The female athlete triad: a current concepts review

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Abstract
Female athletic participation continues to grow throughout the world. This has many positive effects on health and well-being, but it has also led to a unique set of health problems. The female athlete triad was first described in 1992 by the American College of Sports Medicine, consisting of disordered eating, amenorrhea and osteoporosis. An updated position stand was released in 2007 that modified the components of the triad to energy availability, menstrual function and bone mineral density. This article reviews the current definitions of the triad components, epidemiology, pathophysiology, diagnosis and treatment. Each of the components of the triad exists on a continuum from healthy to pathological. Low energy availability, from either dietary restriction or increased expenditure, is the factor that leads to the pathological states of menstrual function and bone mineral density. Athletes especially at risk are those in sports requiring leanness or low body weight. Prevention and early recognition of triad disorders is crucial to ensure timely intervention and treatment. Treatment is centered on restoring energy availability to adequate levels (30 kcal.kg\(^{-1}\).d\(^{-1}\)) to re-establish normal metabolic functioning. All those who work with female athletes must remain vigilant in the education, recognition and treatment of athletes at risk. Continued research and knowledge of the triad disorders aids the development of prevention and treatment strategies to allow women to continue to enjoy the benefits of regular exercise and physical activity throughout their lives.

Introduction
Similar to the increases seen in athletic participation within the United States, female athletic participation continues to increase worldwide. An examination of the participation of women in the Olympic Games over the last century appears to support this. Female participation has steadily increased, most dramatically over the past 50 years. The most recent summer Olympics in Beijing (2008) saw the highest rate of female participation with a total of 4 746 women participating, representing a record 42% of athletes. Women remain under-represented in sport in South Africa, but consistent and progressive rates of participation are being appreciated throughout the country. As rates of female participation continue to increase throughout the country, it is important to consider the unique health challenges that female athletes may encounter.

For most female athletes, sports participation is a positive experience, providing improved physical fitness, enhanced self-esteem and better physical and mental health. However, this increased activity has also led to the emergence of a unique combination of medical problems and injuries specific to the female athlete. This includes a unique triad to conditions specific to the female athlete that include energy availability, menstrual function and bone mineral density.

Definitions
In 1992, the American College of Sports Medicine (ACSM) recognised the association of three distinct entities with female athletes. The female athlete triad was originally defined as the combination of disordered eating, amenorrhea and osteoporosis found in physically active girls and women. Further research since then proved these entities to be far too narrow in scope, existing only at the extreme endpoint of the disorder’s spectrum. By 2007, the ACSM revised position statement modified the three components of the triad into energy availability, menstrual function and bone mineral density. Each of these components consists on a continuum from healthy to subclinical to pathological. Energy intake from diet is necessary for various physiological functions. Energy expenditure is the energy expended by the body during normal daily activities and exercise training. Energy availability defines the amount of energy remaining for physiological functions after expended from physical activity. Decreased caloric consumption and/or increased exercise energy expenditure results in low energy available for normal physiology functions, including cellular maintenance, thermoregulation, growth and reproduction.

Clinical eating disorders, namely anorexia nervosa, bulimia nervosa and eating disorders not otherwise specified, exist on the extreme end of this category. However, these conditions carry a largely psychiatric component for diagnosis. A larger population of female athletes practise unhealthy eating and weight control behaviours with significant impact on their health without the psychiatric element. Such subclinical eating disorders include prolonged fasting, low caloric diets, binge eating, purging, or the...
utilisation of diet pills, laxatives and other supplements. Other athletes unknowingly fail to meet their considerable exercise energy requirements due to time constraints, food availability issues or lack of appropriate nutritional knowledge.6

Amenorrhoea is the absence of menstrual cycles, and can be subdivided into primary and secondary amenorrhoea. Primary amenorrhoea is the absence of menarche by age 15, whereas secondary amenorrhoea is an interruption of menses for greater than 3 months after the initiation of menarche.7 The ACSM updated position stand on the female athlete triad recognised other menstrual irregularities that cause an adverse effect to the female athlete’s health.4 Thus, the term menstrual dysfunction was employed as a component of the female athlete triad, which includes luteal suppression, anovulation and oligomenorrhoea in addition to primary and secondary amenorrhoea. Oligomenorrhoea describes a prolonged menstrual cycle greater than 35 days.8 Both luteal suppression and anovulation are asymptomatic conditions, associated with either a shortened luteal phase or lack of ovulation respectively.

Osteoporosis is a decrease in bone mass that increases the risk of fracture.9 This may be due to age-related decreases in bone mass, or the result of inadequate bone accumulation during growth and development resulting in a lower peak bone mass being attained. The World Health Organization (WHO) criterion for diagnosing osteoporosis is a bone mineral density more than 2.5 standard deviations below the mean of young adults.9 Utilisation of this T-score was based on epidemiological data relating bone mineral density to fracture risk in postmenopausal women,10,11 thus it lacks external validity for evaluating adolescent or premenopausal women. In 2005, the International Society for Clinical Densiometry (ISCD) recommended that bone mineral density for premenopausal women and children be expressed as a Z-score. A Z-score greater than or equal to 2.0 standard deviations below the mean of age- and sex-matched controls is defined as ‘low bone density below the expected age range’.12 Together the ISCD and ACSM defined osteoporosis in premenopausal women as having this low Z-score along with secondary clinical risk factors, including chronic malnutrition, eating disorders, hypogonadism, glucocorticoid exposure and previous fractures.12

**Epidemiology**

The prevalence of female athletes with abnormalities in energy availability, menstrual function and/or bone mineral density varies tremendously between studies. Reported data show a range of 1 - 62% of female athletes with an eating disorder,2,15 6 - 79% with menstrual dysfunction,2,16 and 0 - 50% with low bone mineral density.17

Several factors have contributed to these broad results, including the wide range and ever-changing definitions of each component of the female athlete triad. Utilising the strict DSM-IV diagnosis for clinical eating disorders yields a prevalence between 1% and 3%,18 whereas the more ambiguous term ‘disordered eating’ opens up the diagnostic criteria, yielding larger prevalence outcomes between 15% and 62%.18 Similarly, the expanded definition of menstrual dysfunction to include oligomenorrhoea, luteal suppression and anovulation has seen prevalence increase from 6% to as high as 79%.18,19 Finally, studies utilising the WHO classification for bone mineral density found 22 - 50% of patients with osteopenia, and virtually 0% having osteoporosis.17 The new ISCD criterion utilizing Z-scores identified 10.7% of female athletes to have low bone mineral density below the expected age range.20

Other aspects involved in the investigation of the female athlete triad result in an imprecise measurement of prevalence. Diagnosis of low energy availability typically involves self-questionnaires. Most questionnaires are designed for clinical eating disorders, such as the Eating Disorders Inventory (EDI or EDI-2) or the Eating Attitude Test (EAT).21 and focus mostly on the psychiatric component of the disease. They do not take into account other subclinical causes of low energy availability, including excess exercise energy expenditure and abnormal eating behaviours. This can be better assessed with documentation of energy balance through food logs and activity records. However, both evaluations require self-reporting, with inherent limitations due to underreporting of symptoms.1

The heterogeneity of the population being investigated for the female athlete triad has also created discrepancies in prevalence. The age of an athlete significantly affects their risk for each component of the triad, as does their stage in growth, development and menarche. Thus, comparing athletes over a wide range of ages during adolescence and pre-adulthood becomes problematic. In addition, because females develop at different rates, comparing them at similar ages can also cause difficulties.

The level of competition also affects the epidemiological studies of the female athlete triad. Comparison of elite athletes, collegiate athletes and recreational athletes has shown an increase risk for components of the female athlete triad with increasing level of competition. A meta-analysis of 34 studies with 2 459 athletes found an increased risk of disordered eating with elite athletes, and that non-elite athletes, especially those in high school, were somewhat protected.22 Early start of sport-specific training and dieting, a sudden increase in training volume, and sports-related injury are also risk factors for the female athlete triad more commonly seen in high-level athletes.23 The increased pressure for winning by the athletes, as well as coaches, parents and society in general, places these athletes at higher levels of competition at increased risk for behaviours associated with the female athlete triad.1

Different sports have different risk factors for the individual components of the female athlete triad. Low energy availability is more commonly seen in sports that emphasise a lean physique or low body weight (Table I).2 Byrne et al.24 identified 31% of elite female athletes in ‘thin-build’ sports to have an eating disorder compared with 8% in the ‘normal-build’ athletes. Thin-build athletes are those involved in sports emphasising leanness or low body weight (ballet, gymnastics, long distance running, swimming and diving), where normal-build athletes are those in sports with less emphasis on leanness (basketball, volleyball, hockey, softball).24 Menstrual dysfunction is more common in endurance sports such as running, with prevalence up to 69%.25-26 High-impact sports such as basketball, volleyball and gymnastics26,27,28 are associated with higher bone mineral density than lower-impact sports such as

<table>
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<th>TABLE I. Sport-specific risks to low energy availability</th>
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<tr>
<td><strong>Category</strong></td>
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<td>Sports subjectively scored</td>
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<tr>
<td>Dance, figure skating, diving, gymnastics, aerobics</td>
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<tr>
<td>Sports emphasising low body weight</td>
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<tr>
<td>Distance running, cycling, cross-country skiing</td>
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<td>Sports requiring body contour-revealing clothing</td>
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<tr>
<td>Volleyball, swimming, diving, cross-country running, cross-country skiing, track, cheerleading</td>
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<tr>
<td>Sports with weight categories</td>
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<tr>
<td>Horse racing, martial arts, wres-ting, rowing</td>
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<tr>
<td>Sports emphasising prepuberal body habitus</td>
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<td>Figure skating, gymnastics, diving</td>
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running and swimming. Consequently, studies examining single sports may either overestimate or underestimate the true overall prevalence of the female athlete triad.

Despite all the difficulties reporting on the female athlete triad, including inconsistent definitions, methodological issues and the heterogeneity of the population, three studies establish prevalence for the simultaneous diagnosis of all three components of the female athlete triad. These studies utilised large, heterogeneous samples of athletes as well as a more validated definition of low energy availability, menstrual dysfunction and low bone mineral density. Disordered eating and menstrual dysfunction were determined from a questionnaire, while bone mineral density was evaluated via dual-energy X-ray absorptiometry (DXA). The resultant prevalence was between 1.2% and 4.3% of female athletes for all three components. However, these values are likely underrepresented because neither low energy availability nor subclinical menstrual disorders, including luteal suppression, anovulation and oligomenorrhoea, were part of the diagnostic criteria. Also, as discussed previously, self-reporting has inherent bias. Finally, although the prevalence is low for all components of the triad, one study found 6% of female athletes had two components of the female athlete triad and 18 - 24% had one component.

Pathophysiology

In addition to redefining the components of the female athlete triad, the ACSM focused on the pathogenesis of the female athlete triad. The majority of the current research on the female athlete triad centres on low energy availability as the key entity underlying the entire process.

A specific aetiology has not been identified regarding the pathogenesis of eating disorders or low energy availability. There likely is a complex relationship between social, cultural, demographic, environmental, biological, psychological and behavioural factors. Controversy exists regarding whether athletes have a higher prevalence of disordered eating behaviours when compared with non-athletes, with some studies showing increased prevalence in the athletic population, while others show no difference.

However, there is evidence suggesting athletes face sport-specific pressures that may trigger eating disorders in vulnerable athletes. As discussed previously, sports with emphasis on low body weight place these athletes at increased risk for developing a clinical eating disorder. In addition, increased training and practice associated with organised athletics may lead to inadvertent low energy availability. Experiments have shown that dietary restriction increased hunger, but similar levels of energy deficiency by increased exercise expenditure did not increase appetite. As a result, athletes unknowingly do not increase their energy intake due to increased exercise expenditure due to a lack of a physiological response.

Energy availability is not just related to diet and exercise, but also dependent on other hormonal and physiological processes. Two different hormones, leptin and ghrelin, have been identified as potential regulators of metabolic rate and energy homeostasis. The hormone leptin regulates the resting metabolic rate, and has been proposed as a marker of nutritional status. Leptin is decreased in athletes with disordered eating and amenorrhoea. A leptin value of 1.85 mg has been identified as a minimum value to maintain a normal menstrual cycle. Ghrelin is a hormone produced in the stomach and gastrointestinal tract that acts as a metabolic signal for hunger and energy homeostasis. De Souza et al. showed high levels of ghrelin in amenorrhoeic women indicate an energy deficit. Ghrelin may also play a physiological role in reproductive function via actions on the luteinising hormone (LH) pulsatility. Increased physical activity has been linked to the cessation of menses. This has been found as a result of a disruption in the pulsatile secretion of LH by the pituitary gland, which is caused by a disruption earlier in the pulsatile secretion of the gonadotropin-releasing hormone by the hypothalamus. In multiple studies, Loucks showed that low energy availability is at the root of hypothalamic menstrual dysfunction. The threshold of energy availability necessary to maintain normal menstrual function in 30 kcal.kg\(^{-1}\) lean body mass per day. Strenuous training alone is not enough to disrupt menstrual function, unless it is also accompanied by dietary restriction. Normal cycles can be restored with increasing dietary intake; however, this is only accomplished with sustained increased intake, not simply one day of aggressive refeeding.

Altered bone mineral density is related to deficiency in the hormones oestrogen and progesterone, as well as caloric deprivation and inadequate calcium intake. The most important function of oestrogen on bone health is its suppressing effect of oestrogen activity. BMD declines as the number of missed menstrual cycles accumulates and these losses may not be fully reversible. A study by Ihl and Loucks showed that low energy availability may have a direct effect of bone. They demonstrated markers of bone formation and resorption changed negatively within 5 days in sedentary women exposed to low energy availability through dietary restriction or increased exercise energy expenditure. It is plausible to expect similar results to be seen in an athletic population. Additional work by Zanker has shown that low BMI and an oestrogen deficiency were associated with disruption of bone formation in amenorrhoeic women. However, there was a lack of correlation between serum levels of bone formation markers, BMI and serum oestradiol concentration in the eumenorrhoeic athletes, suggesting that there may be a threshold value of BMI, or serum oestradiol concentration, above which bone formation is not influenced by either of these variables. In fact, markers of bone formation are low in athletes with menstrual dysfunction, suggesting that low bone formation is a result of undernutrition.

Additional studies have indicated that low energy availability affects other metabolites and hormones, such as insulin, growth hormone, insulin-like growth factor-1, cortisol and thyroid hormone. These are all important for optimal bone health and metabolism independent of a hypo-oestrogenic state.

Health consequences

The body compensates for low energy availability by decreasing the resting metabolic rate, resulting in decreased energy to other normal physiological mechanisms, such as cellular maintenance, thermoregulation, growth and reproduction. Thus, although a stable body weight may be maintained, the health of the athlete is adversely affected.

Sustained low energy availability can have significant effects on health. Undernutrition impairs both menstrual and skeletal health. Additionally, several studies have shown medical effects and physical symptoms involving the dermatological, cardiovascular, endocrine, gastrointestinal, renal and central nervous systems (Table II). Montero et al. examined the effect of low energy availability on the immune system of the female athlete (Fig. 1). Increased exercise combined with low calorie intake seems to decrease immunocompetence, resulting in decreased endurance, decreased performance and increased chance of subclinical and clinical infections.

The effect that low energy availability has on athletic performance varies depending on the severity and duration of the low energy state.
as well as the physical demands of the sport. Longer durations of restricted energy availability are associated with more dramatic negative effects on performance. Likewise, athletes in endurance sports with high energy demands (distance running, swimming, cycling, basketball) show more effects on performance than athletes in lower energy demand sports (gymnastics, diving, weightlifting).

The consequences of menstrual dysfunction have been well documented and include infertility, decreased immune function, increased cardiovascular risk factors and decreased bone mineral density with its associated increased risk of premature osteoporosis. It should be emphasised to athletes that menstrual dysfunction is not a normal response to athletic training. Athletes may show little concern for disruption of their cycles and may even ‘appreciate the break’. However, this should be viewed as a clear indication that health is being compromised.

An athlete’s bone mineral density is a reflection of her cumulative history of energy availability and menstrual status, in combination with genetics and other environmental factors. It is important to always consider the current bone mineral density status and where it is moving along the spectrum. Stress fractures occur more frequently in females who display menstrual irregularities and/or low bone mineral density. A premenopausal fracture not related to a significant traumatic event is a strong predictor for postmenopausal fractures. Even with restoration of adequate energy availability and regular menses the loss of bone mineral density may not be fully reversible.

**Screening and diagnosis of triad disorders**

Screening for and diagnosis of the triad disorders can be challenging due to the symptoms and health consequences not always being readily apparent. A high degree of suspicion and an understanding of the relationship between the triad components are critical to diagnosis. Many of the affected athletes are involved in those sports requiring leanness or thinness as an advantage in performance; however, individuals participating in any sport or regular physical activity are also at risk. Additionally, individuals of all levels may be at risk, not just the elite population. Optimal screening times are during pre-participation physicals and during annual health exams. Other opportunities present when athletes are evaluated for related problems. Any time an athlete presents with one component of the triad, they should be assessed for the others.

**Energy availability/disordered eating**

A high index of suspicion is necessary for the recognition and diagnosis of disordered eating behaviours. Psychological or behavioural characteristics may alert the physician or other member of the health care team. Examples include: anxiety with eating or weight gain, unusual dieting behaviour, binging or secretive eating, social withdrawal, abuse of laxatives or other weight loss medications and over-exercising. Additionally, recognition of physical symptoms likely related to malnutrition and/or purging behaviours may be seen. Weight loss methods may include restriction of food intake, fasting, self-induced vomiting, diet pills, laxatives, diuretics and excessive exercise. Physical exam is important in the recognition of eating disorders, and should start with height, weight and vital signs. Orthostatic hypotension should also be assessed. Other physical findings include cold or discoloured hands and feet, hypercarotenemia, lanugo hair and parotid gland enlargement. Laboratory assessment should initially include a complete blood count with differential, chemistry profiles, erythrocyte sedimentation rate, C-reactive protein, thyroid function studies (and other sex steroid hormones depending on physical findings) and a urinalysis. An electrocardiogram should also be done to evaluate for any arrhythmias and prolongation of the QT interval.

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**Table II. Physical symptoms of athletes with eating disorders**

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<thead>
<tr>
<th>Dermatological/dental</th>
<th>Cardiovascular</th>
<th>Endocrine</th>
<th>Gastrointestinal</th>
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<tr>
<td>Hair loss</td>
<td>Bradycardia</td>
<td>Hypoglycaemia</td>
<td>Swollen parotid glands</td>
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<tr>
<td>Lanugo hair</td>
<td>Hypotension</td>
<td>Delayed onset of puberty</td>
<td>Constipation/diarrhoea</td>
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<tr>
<td>Dorsal hand callus or abrasions</td>
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<td>Menstrual dysfunction</td>
<td>Postprandial distress</td>
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<td>Dental and gum problems</td>
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<td>Stress fractures</td>
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<tr>
<th>Renal</th>
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<tr>
<td>Dehydration</td>
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<td>Oedema</td>
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<tr>
<td>Electrolyte disturbances</td>
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<tr>
<td>Hypokalaemia</td>
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<tr>
<td>Muscle cramps</td>
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<td>Metabolic alkalosis</td>
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<th>CNS/Other</th>
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<tr>
<td>Significant weight loss</td>
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<td>Frequent weight fluctuations</td>
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<tr>
<td>Extreme fatigue</td>
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<tr>
<td>Muscle weakness</td>
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<tr>
<td>Hyperactivity</td>
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<td>Anaemia</td>
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**Fig. 1. Consequences of increased exercise and a low BMI in the female athlete.**
Menstrual dysfunction

Similar to identification of eating disorders, a high degree of suspicion, thorough history taking and physical exam are important in identification of menstrual dysfunction. Menstrual dysfunction commonly accompanies disordered eating, but if it continues after weight is restored to within 10% or normal, further workup for amenorrhea is indicated. Patients with functional hypothalamic amenorrhea often have a normal physical exam, but may show vaginal atrophy on pelvic exam if hypo-oestrogenism is present. Laboratory evaluation for secondary amenorrhea should include a pregnancy test, gonadotropin measurement (FSH, LH), probing and thyroid studies. If there is evidence of androgen excess on physical exam, free testosterone and dehydroepiandrosterone sulfate may be obtained. Additional workup may be needed based on patient history and physical exam, as well as with cases of primary amenorrhea. Consultation with a physician experienced in reproductive medicine is recommended if menses are not restored after 3 - 6 months of treatment.

Bone mineral density

The gold standard for evaluation of bone mineral density is with DXA. Current recommendations for evaluation with DXA include those athletes with a history of stress fractures, fractures with minimal trauma, a history of hypo-oestrogenism, and disordered eating or eating disorders for a cumulative total of 6 months or more.44 It is important to remember that in normal female athletes, particularly those in weight-bearing sports, have bone mineral density measurements 12 - 15% higher than non-athletes.20 Altered bone mineral density may be obvious in the athlete with a stress fracture or fragility fracture. Additionally, affected athletes are more likely to report sprains, strains and other soft-tissue injuries.65

Treatment recommendations

Prevention and treatment of the triad should employ a multidisciplinary team for best results. The health care team should include a physician, registered dietician and a mental health practitioner, especially for those athletes with eating disorders. Additional team members may include athletic trainers, an exercise physiologist, coaches, and parents and other family members.

Prevention

The best way to prevent the detrimental health effects of the triad components is through early recognition of athletes at risk and then incorporation of prompt education and intervention in those athletes. Special emphasis should be placed on optimising energy availability and maximising bone mineral accumulation in younger athletes. Athletes should be counselled on appropriate nutritional requirements for their age. Females of all ages should also be educated on the benefits of regular weight-bearing exercise for bone health. Athletes need to maintain minimum criteria to continue with training and competition. Close follow-up and open communication with the entire health care team is essential. Athletes who fail to maintain appropriate levels of energy availability, refuse treatment, or fail to improve eating behaviour should be withheld from training and competition to focus on their health.

In some athletes the addition of pharmacological therapy is beneficial in the treatment of triad disorders. Athletes with low energy availability secondary to clinical eating disorders may benefit from antidepressant medications. Antidepressants are also used in athletes with concomitant depression and anxiety disorders.69 The other established and recommended pharmacological treatments in athletes with the triad are aimed at maintenance and restoration of bone mineral density. In females with functional hypothalamic amenorrhea, hormonal therapy, commonly oral contraceptive pills (OCP) are used to protect bone mineral density. When OCP are used to restore regular menstrual cycles, it will not normalise metabolic factors that impair bone formation, health and performance. Therefore it is unlikely to fully reverse low bone mineral density in this population, and studies have shown inconsistent osteogenic effects with OCP.70,71 Current indications for the initiation of treatment with OCP are females older than age 16 years with declining bone mineral density and persistent functional hypothalamic amenorrhea despite adequate energy availability, with the goal of minimising further bone loss.73 Agents for increasing bone mineral density, such as bisphosphonates and selective oestrogen receptor modulators, are not recommended for use in premenopausal women.71 Bisphosphonates have not been proven effective in women of childbearing age.72 Additionally, they may be stored in the bone for many years, raising concern for possible harm to a fetus during pregnancy.72 Further research into the use of any currently available or novel forms of hormone therapy will be important in increasing bone mineral density in athletes with functional amenorrhea.

Summary

Low energy availability, menstrual function and altered bone mineral density, in isolation or in combination, continue to pose significant health risks to female athletes. We continue to see progress in determining the aetiology, pathophysiology, recognition, diagnosis and management of the triad disorders. All those who work with female athletes should remain vigilant in the education, recognition and treatment of athletes at risk. Continued advancement of research efforts and knowledge of the triad disorders aiding in the development of prevention and treatment strategies will allow all women to continue to enjoy the benefits of regular exercise and physical activity throughout their lives.

Declarations

The manuscript has been read and approved by all listed co-authors, and meets requirements of co-authorship. This manuscript has not previously been published. There are no conflicts of interest for any of the authors. There are no other disclosures for the authors.
REFERENCES


