic oral progesterone. Bisphosphonates may be considered in unique cases in consultation with an endocrinologist, but are not currently FDA-approved for treatment in premenopausal women. Furthermore, they are known to be teratogenic, which is particularly concerning in reproductive age women, given the long half-life of bisphosphonates. Specific criteria for the use of pharmacologic measures can be found in the 2014 Female Athlete Triad Consensus Statement [2].

Follow-up

When treating low BMD, it is advantageous to obtain follow-up DXA scans at 1–2 year intervals to monitor for improvements in bone health over time. Time to restoration of menses is highly variable, but in some cases, it may take longer than a year of non-pharmacologic treatment [53]. Furthermore, one study found that resumption of menses took longer in athletes who had amenorrhea for >8 months, compared to those who had amenorrhea <8 months [54]. It is also concerning that although restoration of adequate EA and resumption of menses results in improvements in BMD, it is not clear whether patients can return to normal BMD compared to healthy controls [55, 56]. This highlights the importance of early recognition and prompt treatment of the Triad to minimize long-term health consequences.

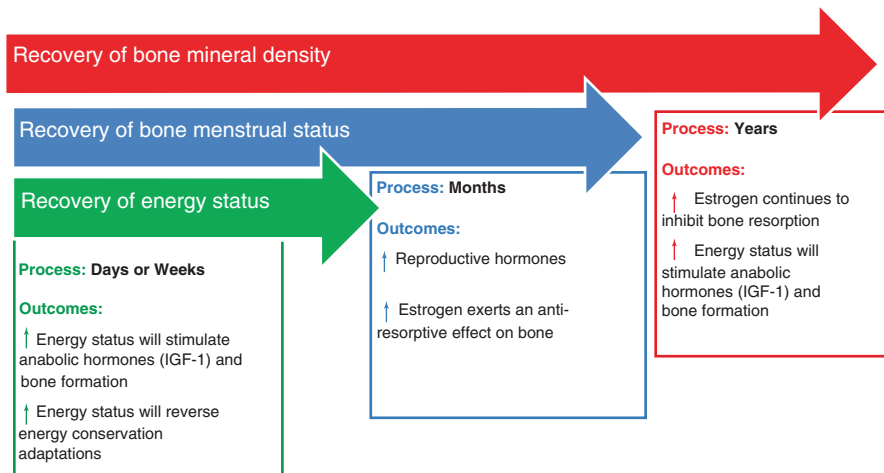
Return-to-Play Guidelines

The 2014 Female Athlete Triad Consensus Statement provided a cumulative risk assessment (CRA) tool to help clinicians quantify an athlete's Triad risk factors (Fig. 11.2) [2]. This tool may be implemented at the time of an athlete's annual PPE, in order to quantify their risk level and connect the athlete with resources to improve their bone health and reduce their risk of injury. In addition, it may also be used when an athlete has been diagnosed with a BSI or with the Triad, as a tool to help

	Cumulative risk score*	Low risk	Moderate risk	High risk
Full clearance	0 – 1 point	<input type="checkbox"/>		
Provisional/limited clearance	2 – 5 points		<input type="checkbox"/> Provisional clearance	
			<input type="checkbox"/> Limited clearance	
Restricted from training and competition	≥ 6 points			<input type="checkbox"/> Restricted from training/competition-provisional <input type="checkbox"/> Disqualified

Fig. 11.3 The female athlete triad clearance and return-to-play guidelines based on risk assessment score. The cumulative risk score is calculated using evidence-based risk factors displayed in Fig. 11.2. Athletes have differing clearance recommendations based on their risk assessment scores. *Cumulative Risk Score determined by summing the score of each risk factor (low, moderate, high risk) from the Cumulative Risk Assessment (Fig. 11.2). Clearance status for athletes who are moderate-to-high risk for the Triad: provisional clearance—clearance determined from risk stratification at time of evaluation (with possibility for status to change over time depending on athlete’s clinical progress); limited clearance—clearance granted, but with modification in training as specified by physician (with possibility for status to change depending on clinical progress and new information gathered); restricted from training/competition (provisional)—athlete not cleared at present time, with clearance status re-evaluated by physician and multidisciplinary team with clinical progress; disqualified—not safe to participate at present time, clearance status to be determined at future date depending on clinical progress, if appropriate. It is the recommendation of the Consensus Panel that athletes diagnosed with anorexia nervosa who have a body mass index (BMI) <16 kg/m² or with moderate-to-severe bulimia nervosa (purging >4 times/week) should be categorically restricted from training and competition. Future participation is dependent on treatment of their eating disorder, including ascertainment of BMI >18.5 kg/m², cessation of bingeing and purging and close interval follow-up with the multidisciplinary team. (Reprinted with permission from BJSM [2])

guide their safe return to participation. Based on the athlete’s risk score, different clearance recommendations are suggested – full clearance, restricted/provisional clearance, and restriction from training and competition (Fig. 11.3). Studies have demonstrated that an athlete’s risk score does correlate with future risk of BSI [39, 40]. Since the Triad occurs on a spectrum of severity and represents a complex relationship between the three conditions which comprise the Triad, return-to-play decisions for patients with the Triad must always be individualized. For example, several of the points gained in the CRA tool are for non-modifiable risk factors. An athlete with a history of delayed menarche will always carry risk points for this, even if she has improved her overall energy availability. Other non-modifiable risk factors include a prior history of an eating disorder and history of past BSI. Clinician judgment is necessary for situations such as these. It has been suggested that a modified weighted risk assessment tool may be useful for follow-up assessments. In addition, physicians should utilize their individual judgment regarding consideration of other



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Fig. 11.4 Recovery from the female athlete triad. The three components of the Triad recover at different rates, and full recovery may be a long process lasting months to years. The three components of the Triad recover at different rates with the appropriate treatment. Recovery of energy status is typically observed after days or weeks of increased energy intake and/or decreased energy expenditure. Recovery of menstrual status is typically observed after months of increased energy intake and/or decreased energy expenditure, which improves energy status. Recovery of bone mineral density may not be observed until years after recovery of energy status and menstrual status has been achieved. IGF-1, insulin-like growth factor-1. (Reprinted with permission from BJSM [2])

known risk factors which are not included in the CRA, such as family history of osteoporosis, vitamin D deficiency, low dietary calcium intake, or malabsorptive disorders such as celiac disease [30]. The IOC has proposed a return-to-play model based on red light (high risk), yellow light (moderate risk), and green light (low risk) groups [5] (Fig. 11.4).

Because safe return-to-play is often contingent on specific criteria (such as the athlete continuing to meet regularly with members of their care team), the use of contracts may be helpful. These are documents which specify all of the necessary conditions for ongoing safe participation, such as keeping appointments, adhering to an exercise or meal plan, or achieving certain minimum weight or body composition. In addition, a contract may specify the consequences of failure to follow the care plan, such as restricted participation, decreased training mileage, or even removal from team training. The contract should be reviewed and agreed upon by all members of the care team, and then reviewed with the athlete as well. The goal of the medical contract is to establish the plan for safe return-to-play which prioritizes an athlete’s health and wellbeing, and does not expose them to undue risk during their return to sport. An example of a contract can be found in the 2014 Female Athlete Triad Consensus Statement [2].

The Male Athlete Triad

Although historically most of the research on the Triad has been done in female athletes, male athletes have also been found to have low EA and increased risks of BSI. This has led to a description of the Male Athlete Triad, which mirrors the female athlete triad, and consists of low energy availability, hypogonadism, and decreased BMD [6, 7]. As in female athletes, low energy availability can be intentional or unintentional. Although eating disorders are more common in female athletes than in male athletes, male athletes have a higher prevalence of eating disorders than male non-athletes. In one study, 20% of female athletes and 8% of male athletes met criteria for an eating disorder, compared to 9% of female controls and 0.5% of male controls [10]. As in female athletes, eating disorders are particularly common in male athletes who participate in lean sports. One study found rates of eating disorders to be 22% in antigravitational sports, 9% in endurance sports, and 5% in ball sports [10]. Another study found the prevalence of eating disorders to be 42% in antigravitational sports, 17% in weight class sports, and 10% in endurance sports [57]. As was noted in female athletes, male athletes also demonstrate high levels of disordered eating behaviors even in the absence of a formal eating disorder diagnosis [58, 12, 59, 60, 61].

Evidence suggests that the EA cut-off for observing physiologic effects (impact on reproductive and metabolic hormones) may be lower in male athletes than in female athletes [62, 63]. One study found that a period of low EA reduced leptin and insulin levels in men (similar to changes seen in women) but did not observe changes in ghrelin, T3, testosterone, and IGF-1 [63]. More research is needed to better understand the physiologic effects of low EA in male athletes [5, 6, 8].

Male athletes do not have menstrual periods that provide an easily observed indicator of hypothalamic function; however, studies suggest that male athletes with high training volumes or low EA may have decreased testosterone levels or sperm counts, or may see changes in the hypothalamic-pituitary-gonadal axis [6]. As in females, low EA and hypogonadism have important consequences for bone health. One study found that either low estrogen (in females) or low testosterone (in males) was associated with 4.5-fold increased risk of BSI [64]. A study in male adolescent runners found that risk factors for low BMD were cumulative and included weight less than 85% of expected, average weekly mileage greater than 30, prior history of stress fracture, and less than one serving of calcium per day [65]. Furthermore, adolescent male runners had lower weight, lower BMI, and lower spine BMD Z-scores when compared with adolescent male athletes who were not runners. It is therefore important to screen not only our female athletes but also our male athletes for the Triad.

The Triad in Para-Athletes

Similar to the general athlete population, para-athletes may also present with the Triad [5]; however, para-athletes may have unique differences in their energy demands, bone health, and menstrual function [66, 8]. Energy availability may vary

for para-athletes – athletes in wheelchairs may have lower energy demands, but some athletes with prosthetics, or with involuntary movements such as dyskinesia or athetosis, may have increased metabolic demands. Central nervous system injuries that disrupt the hypothalamic-pituitary axis may also affect menstrual function. Para-athletes may also be predisposed to disuse osteopenia or osteoporosis from altered skeletal loading patterns, which can further compound the effects of the Triad. Para-athletes should also be screened for the Triad, and clinicians should be mindful of factors which may affect a para-athlete's presentation with the Triad. Although awareness of Triad risk factors in para-athletes is increasing, more research is needed in this area.

Prevention

As we have detailed earlier in this chapter, the potential long-term health consequences of the Triad are significant. The sports medicine community should seek to increase awareness of the Triad as much as possible. Increased awareness among athletes, parents, coaches, athletic trainers, and physicians may help prevent the Triad and may also assist in identifying athletes with the Triad as early as possible in order to facilitate prompt treatment.

Unfortunately, studies have demonstrated that knowledge of the Triad is low in several of these groups. In a study on US High School nurses, only 19% were able to identify the three components of the Triad and only 25% reported working proactively with coaches to help prevent health issues in their female athletes. However, more than 95% expressed interest in learning more about the Triad [67]. In a survey of multi-specialty physicians, only 37% had heard of the Triad, and of these respondents, they named an average of 2.1 components of the Triad correctly [68]. In one small study, 30% of coaches had heard of the Triad, but only 10% could name the three components of the triad. Furthermore, 30% of the coaches thought that menstrual irregularities were normal in athletes. When asked to list consequences of low energy or disordered eating, 70% of the coaches listed impaired performance, but only 30% listed menstrual dysfunction, and 20% listed injury [69]. NCAA Division I collegiate coaches fared slightly better, in that 43% were able to correctly identify the components of the Triad; however, this still leaves significant room for improvement [70]. In one study on Australian women who exercised regularly, only 10% of respondents could name all three components of the Triad, and 45% did not think amenorrhea could affect bone health. Athletes in lean sports, or with a history of amenorrhea or stress fracture, were all significantly less likely to take action about amenorrhea [71]. When athletic trainers at NCAA institutions were surveyed, almost all (98%) had heard of the triad, but on average respondents could only identify two of the three components [72]. Given these numbers, it is not surprising that many young women feel it is “normal” for a competitive athlete to have irregular periods. Beliefs like these are important to address, in order to avoid the long-term health consequences of the Triad.

In addition to raising awareness of the Triad, it is also important to create a healthy approach to exercise and nutrition at home and at school. Several promising evidence-based educational programs have been used in high school or college populations. One study in female college athletes found that two behavioral modification programs were effective at reducing end-points associated with eating disorders at 6 weeks and at 1 year. Furthermore, after the programs, several athletes came forward to seek medical care for the Triad [73]. Another study demonstrated short- and long-term improvement in disordered eating behaviors and body image among high school students who went through the ATHENA program (Athletes Targeting Healthy Exercise and Nutrition Alternatives), which is a sport team-based harm reduction and health promotion program [74]. Research has also shown that in female college students, both clinician-led and peer-led dissonance-based eating disorder prevention programs were effective in reducing eating disorder risk factors and eating disorder onset, and both were more effective than an online program [75].

Conclusion

In conclusion, the Female Athlete Triad is comprised of the three interrelated conditions: low energy availability, menstrual dysfunction, and decreased BMD. Each of these three conditions can occur along a spectrum, and patients may move along each portion of the spectrum at different rates. There are serious potential long-term consequences of the Triad, and early recognition is key in order to facilitate prompt treatment and reduce the risk of long-term complications. Clinicians should be comfortable identifying and treating each of the three primary components of the Triad. Working with a multidisciplinary team is key to successful treatment, and the team should include a mental health professional whenever disordered eating or eating disorders are present. The first-line treatment for the Triad is increasing EA either by reducing energy expenditure, increasing energy intake, or both. If menstrual dysfunction is identified, alternative causes of amenorrhea or oligomenorrhea should be ruled out. In addition, clinicians should optimize bone health as much as possible, including dietary intake of calcium and vitamin D. Return-to-play decisions in athletes with the Triad are complex, and clinicians should rely on the risk assessment tool provided in the 2014 Female Athlete Triad Consensus Statement as well as their own clinical judgment. There is increasing evidence for the Male Athlete Triad, and male athletes should also be screened for low EA, hypogonadism, and decreased BMD. Due to the potential serious health consequences of the Triad, preventative efforts are of the utmost importance. Evidence suggests the need for increased awareness of the Triad. Educational programs about body image and healthy eating demonstrate long-term efficacy and may help to promote healthy attitudes towards exercise and nutrition.

References

1. Nattiv A, Loucks AB, Manore MM, Sanborn CF, Sundgot-Borgen J, Warren MP, et al. American College of Sports Medicine position stand. The female athlete triad. *Med Sci Sports Exerc.* 2007;39(10):1867–82.
2. De Souza MJ, Nattiv A, Joy E, Misra M, Williams NI, Mallinson RJ, et al. 2014 Female Athlete Triad coalition consensus statement on treatment and return to play of the female athlete triad: 1st international conference held in San Francisco, California, May 2012 and 2nd international conference held in Indianapolis, Indiana, May 2013. *Br J Sports Med.* 2014;48(4):289.
3. Yeager KK, Agostini R, Nattiv A, Drinkwater B. The female athlete triad: disordered eating, amenorrhea, osteoporosis. *Med Sci Sports Exerc.* 1993;25(7):775–7.
4. Otis CL, Drinkwater B, Johnson M, Loucks A, Wilmore J. American College of Sports Medicine position stand. The female athlete triad. *Med Sci Sports Exerc.* 1997;29(5):i–ix.
5. Mountjoy M, Sundgot-Borgen J, Burke L, Carter S, Constantini N, Lebrun C, et al. The IOC consensus statement: beyond the Female Athlete Triad--Relative Energy Deficiency in Sport (RED-S). *Br J Sports Med.* 2014;48(7):491–7.
6. Tenforde AS, Barrack MT, Nattiv A, Fredericson M. Parallels with the female athlete triad in male athletes. *Sports Med.* 2016;46(2):171–82.
7. Fredericson M, Nattiv A. ACSM symposium – the male athlete triad: updates and parallels with the female athlete, ACSM 65th annual meeting proceedings, Abstract A-18. 2018: p. 6.
8. Mountjoy M, Sundgot-Borgen JK, Burke LM, Ackerman KE, Blauwet C, Constantini N, et al. IOC consensus statement on relative energy deficiency in sport (RED-S): 2018 update. *Br J Sports Med.* 2018;52(11):687–97.
9. Beals KA, Manore MM. Disorders of the female athlete triad among collegiate athletes. *Int J Sport Nutr Exerc Metab.* 2002;12(3):281–93.
10. 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(1):25–32.
11. Reinking MF, Alexander LE. Prevalence of disordered-eating behaviors in undergraduate female collegiate athletes and nonathletes. *J Athl Train.* 2005;40(1):47–51.
12. Glazer JL. Eating disorders among male athletes. *Curr Sports Med Rep.* 2008;7(6):332–7.
13. Sundgot-Borgen J, Torstveit MK. Aspects of disordered eating continuum in elite high-intensity sports. *Scand J Med Sci Sports.* 2010;20(Suppl 2):112–21.
14. Dusek T. Influence of high intensity training on menstrual cycle disorders in athletes. *Croat Med J.* 2001;42(1):79–82.
15. Abraham SF, Beumont PJ, Fraser IS, Llewellyn-Jones D. Body weight, exercise and menstrual status among ballet dancers in training. *Br J Obstet Gynaecol.* 1982;89(7):507–10.
16. Khan KM, Liu-Ambrose T, Sran MM, Ashe MC, Donaldson MG, Wark JD. New criteria for female athlete triad syndrome? As osteoporosis is rare, should osteopenia be among the criteria for defining the female athlete triad syndrome? *Br J Sports Med.* 2002;36(1):10–3.
17. 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.
18. Thein-Nissenbaum JM, Rauh MJ, Carr KE, Loud KJ, McGuine TA. Associations between disordered eating, menstrual dysfunction, and musculoskeletal injury among high school athletes. *J Orthop Sports Phys Ther.* 2011;41(2):60–9.
19. Thiemann P, Legenbauer T, Vocks S, Platen P, Auyeung B, Herpertz S. Eating disorders and their putative risk factors among female German professional athletes. *Eur Eat Disord Rev.* 2015;23(4):269–76.
20. De Souza MJ, Koltun KJ, Etter CV, Southmayd EA. Current status of the female athlete triad: update and future directions. *Curr Osteoporos Rep.* 2017;15(6):577–87.
21. Brown KA, Dewoolkar AV, Baker N, Dodich C. The female athlete triad: special considerations for adolescent female athletes. *Transl Pediatr.* 2017;6(3):144–9.
22. Kim BY, Nattiv A. Health considerations in female runners. *Phys Med Rehabil Clin N Am.* 2016;27(1):151–78.

23. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington: American Psychiatric Association; 2013.
24. Joy E, Kussman A, Nattiv A. 2016 update on eating disorders in athletes: a comprehensive narrative review with a focus on clinical assessment and management. *Br J Sports Med.* 2016;50(3):154–62.
25. Ackerman KE, Holtzman B, Cooper KM, Flynn EF, Bruinvels G, Tenforde AS, et al. Low energy availability surrogates correlate with health and performance consequences of relative energy deficiency in sport. *Br J Sports Med.* 2018;53(10):bjsports-2017-098958.
26. Vanheest JL, Rodgers CD, Mahoney CE, De Souza MJ. Ovarian suppression impairs sport performance in junior elite female swimmers. *Med Sci Sports Exerc.* 2014;46(1):156–66.
27. Woods AL, Garvican-Lewis LA, Lundy B, Rice AJ, Thompson KG. New approaches to determine fatigue in elite athletes during intensified training: resting metabolic rate and pacing profile. *PLoS One.* 2017;12(3):e0173807.
28. Tornberg Å, Melin A, Koivula FM, Johansson A, Skouby S, Faber J, et al. Reduced neuromuscular performance in amenorrheic elite endurance athletes. *Med Sci Sports Exerc.* 2017;49(12):2478–85.
29. Petkus DL, Murray-Kolb LE, De Souza MJ. The unexplored crossroads of the female athlete triad and iron deficiency: a narrative review. *Sports Med.* 2017;47(9):1721–37.
30. Joy EA, Nattiv A. Clearance and return to play for the female athlete triad: clinical guidelines, clinical judgment, and evolving evidence. *Curr Sports Med Rep.* 2017;16(6):382–5.
31. Rauh MJ, Nichols JF, Barrack MT. Relationships among injury and disordered eating, menstrual dysfunction, and low bone mineral density in high school athletes: a prospective study. *J Athl Train.* 2010;45(3):243–52.
32. De Souza MJ, Miller BE, Loucks AB, Luciano AA, Pescatello LS, Campbell CG, et al. High frequency of luteal phase deficiency and anovulation in recreational women runners: blunted elevation in follicle-stimulating hormone observed during luteal-follicular transition. *J Clin Endocrinol Metab.* 1998;83(12):4220–32.
33. Goolsby MA, Boniquit N. Bone health in athletes. *Sports Health.* 2017;9(2):108–17.
34. Nattiv A, Kennedy G, Barrack MT, Abdelkerim A, Goolsby MA, Arends JC, et al. Correlation of MRI grading of bone stress injuries with clinical risk factors and return to play: a 5-year prospective study in collegiate track and field athletes. *Am J Sports Med.* 2013;41(8):1930–41.
35. Barrack MT, Gibbs JC, De Souza MJ, Williams NI, Nichols JF, Rauh MJ, et al. Higher incidence of bone stress injuries with increasing female athlete triad-related risk factors: a prospective multisite study of exercising girls and women. *Am J Sports Med.* 2014;42(4):949–58.
36. Ackerman KE, Putman M, Guereca G, Taylor AP, Pierce L, Herzog DB, et al. Cortical microstructure and estimated bone strength in young amenorrheic athletes, eumenorrheic athletes and non-athletes. *Bone.* 2012;51(4):680–7.
37. Bernhardt DT, Roberts WO, American Academy of Family Physicians, American Academy of Pediatrics. PPE: preparticipation physical evaluation. Elk Grove Village: American Academy of Pediatrics; 2010.
38. American College of Gynecologists. Committee opinion 702: female athlete triad. *Obstet Gynecol.* 2017;129(6):e160–7.
39. Tenforde AS, Carlson JL, Chang A, Sainani KL, Shultz R, Kim JH, et al. Association of the female athlete triad risk assessment stratification to the development of bone stress injuries in collegiate athletes. *Am J Sports Med.* 2017;45(2):302–10.
40. Kussman A, Fredericson M, Kraus E, Singh S, Deakins-Roche M, Miller E, Kim BY, Tenforde A, Barrack M, Sainani K, Nattiv A. The female athlete triad cumulative risk assessment score implemented at the preparticipation physical exam correlates with risk of bone stress injury in collegiate distance runners: a 4-year prospective study. Abstract published in *Clin J Sport Med.* 2018;28(2):247.
41. Cobb KL, Bachrach LK, Sowers M, Nieves J, Greendale GA, Kent KK, et al. The effect of oral contraceptives on bone mass and stress fractures in female runners. *Med Sci Sports Exerc.* 2007;39(9):1464–73.

42. Lappe J, Cullen D, Haynatzki G, Recker R, Ahlf R, Thompson K. Calcium and vitamin d supplementation decreases incidence of stress fractures in female navy recruits. *J Bone Miner Res.* 2008;23(5):741–9.
43. Nieves JW, Melsop K, Curtis M, Kelsey JL, Bachrach LK, Greendale G, et al. Nutritional factors that influence change in bone density and stress fracture risk among young female cross-country runners. *PM R.* 2010;2(8):740–50; quiz 94.
44. Kelsey JL, Bachrach LK, Procter-Gray E, Nieves J, Greendale GA, Sowers M, et al. Risk factors for stress fracture among young female cross-country runners. *Med Sci Sports Exerc.* 2007;39(9):1457–63.
45. Institute of Medicine. Dietary reference intakes for calcium and vitamin D. Washington DC: National Academies Press; 2011. 1132 p.
46. Bolland MJ, Avenell A, Baron JA, Grey A, MacLennan GS, Gamble GD, Reid IR. Effect of calcium supplements on risk of myocardial infarction and cardiovascular events: meta-analysis. *BMJ.* 2010;341:c3691.
47. Curhan GC, Willett WC, Speizer FE, Spiegelman D, Stampfer MJ. Comparison of dietary calcium with supplemental calcium and other nutrients as factors affecting the risk for kidney stones in women. *Ann Intern Med.* 1997;126(7):497–504.
48. Maroon JC, Mathyssek CM, Bost JW, Amos A, Winkelman R, Yates AP, et al. Vitamin D profile in National Football League players. *Am J Sports Med.* 2015;43(5):1241–5.
49. Kim BY, Kraus E, Fredericson M, Tenforde A, Singh S, Kussman A, Barrack M, Deakins-Roche M, Nattiv A. Serum vitamin D levels are inversely associated with time lost to bone stress injury in a cohort of NCAA Division I distance runners. Abstract published in *Clin J Sport Med.* 2016;26(2):e58–68.
50. Sanders KM, Stuart AL, Williamson EJ, Simpson JA, Kotowicz MA, Young D, et al. Annual high-dose oral vitamin D and falls and fractures in older women: a randomized controlled trial. *JAMA.* 2010;303(18):1815–22.
51. Fredericson M, Ngo J, Cobb K. Effects of ball sports on future risk of stress fracture in runners. *Clin J Sport Med.* 2005;15(3):136–41.
52. Torstveit MK, Sundgot-Borgen J. Low bone mineral density is two to three times more prevalent in non-athletic premenopausal women than in elite athletes: a comprehensive controlled study. *Br J Sports Med.* 2005;39(5):282–7; discussion -7.
53. Arends JC, Cheung MY, Barrack MT, Nattiv A. 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(2):98–108.
54. Cialdella-Kam L, Guebels CP, Maddalozzo GF, Manore MM. Dietary intervention restored menses in female athletes with exercise-associated menstrual dysfunction with limited impact on bone and muscle health. *Nutrients.* 2014;6(8):3018–39.
55. Jonnavithula S, Warren MP, Fox RP, Lazaro MI. Bone density is compromised in amenorrheic women despite return of menses: a 2-year study. *Obstet Gynecol.* 1993;81(5 (Pt 1)):669–74.
56. Keen AD, Drinkwater BL. Irreversible bone loss in former amenorrheic athletes. *Osteoporos Int.* 1997;7(4):311–5.
57. Rosendahl J, Bormann B, Aschenbrenner K, Aschenbrenner F, Strauss B. Dieting and disordered eating in German high school athletes and non-athletes. *Scand J Med Sci Sports.* 2009;19(5):731–9.
58. Thiel A, Gottfried H, Hesse FW. Subclinical eating disorders in male athletes. A study of the low weight category in rowers and wrestlers. *Acta Psychiatr Scand.* 1993;88(4):259–65.
59. Chatterton JM, Petrie TA. Prevalence of disordered eating and pathogenic weight control behaviors among male collegiate athletes. *Eat Disord.* 2013;21(4):328–41.
60. Steen SN, Brownell KD. Patterns of weight loss and regain in wrestlers: has the tradition changed? *Med Sci Sports Exerc.* 1990;22(6):762–8.
61. Riebl SK, Subudhi AW, Broker JP, Schenck K, Berning JR. The prevalence of subclinical eating disorders among male cyclists. *J Am Diet Assoc.* 2007;107(7):1214–7.

62. Fagerberg P. Negative consequences of low energy availability in natural male bodybuilding: a review. *Int J Sport Nutr Exerc Metab.* 2018;28(4):385–402.
63. Koehler K, Hoerner NR, Gibbs JC, Zinner C, Braun H, De Souza MJ, et al. Low energy availability in exercising men is associated with reduced leptin and insulin but not with changes in other metabolic hormones. *J Sports Sci.* 2016;34(20):1921–9.
64. Heikura IA, Uusitalo ALT, Stellingwerff T, Bergland D, Mero AA, Burke LM. Low energy availability is difficult to assess but outcomes have large impact on bone injury rates in elite distance athletes. *Int J Sport Nutr Exerc Metab.* 2018;28(4):403–11.
65. Barrack MT, Fredericson M, Tenforde AS, Nattiv A. Evidence of a cumulative effect for risk factors predicting low bone mass among male adolescent athletes. *Br J Sports Med.* 2017;51(3):200–5.
66. Blauwet CA, Brook EM, Tenforde AS, Broad E, Hu CH, Abdu-Glass E, et al. Low energy availability, menstrual dysfunction, and low bone mineral density in individuals with a disability: implications for the para athlete population. *Sports Med.* 2017;47(9):1697–708.
67. Kroshus E, Fischer AN, Nichols JF. Assessing the awareness and behaviors of U.S. High School nurses with respect to the female athlete triad. *J Sch Nurs.* 2015;31(4):272–9.
68. Curry EJ, Logan C, Ackerman K, McInnis KC, Matzkin EG. Female athlete triad awareness among multispecialty physicians. *Sports Med Open.* 2015;1(1):38.
69. Brown KN, Wengreen HJ, Beals KA. Knowledge of the female athlete triad, and prevalence of triad risk factors among female high school athletes and their coaches. *J Pediatr Adolesc Gynecol.* 2014;27(5):278–82.
70. 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(4):195–207.
71. Miller SM, Kukuljan S, Turner AI, van der Pligt P, Ducher G. Energy deficiency, menstrual disturbances, and low bone mass: what do exercising Australian women know about the female athlete triad? *Int J Sport Nutr Exerc Metab.* 2012;22(2):131–8.
72. Kroshus E, DeFreese JD, Kerr ZY. Collegiate athletic trainers' knowledge of the female athlete triad and relative energy deficiency in sport. *J Athl Train.* 2018;53(1):51–9.
73. Becker CB, 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(1):31–42.
74. Elliot DL, Goldberg L, Moe EL, Defrancesco CA, Durham MB, McGinnis W, et al. Long-term outcomes of the ATHENA (Athletes Targeting Healthy Exercise & Nutrition Alternatives) program for female high school athletes. *J Alcohol Drug Educ.* 2008;52(2):73–92.
75. Stice E, Rohde P, Shaw H, Gau JM. Clinician-led, peer-led, and internet-delivered dissonance-based eating disorder prevention programs: acute effectiveness of these delivery modalities. *J Consult Clin Psychol.* 2017;85(9):883–95.