The Physician and Sportsmedicine

ISSN: 0091-3847 (Print) 2326-3660 (Online) Journal homepage: https://www.tandfonline.com/loi/ipsm20

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To cite this article: David R. Hooper, Adam S. Tenforde & Anthony C. Hackney (2018) Treating exercise-associated low testosterone and its related symptoms, The Physician and Sportsmedicine, 46:4, 427-434, DOI: 10.1080/00913847.2018.1507234

To link to this article: https://doi.org/10.1080/00913847.2018.1507234

Accepted author version posted online: 31 Jul 2018.
Published online: 27 Aug 2018.

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THE PHYSICIAN AND SPORTSMEDICINE
2018, VOL. 46, NO. 4, 427–434
https://doi.org/10.1080/00913847.2018.1507234

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CLINICAL FEATURE
REVIEW

Treating exercise-associated low testosterone and its related symptoms

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ABSTRACT
The Exercise-Hypogonadal Male Condition (EHMC) has been described to occur in athletes who experience low serum testosterone and associated symptoms. While high volumes of endurance exercise can lead to reduced testosterone concentrations in men, similar changes may occur in other sports including anerobic and power sports such as American football, and weight class sports such as wrestling. A reduced testosterone concentration alone does not necessarily warrant treatment, but when it is accompanied by symptoms of hypogonadism, such as fatigue, sexual dysfunction, and/or low bone mineral density (BMD), an athlete's performance and/or health may suffer. While pharmacological treatments such as testosterone or clomiphene citrate have been shown to be effective in treating hypogonadism, these options are not available to athletes competing in sports governed by the World Anti-Doping Agency. We recommend treatment of EHMC in athletes should include nutritional intervention and modification of training. Recognizing EHMC's existence in male athletes is important to optimize their health.

ARTICLE HISTORY
Received 12 March 2018
Accepted 30 July 2018

KEYWORDS
Exercise-hypogonadal male condition; relative energy deficiency in sports; hypogonadism; overtraining

Introduction

The association between high volumes of endurance exercise and reduced sex hormone concentrations in men was initially reported over three decades ago, after a similar response had been previously noted in women [1]. Men running at least 64 km per week were compared to sedentary controls and observed to have significantly lower concentrations of serum total testosterone, free testosterone, and prolactin [1]. Multiple subsequent investigations have identified disruptions to the hypothalamic pituitary gonadal axis in male athletes, especially those engaged in endurance exercise training [2–10]. Years later, this manifestation and its accompanied symptoms were termed the 'Exercise-Hypogonadal Male Condition' (EHMC) [11]. Without large-scale epidemiological studies in this area, clear prevalence data is not available. However, the aforementioned studies, despite low sample sizes, show a clear and consistently reduced serum testosterone concentration in highly aerobically trained individuals, suggesting EHMC can be a common response. It also appears that as the level of athlete increases, so too does the incidence and severity of the condition. For example, in 22 athletes competing at the Kona Ironman World Championships, only 9 demonstrated serum testosterone concentrations that would be typically considered normal, suggesting a prevalence of EHMC in elite endurance athletes at well over 50% [12]. In addition, with no long-term data currently available, it is unclear whether the presence of reduced testosterone varies throughout a competitive season, and how long it takes for testosterone to return to normal, if at all.

This reduction in testosterone in and of itself is not necessarily a maladaptation. It has been previously argued that EHMC could represent an adjustment within the hypothalamic-pituitary-testicular axis causing a new, lowered set point for circulating testosterone [13]. However, this reduction in testosterone resulting from high volumes of endurance exercise can be accompanied by other classic symptoms of hypogonadism, such as sexual dysfunction [14] or low bone mineral density (BMD) [9]. In these cases, EHMC could possibly be viewed as a form of overreaching or overtraining, where an accumulation of training has not been accompanied by adequate rest resulting in physiological or psychological signs and symptoms of maladaptation [15].

As well as sharing similarities with overreaching or overtraining, EHMC has also been described in male athletes as a parallel process to the Female Athlete Triad, with hypogonadism replacing functional hypothalamic amenorrhea [16,17]. The importance of describing the effects of nutrition and exercise on physiological function in both sexes is highlighted by the International Olympic Committee Medical Commission's proposed new terminology of Relative Energy Deficiency in Sport (RED-S) medical condition [18]. The existence of the EHMC fits into the terminology of RED-S as clinical manifestation of it may include sexual dysfunction like infertility [19] and reduced libido [14] as well as reduced BMD [9] with associated increase in risk of bone stress injury.

Although EHMC shares similarities with overreaching/overtraining, the Female Athlete Triad and RED-S, there are subtle differences between these conditions. As stated by the Joint Consensus Statement of the European College of Sport Science and the American College of Sports Medicine, when

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approaching a possible case of overreaching/overtraining, it is important to first exclude other potential contributing factors, such as negative energy balance [15]. With this in mind, if energy deficit is the cause of the symptoms, the condition does not fall directly under the overreaching/overtraining umbrella, but is rather a case of RED-S. However, it is important to note that not all cases of reduced serum testosterone associated with high volumes of endurance exercises are associated with energy deficit. Although many cases are, there are examples of endurance athletes with clearly reduced testosterone concentrations despite adequate energy intake [9]. In this study, although the group of endurance athletes as a whole had low energy intake, the large variance is indicative of the fact that some endurance athletes do have adequate energy intake and yet still demonstrate reduced testosterone. In these cases, EHMC differentiates from RED-S.

The medical definition of hypogonadism also differs slightly from the EHMC. While studies have demonstrated a reduction in testosterone associated with aerobic exercise and values lower than non-exercising men, testosterone often remains within the normal laboratory reference values. For example, Hackney et al. demonstrated a significantly lower serum testosterone concentration in endurance trained men when compared to age-matched healthy controls (16.6 vs. 24.6 $nmol{\cdot}L^{-1}$) [6]. The average values in both populations studied fell within reference values suggested by the Endocrine Society, defining the lower limit of normal testosterone to be approximately 9.8–10.4 $nmol{\cdot}L^{-1}$ [20]. However, as analytical techniques have changed over the years, these recently published criteria are based on the use of mass spectrometry rather than immunoassays, which have been used in all of the research in this area to date. Thus, these direct comparisons between Endocrine Society guidelines and EHMC literature are made with caution. With that being said, other studies have demonstrated average testosterone levels below this threshold, particularly in populations of ultra-endurance athletes [21–23].

While low testosterone may occur in a subset of athletes, treatment of hypogonadism requires both low serum testosterone levels with associated psychological and physiological symptoms [20,24]. By extension, a male athlete may be diagnosed as hypogonadal based on symptoms while another athlete without symptoms may have lower testosterone levels than the hypogonadal athlete [24]. Thus, there is a clear distinction between a biochemical hypogonadism where testosterone is reduced without any clear accompanying signs or symptoms, versus a clinical hypogonadism where these negative consequences are seen. Examples of EHMC demonstrating symptoms of hypogonadism include a significant association between higher intensity levels and greater durations of endurance training on a regular basis with decreased libido [14] as well as higher AMS scores and lower BMD in endurance athletes with reduced testosterone [9]. Medical providers unfamiliar with EHMC may not prescribe the optimal treatment regimen using testosterone levels as screening criteria and not accounting for symptoms.

When symptoms of hypogonadism are apparent in conjunction with low testosterone in an endurance trained male, the health effects can be quite severe. This was illustrated by Burge et al. [25] who presented the case of a long-distance runner exhibiting low testosterone, sexual dysfunction, and very low BMD (Z-score −3 SD below age-matched controls) that had presented with pelvic stress fracture. Notably this athlete was treated with clomiphene, a medication that has anabolic properties. While the medication was successful in easing the symptoms, this would not be an option for an athlete required to avoid substances on the prohibited list determined by the World Anti-Doping Agency (WADA). Notably, the case report did not mention addressing dietary intake, volume of aerobic exercise, or other factors that may have also contributed to this athlete's low energy availability and low testosterone. Non-pharmacological strategies including addressing nutrition and energy demands are recommended for management of RED-S and the Female Athlete Triad and reducing risk for illness [18,26].

The nuance involved with identifying EHMC and the potential severity of its symptoms requires a synthesis of the current data regarding this condition. The aim of this review is to outline the current knowledge in the EHMC with the goal to guide advances in the screening, treatment, and prevention of this condition and its associated health outcomes as well as recommend future research directions.

Defining the problem

Changes in sex hormones associated to exercise were identified in both men [1] and women [27] by observing the effects of long-distance running on testosterone and amenorrhea, respectively. For example, a significant correlation was identified between weekly training mileage and incidence of amenorrhea in women [27]. In men, it was discovered that those running at least 64 km per week exhibited lower serum testosterone and prolactin than sedentary controls [1]. Indeed, these effects do appear to be particularly prevalent in those regularly engaging in high volumes of aerobic activity [1–9]; however, on average male athletes in these studies had testosterone levels measured within reference ranges. Notably, a subset of men did meet criteria for low testosterone within the full population studied. Separate investigations identified a large proportion of athletes with low testosterone in high performance ultra-endurance competitions including Western States Ultramarathon [23], the Alaska Iditarod Trail Run [21], and the Kona Ironman World Championships [22]. Collectively, these studies suggest high volume and intense aerobic activity is associated with low serum testosterone, particularly in elite athletes as has been previously suggested [28].

Early studies in this area attempted to identify the pathogenesis of the condition by investigating gonadotropin concentrations, particularly luteinizing hormone (LH). The purpose of these studies were therefore to assess whether reduced testosterone was a result of inadequate signal to the testes (a secondary hypogonadism), as indicated by low LH, or the result of under responsive testes (a primary hypogonadism) indicated by normal LH. The literature in this area has been equivocal, with some studies showing no statistically significant differences in LH [1,6,7,9], while others have [2,8]. This particular area of research is extremely challenging due to the difficulty in assessing a highly pulsatile hormone such as LH,

which requires frequent blood draws (ideally every 10–15 min) over several hours (ideally 24 h). While these studies were performed successfully in women [29], these studies in men have failed to meet the same frequency of blood draws for the same duration of time, which could explain the failure to clearly demonstrate the reduced LH pulse frequency that results in lower sex hormone concentrations in women. The inconsistent differences in LH changes may also reflect possible sex-specific differences that require long-term prospective studies to fully understand the disruptions of LH to the low energy availability state in male athletes [30].

In addition, the distribution of total testosterone, free testosterone, and sex hormone binding globulin (SHBG) have been previously investigated in EHMC [4]. In this study, both total testosterone and free testosterone were significantly reduced in endurance-trained individuals when compared to sedentary controls. In addition, no differences were seen between the groups for SHBG, which suggested an alteration in the hypothalamic-pituitary-testicular axis was responsible for the reduced testosterone as LH was not elevated in the EHMC group.

One aspect of the pathogenesis of this condition in men that has seen agreement in the literature is the role of the hypothalamic-pituitary-adrenal axis, or lack thereof [1,3,4,6–9]. It is sometimes suggested that cortisol may play a direct role in the inhibition of testosterone secretion at the testes as hydrocortisone injections can inhibit testosterone secretion without changes in LH [31]. However, cross-sectional studies comparing men exhibiting EHMC with sedentary controls have repeatedly shown differences in serum testosterone concentrations without differences in resting cortisol, suggesting that the hypothalamic-pituitary-adrenal axis is not playing any role in the development of EHMC [1,3,4,6–9].

Low serum testosterone can be seen in combination of prolonged physical demands and low energy availability. For example, when 34 men participated in an 8 day military exercise that involved high energy expenditure, low energy intake, and sleep deprivation, total testosterone concentrations dropped 50%, with multiple cases below 10.4 $nmol{\cdot}L^{-1}$ [32]. In this particular study, the intensity of the exercise was relatively low at approximately 35% maximal oxygen consumption, suggesting that the reduced testosterone was not necessarily due to the exercise stress itself, but perhaps a consequence more so of the energy deficit that high volumes of aerobic exercise can produce. Similar results have also been found previously, where both adrenal and testicular androgens were shown to decrease following 5 days of military endurance training, 1–3 h of nightly sleep and severe energy restriction [33]. These studies together demonstrate that multiple other factors, in addition to high volumes of exercise, such as sleep deprivation, energy restriction, and psychological stress can all contribute to reductions in serum testosterone.

Energy deficit, whether dictated by high energy expenditure or reduced energy intake, will likely lead to changes in body composition, particularly low body fat. It is well established that body fat is linked to testosterone concentration in the case of obesity, which is strongly associated with low serum testosterone concentrations [34]. On the other end of the spectrum, however, there is a clear link between low body fat and low testosterone, such as in RED-S [35]. In the case of RED-S, it could be argued that the association between low body fat and testosterone is a result specifically of the energy deficit rather than the fact that the body fat is reduced. However, in a clear distinction between RED-S and EHMC, there are cases where testosterone can be reduced in endurance athletes without a clear energy deficit [9]. In this study, while on average the endurance athletes did not consume more energy than sedentary controls, the large variation in energy intake suggests some athletes with low testosterone do consume adequate energy. In these cases, it is potentially the low levels of body fat that are causing the low testosterone.

Athletes in non-endurance sports including American football may also demonstrate reduced serum testosterone concentrations and associated impaired performance. Following pre-season camp in Division I collegiate American football players, measured reductions in testosterone were noted in the athletes [36]. The camp activities involved one or two practices per day and involved football related activities ranging from no contact to full contact in standard American football gear. While the testosterone did drop significantly, the average concentration did not decline below clinical levels; individual data were not presented. Similar results have also been documented previously following 9 weeks of intense strength training and conditioning in collegiate American football players [37] resulting in reductions of testosterone. On this occasion, reductions in strength as measured by barbell squat and power clean 1-repetition maximum also accompanied the reduction in testosterone. While a cause-and-effect relationship cannot be established from these data, reduced testosterone may result in fatigue and reduced performance. While it certainly appears that EHMC is more prevalent in athletes in endurance and weight class sports, these data clearly demonstrate that reductions in testosterone can impact all athletes. Additionally, collective studies suggest the established range of testosterone that is used to help diagnose hypogonadism may not be appropriate for athletes as testosterone values in the lower normal range have been associated with reduced athletic performance.

Screening guidelines

Signs and symptoms of testosterone deficiency are often hidden due to lack of access to facilities, such as dual energy x-ray absorptiometry (DXA) or blood analysis, and/or due to an unwillingness to report symptoms associated with EHMC, such as sexual dysfunction. Thus, it is important for the sport science support staff (coach, sport scientist, strength and conditioning coach, sports medicine staff) to be proactive in determining whether their athlete is potentially exhibiting the EHMC, which can be achieved through careful screening (Table 1).

The presence of symptoms of hypogonadism is essential to differentiate between simply low testosterone versus clinical hypogonadism [24]. One option is to administer one of the validated questionnaires that are commonly used in research

Table 1. A list of the athletes documented to have demonstrated reduced testosterone concentrations and potential criteria for screening athletes that may be exhibiting the exercise-hypogonadal male condition (EHMC).

Potential Athletes Affected	Screening Measures
Endurance athletes [2–9,21–23] Military populations [32,38] Strength/Power sports during high training volumes [36] Weight class sports [39]	Blood testosterone: <8 nmol·L^{-1} indicative of androgen deficiency 8–12 nmol·L^{-1}: grey zone >12 nmol·L^{-1}: likely normal [24] Questionnaires: Aging Male Symptoms [41] Androgen Deficiency in Aging Males [40] Sexual Desire Inventory [42] Bone Mineral Density: <−1.0 BMD or BMC Z-score [50]

and clinical settings, such as the Androgen Deficiency in the Aging Male (ADAM) questionnaire [40], the Aging Male Symptoms (AMS) scale [41], or the Sexual Desire Inventory (SDI) [42]. It is also important to note that the sensitivity of these questionnaires has been demonstrated to be very high (60%–97%), while the specificity is comparatively low (30%–65%) thus potentially leading to false positive findings [24]. If the athlete is exhibiting symptoms associated with testosterone deficiency, further assessments may be warranted, including an assessment of serum testosterone concentration and/or BMD.

Serum testosterone should be assessed in the rested and fasted state, following a normal night's sleep and between the hours of 7 and 10 AM [24]. The Endocrine Society defines the lower limit of normal testosterone to be approximately 9.8–10.4 nmol·L^{-1} [20], but it is important to note that a blood concentration of testosterone alone is not sufficient to diagnose hypogonadism, and that no single level of testosterone measurement unambiguously separates normal from hypogonadal men [43]. A practical approach to clinically evaluating testosterone is to consider different thresholds, such as levels below 8 nmol·L^{-1} as suggestive of androgen deficiency, 8–12 nmol·L^{-1} as a gray zone, and above 12 nmol·L^{-1} as most likely normal [24]. Free testosterone could also be measured, or estimated from total testosterone and SHBG concentrations [44] and compared to reference values [45].

Impact on BMD

One common presentation of athletes with EHMC is development of bone stress injuries or other measures of impaired bone health. This is somewhat counterintuitive as the common recommendation for enhancing bone mineral density (BMD) is undertaking weight bearing, high intensity loading forces and specifically jogging [40]. It is possible that endocrine disruption may partially explain why weight bearing exercises do not always enhance BMD. This section reviews the measures of bone health, known risk factors for low BMD in male athletes, and athlete populations at increased risk for impaired skeletal health.

Measures of bone health

Characteristics of bone health may include bone density and geometry, both measures of bone strength. BMD is most commonly used for clinical evaluation of bone health using DXA. DXA obtains measures of bone density using two-dimensional areal BMD values [46]. The International Society of Clinical Densitometry recommends indications for dual energy x-ray absorptiometry (DXA) to measure BMD for screening in men greater than 70 years of age, or men under 70 years of age with one or more risk factor including low body weight, prior fracture, high-risk medication use, or a disease associated with bone loss [47]. In the case of individuals exhibiting EHMC, due to concerns that reduced testosterone is associated with low BMD [20], screening DXA may be considered to evaluate bone health. Clinical assessment includes total body less head and lumbar spine for individuals less than 18 years of age and lumbar spine and femur values in men ages 18 and older [48]. BMD and bone mineral content (BMC) values are standardized to Z-scores in younger males using reference values based on age, sex, and ethnicity.

The threshold defined for impaired skeletal health is different between medical organizations. The International Society of Clinical Densitometry defines low bone mass for age as BMD or BMC Z-score ≤ −2.0. In contrast, the American College of Sports Medicine defines low bone mass as BMD or BMC Z-score in weight bearing female athletes of less than −1.0, as athletes typically have greater bone mass than sedentary populations [49]. Research performed in adolescent male athletes reveals a subset of male athletes may have BMD Z-score < −1.0. In one investigation in adolescent male athletes, cumulative risk factors for BMD Z-score <-1.0 include prior stress fracture, running greater than 50 km per week, consuming less than one serving of dairy daily, and having expected body weight 85% or below [50]. Given that each risk factor identified has biological plausibility to negatively influence skeletal health, the threshold of BMD Z-score less than −1.0 was proposed by investigators as being of concern for low bone mass in male athletes [50].

Influence of caloric status on bone health

A male athlete with low serum testosterone may sustain injury to multiple organ systems. The Female Athlete Triad (Triad) describes the interrelationship of low energy availability, hypothalamic functional amenorrhea, and low BMD in female athletes [26]. A parallel relationship has been suggested to occur in male athletes, substituting hypogonadotrophic hypogonadism for hypothalamic functional amenorrhea [16,17]. The concept of Triad has been expanded to the new term, Relative Energy Deficiency in Sport (RED-S). RED-S is the concept defined by the International Olympic Committee in 2014 that describes the influence of inadequate caloric intake to meet demands of sport to involve multiple physiological systems in both female and male athletes [18]. Male athletes with RED-S may have both disruptions to endocrine function including low serum testosterone and reduced bone health. While the prevalence of RED-S has not been defined in male

athletes, endurance athletes, and sports emphasizing leanness appear to be at heightened risk of low energy availability [51].

Athlete populations at risk for impaired skeletal health

Low BMD in the male athlete is often multifactorial including sport-specific skeletal loading and other behaviors associated with sport participation. Both female and male athletes who participate in high impact and multidirectional loading sports appear to have the highest measures of BMD and strength [52]. In a review of male athletes [16], weight class sports and endurance sports of cycling and running may have higher prevalence of low BMD associated with reduced testosterone and low energy availability. Studies to date reported 19%–40% of runners [53–55] and 25%–89% of cyclists [56,57] have impaired bone health. Interestingly, despite demonstrating several osteoporosis risk factors, 40 dancers, including 10 men, demonstrated elevated cortical and trabecular BMD [58]. True prevalence cannot be determined from these studies given the wide age range and ability level of the subject populations and the fact that measures of low BMD differed between investigations.

Treating low BMD

Impaired skeletal health in athletes requires careful consideration for treatment. Authors have consensus that testosterone replacement to address low BMD is not an appropriate treatment option. Testosterone is on the banned substance list for World Anti-Doping Agency and other sport governing bodies due to being performance enhancing and concerns of safety for athletes. Furthermore, clear guidelines to determine what constitutes a normal range for testosterone in male athletes do not currently exist. Notably, female athletes were historically treated with hormonal therapy; non-pharmacological treatment for low estradiol in female athletes is currently recommended for initial treatment [18,26]. We propose treating low testosterone in male athletes through behavioral strategies focused on diet, sleep, and modified exercise.

Nutrition

Addressing energy deficiency in male athletes with low testosterone should be the primary target for intervention to improve overall skeletal health. While adequate energy availability is unknown in male athletes, the target of 45 kcal/kg/fat free mass per day has been suggested as being sufficient for reproductive and metabolic hormone function in women [59]. Targeting increased energy availability through a combination of increased energy intake, reduced exercise energy expenditure, or both seems prudent to address energy availability in male athletes [51]

In addition to caloric demands, targets of micronutrients including vitamin D, calcium, and magnesium are important for skeletal health [60,61]. We recommend meeting the Institute of Medicine 2010 guidelines of 1,300 mg calcium intake for boys 9–18 and 1,000 mg daily for men ages 19–70 [60]. Meeting calcium needs through diet may facilitate greatest gains in skeletal health [50]. Vitamin D target is 600 IU daily and is something that can be achieved through both supplementation and diet [60]. Vitamin D is important for maintaining skeletal health and calcium absorption. The vitamin D status for an athlete is commonly measured using serum 25-hydroxy vitamin D measures; higher serum values do not clearly lead to higher BMD in male athletes [62]. Additionally, a separate trial in male jockeys of calcium and vitamin D identified improvements in bone turnover marker but failed to identify measurable gains in skeletal health. These studies highlight that while micronutrients are necessary for skeletal health, they are not sufficient alone and should be combined with increasing energy availability and other means to improve bone health.

Sleep

The influence of sleep on skeletal health in athletes is unknown. However, acute changes in sleep quality have been shown to lead to rapid loss in BMD and reduced skeletal strength. Ben-Sasson et al (1994) identified that 40% of male military recruits randomized to 62 h of sleep deprivation or sleeping 6 h for three nights in a vertical position had 5% reductions in skeletal mass in a seven day period [63]. Finestone and Milgrom applied a strategy of 6 h of sleep requirement and reduced military marching with an observed 62% reduction in stress fractures in 8 weeks of basic military training [64]. Investigators also noted that the grade of injury was reduced, and gains of 0.5% in BMD value were observed in men who did not sustain injuries [64]. Optimal duration of sleep to facilitate gains in bone mass has not been adequately described to provide specific recommendations and the mechanism by which sleep may aid skeletal health is unclear; nonetheless, restful and uninterrupted sleep are reasonable strategies to consider for the general health of athletes.

Weight-bearing activities

Given that most populations of athletes affected with low bone mass are in endurance sports or weight class sports, encouraging impact loading activities is prudent to stimulate gains in bone mass. In addition to higher measures of bone strength and reduced risk for subsequent stress fracture [52,65–67], participation in team sport may stimulate gains in testosterone levels. An investigation in adolescent male soccer players identified gains in serum testosterone levels over sedentary controls over course of two seasons of soccer participation [68].

Treating low T

The medical standard of care for treatment of hypogonadism in men typically revolves around the use of pharmaceutical agents to address low serum testosterone, either through exogenous testosterone or medications to stimulate the production of testosterone. Competitive athletes suffering from EHMC may not use pharmacological treatment per WADA regulations. Testosterone and gonadotropin stimulator agents fall into the WADA 'List of Prohibited Substances and

Methods' (categories: S1 *Anabolic agents*; S2, *Peptide hormones, growth factors related substances, and mimetics*) which if used constitutes a doping violation by the athlete [69]. WADA does have a Therapeutic Use Exemption (TUE) option by which athletes can take banned drugs for medical reasons, but the scenario by which EMHC occurs as a consequence of exercise training does not fit into the circumstances by which WADA would grant a TUE to an athlete.

Although anabolic agents are not permitted by WADA, if the athlete is suffering from low BMD as result of the EHMC, bisphosphonates may be a viable option that are permitted by WADA, particularly as males do not have the same concerns for teratogenicity as women, although they have not been specifically studied in EHMC. However, as is recommended to those exhibiting the Female Athlete Triad, pharmacological management is to be considered if there is a lack of response to non-pharmacological therapy for at least 1 year and if new fractures occur during the non-pharmacological management [26], therefore we suggest a similar recommendation to men exhibiting low BMD with EHMC. This is particularly emphasized by the lack of reduction in stress fracture risk that occurred with bisphosphonate use in military recruits [70], perhaps due the failure to handle the underlying cause of the increased stress fracture risk.

There have been reports in the scientific literature of increased total or free testosterone concentrations through legal supplements, such as D-aspartic acid [71] and fenugreek [72,73]. However, it is important to note that these changes have been negligible and that there is insufficient evidence to support their use, as well as their failure to address the underpinning physiology and behaviors from sport that are causing low testosterone. Therefore, we suggest current treatment for EHMC should focus on lifestyle factors and non-pharmacological treatment.

Concluding remarks

Low serum testosterone with associated symptoms may be of concern for the health of male athletes. This condition, termed EHMC is often seen in endurance athletes, but low testosterone has also been identified in anerobic sports during high training volumes, such as summer conditioning in American football. It is important to note that serum testosterone alone does not define hypogonadism. Testosterone is often within normal range in most athletes. EHMC is characterized as a combination of low testosterone for the individual athlete with hypogonadal symptoms, which may include fatigue, sexual dysfunction and infertility, as well as reduced BMD. We recommend sports medicine providers screen for EHMC to address the health consequences. Evaluation may include measuring testosterone levels in early morning and screening for symptoms of hypogonadism. Measuring bone density through DXA is also important to identify athletes with impaired bone health. Treatment is recommended for athletes with symptoms that are attributed to reduced testosterone concentrations. However, testosterone replacement or use of other hormonal treatments such as clomiphene citrate is not recommended as this does not address the underlying etiology of the condition and is prohibited by WADA. Treatment should be centered on non-pharmacological strategies including nutritional intervention, and modifications in training volume to improve energy availability and support normal hormonal function in the male athlete.

Funding

This manuscript was not funded.

Declaration of interest

The authors have no relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties. Peer reviewers on this manuscript have no relevant financial relationships to disclose.

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