The Male Athlete Triad—A Consensus Statement From the Female and Male Athlete Triad Coalition Part 1: Definition and Scientific Basis

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Abstract: The Male Athlete Triad is a syndrome of 3 interrelated conditions most common in adolescent and young adult male endurance and weight-class athletes and includes the clinically relevant outcomes of (1) energy deficiency/low energy availability (EA) with or without disordered eating/eating disorders, (2) functional hypothalamic hypogonadism, and (3) osteoporosis or low bone mineral density with or without bone stress injury (BSI). The causal role of low EA in the modulation of reproductive function and skeletal health in the male athlete reinforces the notion that skeletal health and reproductive outcomes are the primary clinical concerns. At present, the specific intermediate subclinical outcomes are less clearly defined in male athletes than those in female athletes and are represented as subtle alterations in the hypothalamic–pituitary–gonadal axis and increased risk for BSI. The degree of energy deficiency/low EA associated with such alterations remains unclear. However, available data suggest a more severe energy deficiency/low EA state is needed to affect reproductive and skeletal health in the Male Athlete Triad than in the Female Athlete Triad. Additional research is needed to further clarify and quantify this association. The Female and Male Athlete Triad Coalition Consensus Statements include evidence statements developed after a roundtable of experts held in conjunction with the American College of Sports Medicine 64th Annual Meeting in Denver, Colorado, in 2017 and are in 2 parts—Part I: Definition and Scientific Basis and Part 2: The Male Athlete Triad: Diagnosis, Treatment, and Return-to-Play. In this first article, we discuss the scientific evidence to support the Male Athlete Triad model.

Key Words: female athlete triad, energy deficiency, low energy availability, male athlete, young athlete, bone health, distance runners

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INTRODUCTION

Over the past 3 decades, our understanding of the Female Athlete Triad has evolved in response to research advances over time, such as the establishment of prevalence data, the expansion of the spectra to include the subclinical expressions of each of the 3 conditions, and, most importantly, the causal role of energy deficiency/low energy availability (EA) on reproductive dysfunction as determined through short-term and long-term laboratory experiments including randomized controlled trials (RCTs). In male athletes, a Triad-like syndrome has been frequently noted in the literature. With increasing awareness, there has been a recent uptick in Triad-like research in men. Because the causal role of low EA in the modulation of reproductive function has been established in the female and male literature[1–7] and because the observations thus far in men, like women, reinforce the notion that skeletal health and reproductive outcomes are the primary clinical concerns, the authors believe that the science in men is best built on the scaffolding of the Female Athlete Triad model. This point is reinforced given that most of the scientific evidence that forms the foundation for the emerging concept of Relative Energy Deficiency in Sport is limited to the scientific evidence supporting the Triad model in both women and men. As such, the Female and Male Athlete Triad Coalition present a model for the Male Athlete Triad with the scientific evidence available to date. Because of the comprehensive nature of each topic, we have elected to publish 2 articles focused on the Male Athlete Triad. The first article, "The Male Athlete Triad: Definition and Scientific Basis," will present the scientific evidence to support the model, and the second article, "The Male Athlete Triad: Diagnosis, Treatment and Return-to-Play," will present the clinical diagnostic criteria and the risk assessment criteria for a clearance and return-to-play model.

The Female and Male Athlete Triad Coalition convened an expert panel in conjunction with the American College of Sports Medicine (ACSM) 64th Annual Meeting in Denver, Colorado, May 30, 2017, to consolidate evidence-based data

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and develop recommendations for the Male Athlete Triad and this consensus statement. We drew on emerging data developments and the experience of a diverse group of clinicians and basic scientists. The meeting began with an introduction of the problem and case presentations, followed by selected presentations from invited expert panel members and a panel discussion on various clinical questions and recommendations for team physicians and other sports medicine professionals in preparation for a consensus article.

Invitees to the expert panel included recognized national and international representatives, including expert clinical and basic scientists who have published extensively on the Triad and related topics, particularly as it relates to the male athlete. The panel included participants from a variety of medical and scientific experts in applied physiology, kinesiology, nutrition, women's and men's health, exercise physiology, pediatric and adult medical endocrinology, bone health, reproductive medicine, clinical sports medicine, and epidemiology.

We aimed to examine, consolidate, and implement practical, sport-specific strategies for diagnosing, managing, and preventing the manifestations of the Triad in male athletes guided by evidence-based science and expert opinion. The 2-part Female and Male Athlete Triad Coalition Consensus Statement on the Male Athlete Triad and accompanying figures represent the key themes discussed at the meeting, with scientific support and summary of vital clinical implications for male athletes.

DEFINITION OF THE MALE ATHLETE TRIAD

The Male Athlete Triad is a syndrome of 3 interrelated conditions, including energy deficiency/low EA, impaired bone health, and suppression of the hypothalamic–pituitary–gonadal (HPG) axis (Figure 1). Energy deficiency/low EA refers to the level of energetic status where one or more of the following compensatory metabolic adaptations have occurred: suppression of resting metabolic rate (RMR) normalized for body size or fat-free mass (FFM),[8] a loss of body weight that is associated with a new chronically low set point,[8,9] low body mass index (BMI),[10,11] and/or suppression of metabolic hormones, such as triiodothyronine (T_3) and leptin,[6,7,12] reflecting the prolonged failure to consume adequate energy to support energy expenditure. At the optimal, "healthy," end of the continuum, energy intake (EI) is sufficient to meet the combined needs of exercise and all physiological processes; bone health is appropriate for age, sex, and exercise exposure; and the HPG axis is functioning normally. As energy deficiency ensues and EA is reduced, there is a progressive deterioration in reproductive health and bone outcomes. At the extreme, "unhealthy," end of the continuum, clinical outcomes include energy deficiency/low EA associated with or without eating disorders or disordered eating, hypogonadotropic hypogonadism, oligospermia, decreased libido, and osteoporosis with or without bone stress injury (BSI). At present, and in contrast to the Female Athlete Triad model, the specific intermediate subclinical outcomes are less clearly defined in men and are represented as subtle alterations in the HPG axis and increased risk for BSI. The degree of energy deficiency/low EA associated with such alterations remains unclear. However, the available data suggest a more severe energy deficiency/low EA state is needed to affect reproductive and skeletal health in the Male Athlete Triad than in the Female Athlete Triad,[7,12–15] although additional research is needed to further clarify and quantify this association. The bidirectional arrows indicate that the outcomes associated with energy deficiency/low EA can be reversed, with the exception of bone outcomes, for which additional research is needed to clarify.

The data available to date are presented below and include summary statements regarding the strength of scientific evidence. Because of challenges associated with evidence being drawn from studies using a variety of methodologies, we have used a taxonomy in which both RCT and observational data are considered important and that has been used in ACSM position stands and by the Agency for Healthcare Research and Quality.[16,17] The specific evidence scoring criteria are as follows:

1. Evidence Level A: Consistent pattern of findings on the basis of substantial data from RCTs and/or observational studies.
2. Evidence Level B: Strong evidence from RCT and/or observational studies but with some inconsistent results from the overall conclusion.
3. Evidence Level C: Evidence from a smaller number of observational and/or uncontrolled or nonrandomized trials, which is generally suggestive of an overall conclusion.
4. Evidence Level D: Insufficient evidence for categories A to C; panel consensus judgment.

ENERGY DEFICIENCY/LOW ENERGY AVAILABILITY IN MALE ATHLETES

What Is the Evidence for Chronic Energy Deficiency/Low Energy Availability in Male Athletes?

Objective indicators of compensatory adaptations to chronic energy deficiency include changes in body weight and body composition,[8–11] the suppression of RMR normalized for body size or FFM,[8] and changes in circulating metabolic hormones, such as suppressed T_3 and leptin concentrations.[6,7,12] In addition to these measures, EA has been used by many investigators and is conceptually described as the energy remaining for bodily processes after the energy cost of exercise training is accounted for.[7,18] This expression of EA from Loucks[4,7,18,19] has persisted in the Triad literature despite the lack of consideration of nonpurposeful energy expenditure, which can be quite variable (and significant in many cases), concerns regarding the accuracy with which it can be determined in free living athletes, and the reliance of this measure on self-reported variables, especially dietary EI.[20] Similarly, assessments of FFM may be specific to laboratory settings but may be calculated based on the weight of the athlete in kilograms, as well as the percent body fat, which may be estimated by dual X-ray absorptiometry, air displacement plethysmography, skinfold measurements, Bod Pod, or bioelectrical impedance.[21,22]

In this consensus article, we provide an operational definition of the concept of "low" EA. We shall use the terminology of low EA to refer to a level of EA that reflects energy deficiency such that there is evidence that compensatory metabolic adaptations are present that reflect the failure to consume adequate energy to support energy expenditure. The compensatory adaptations include the suppression of RMR normalized for body size or FFM,[8] a loss of body weight

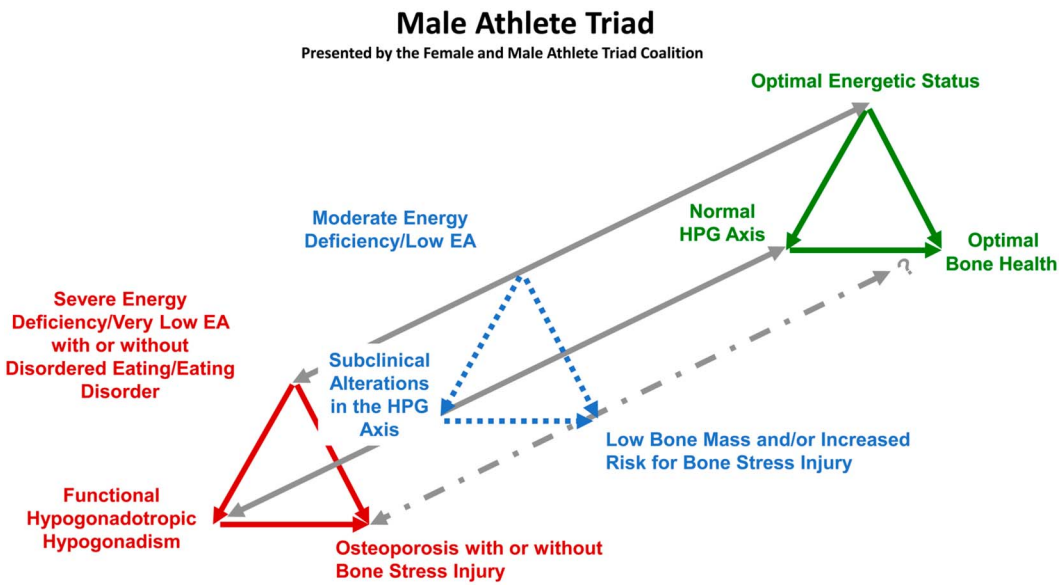

Figure 1. Model of the Male Athlete Triad. The unidirectional arrows from energetic status/EA toward bone health and the HPG axis indicate the causal role of EA on both bone health and HPG axis function. Similarly, the unidirectional arrow from HPG toward bone health indicates the causal effect of reproductive hormones on bone health. Furthermore, the bidirectional arrows along each continuum of severity represent the "reversibility" of the condition such that an individual can improve or worsen over time. The line showing reversal of bone health outcomes is dashed with a question mark because the reversal of BMD is less known, and more research is needed. Notably, with the Male Athlete Triad, the subclinical and clinical sequelae present at lower EA levels than what is often required for the development of health consequences in exercising women.

that is associated with a new chronically low set point,[8,9] low BMI,[10,11] and or suppressed metabolic hormones, such as T_3 and leptin.[6,7,12] These changes occur over weeks to months and are therefore reflective of adaptations to chronic energy deficiency.

Causal evidence that energy deficiency impairs reproductive function is available.[5–7,23] In women, Loucks and Thuma[7] identified an EA threshold of 30 kcal/kg FFM/d, below which changes in menstrual function ensued. Although more research is necessary, this particular EA threshold of 30 kcal/kg FFM/d failed to predict the onset of menstrual disturbances with energy deficiency/low EA-associated exercise training for 3 months in young, untrained, ovulatory women[12] and, in cross-sectional reports of menstrual status, it failed to discriminate menstrual status of varying categories.[24] As such, recent data do not support the idea that there is a particular threshold below which reproductive outcomes are impaired.[25] However, data do support a strong relationship between EA and menstrual suppression, such that as EA decreases, the likelihood of menstrual disruption increases.[24] In men, the literature to date has not identified a clear threshold of EA below which reproductive function impairment is observed. Therefore, at this point in the development of the Male Athlete Triad model, there are not sufficient data to provide a quantitative definition of low EA in male athletes. Thus, this article will use the term "chronic energy deficiency/low EA" to refer to the compensatory metabolic adaptations that occur during prolonged periods of inadequate dietary EI relative to total energy expenditure.

Currently, no standard protocol exists for the measurement of EA among athletes in the field. Although we recommend that EA is a measure best reserved for laboratory assessment, it can be useful for the nutritionist or healthcare professional working with an athlete in a clinical setting because treatment goals based on improving EA may be more acceptable to the athlete than goals based on increasing body weight. The challenges faced by investigators and clinicians aiming to measure EA are described in detail in a recent review and include the lack of a universal protocol, the resources needed to estimate each component of the EA equation, and error associated with estimating each component, particularly dietary EI.[20] The limitations of assessing EA notwithstanding, investigations that estimate EA in male athletes can nonetheless provide some initial insights into the subpopulations at risk, and if low EA is referred to, it is presumably referring to an EA level that reflects an energy deficiency. In the clinical article "The Male Athlete Triad: Diagnosis, Treatment, and Return-to-Play," we will address the usefulness of EA assessments when counseling athletes and suggest communication strategies.

Cross-sectional data identifying EA below 30 kcal/kg FFM/d in male athletes have been reported, particularly for athletes who participate in sports that have a leanness component such as weight class[26,27] and endurance sports,[28–31] although an exact definition for "low" EA is still to be determined in male athletes. The prevalence of EA below 30 kcal/kg FFM/d in exercising men is similar to what has been reported in women.[26,29,31] For example, the prevalence of EA below 30 kcal/kg FFM/d is 56% versus 51% in adolescent male and female athletes across a variety of sports,[26] 25% versus 31% in male and female endurance runners/race walkers,[29] and 42% versus 29% in male and female cross-country runners.[31] Notably, the methods used to assess EA vary greatly among published studies making direct comparisons difficult and underscoring the inherent problems in the assessment of EA, which is dependent on several self-report measures, that is, EI and exercise energy expenditure (EEE).

Other investigators do not report EA specifically, but rather have included concurrent assessments of EI and EEE in

athletes. Male athletes, particularly those participating in leanness sports, often have low EI according to the Institute of Medicine Daily Recommended Intakes or Food and Agriculture Organization of the United Nations/World Health Organization recommendations.[32] Although it is recognized that accurate recording of food intake is challenging,[33] estimates available among male athletes, especially in sports requiring leanness or with weight limitations, often suggest EI of 500 to 1000 kcal/d lower than that required for EEE[32,34–36] or the recommended EI.[37] For example, in a systematic review, it was reported that among studies that measured EI and EEE (n = 55; 1195 athletes) male endurance athletes exhibited, on average, an energy deficit of 577 kcal/d.[34] Lower EI relative to EEE has also specifically been reported in professional male endurance athletes including runners (2987 ± 293 kcal/d intake vs 3605 ± 119 kcal/d expenditure),[35] and cyclists (3224 ± 358 kcal/d intake vs 4562 ± 979 kcal/d expenditure).[36] Furthermore, after a 54-hour 1230-km ultra-endurance cycling event, male athletes averaged an energy deficit of 2468 kcal/24 hours.[38]

Energy deficiency and EA below 30 kcal/kg FFM/d have been documented in exercising men.[39] The results from 2 investigators[13,14] who manipulated EI and EEE in men to test a low level of EA (15 kcal/kg FFM/d) and an adequate level of EA (40-45 kcal/kg FFM/d) did not report many of the metabolic hormone perturbations that have been previously reported by Loucks et al[4,7,18] in women with an EA of 30, 20, and 10 kcal/kg FFM/d (ie, reductions in T_3, leptin, and insulin). For example, Papageorgiou et al[13] manipulated exercise and EI for 4 to 5 days in exercise-trained men, as was performed in the study by Loucks et al in sedentary women. The low set point of 15 kcal/kg FFM/d failed to induce any significant reductions in T_3, leptin, insulin-like growth factor 1 (IGF-1), or insulin.[13] In a very similar study, Koehler et al[14] manipulated both EI and EEE and failed to report any significant reductions in T_3, IGF-1, or testosterone concentrations after exposure for 4 days to 15 kcal/kg FFM/d (vs 40 kcal/kg FFM/d), but insulin and leptin concentrations were significantly reduced at an EA of 15 kcal/kg FFM/d. As such, if an appropriate definition of "low" EA in men is to be considered, more rigorous scientific evidence of the magnitude of energy deficiency/low EA associated with indices of metabolic compensation (ie, inducing metabolic and hormonal adaptations) will be required. Similarly, understanding the influence of time spent in varying degrees of energy deficiency/low EA on indices of metabolic compensation is needed as well. The data available to date are very limited but are in contrast to similar studies in women. In both the Papageorgiou et al[13] and the Koehler et al[14] studies in men, study participants were exercise-trained men, whereas in the studies by Loucks,[4,7,18] the participants were sedentary women. The extent to which this fact affected the outcomes remains unclear, and more research is necessary to clarify these issues.

Other data that demonstrate better evidence of energy deficiency affecting metabolic hormones in men include the multistressor studies in Army Rangers during an 8-week training period of exposure to desert, swamp, mountain, and forest conditions during severe energy restriction.[40] After the severe energy restriction (~1100 kcal/d) and extreme environments, dramatic reductions in concentrations of IGF-1 (−57%) and T_3 (−15%) were observed.[40] Although Rangers were exposed to multiple stressors simultaneously, the specific effects of energy restriction are highlighted by the findings that, within a subgroup of Rangers who were fed a supplemental ~400 kcal/d, declines in IGF-1 and T_3 were attenuated.[40] Finally, in all Army Rangers, metabolic hormone concentrations were restored to pre-exposure concentrations rapidly within 1 to 4 weeks, after cessation of the 8-week training.[40] As such, these data provide evidence that energy deficiency/low EA in men induces metabolic perturbations, although the energy deficits required to induce metabolic hormone perturbations need to be quantified and men seem to require more severe energy deficits than women before decrements to metabolism are observed.

Evidence Level B. Severe energy deficit (~1100 kcal/d) in men is associated with alterations in metabolic hormones indicative of metabolic compensation. Data are not consistent for alterations in these hormones in states of low EA (defined as <15 kcal/kg FFM/day).

What Are the Pathways to Energy Deficiency/Low Energy Availability in Male Athletes?

The psychological and behavioral underpinnings of inadequate EI and/or excessive exercise that contribute to energy deficiency/low EA also warrant discussion. As with female athletes, male athletes may exhibit multiple pathways, that is, intentional weight loss, inadvertent undereating, disordered eating, and clinical eating disorders, that contribute to their failure to consume adequate volume of calories to meet the needs of EEE. However, the underlying motivations that contribute to undereating may differ between men and women, and particularly male and female athletes; therefore, relying on the same criteria that have been applied to exercising women may not be appropriate for male athletes. For example, high cognitive dietary restraint,[41–43] as measured by the Three-Factor Eating Questionnaire,[44] and high drive for thinness,[45,46] as measured by the Eating Disorders Inventory,[47] have both been used as surrogate measures of energy deficiency and as screening tools to identify women at-risk for disordered eating. Currently, scarce information is available regarding how cognitive restraint and drive for thinness may relate to disordered eating and energy deficiency in exercising men, and what little information is available suggests that men have lower drive for thinness subscale scores than women.[48,49] Furthermore, because of differing societal and cultural body ideals between the sexes, such as men wanting to improve muscularity[50] or striving for a combination of muscularity and thinness,[51] disordered eating behaviors may manifest differently in men versus women.[52] Notably, both muscularity and thinness were independently and positively associated with disordered eating in college-aged men.[51] A tool has been developed to identify the interaction between muscularity and thinness, which has been referred to as a "drive for leanness"—this may be relevant to a Triad-like condition in men.[53] Much additional work must be conducted to better understand disordered eating behaviors in men.

To date, the most comprehensive information available regarding the prevalence of eating disorders in male athletes identified that 8% of athletes (55/687) were at-risk and subsequently met the diagnostic criteria for an eating disorder.[54] Sports with the highest prevalence of male athletes who met the criteria for an eating disorder were those categorized as antigravitation sports (ie, high/long/triple jump, pole vault, and rock climbing, 22%, n = 8/37) and

weight-class sports (ie, wrestling, martial arts, and weight lifting, 18%, n = 14/79).[54] Similarly, a high prevalence of disordered eating or extreme body weight-cutting behaviors has been reported in cycling and in weight-class and leanness sports.[54–60]

Evidence Level C. Exercising men, particularly those in leanness sports, are at risk for developing disordered eating/eating disorders; however, the specific eating behaviors may differ from those observed in exercising women.

HYPOGONADOTROPIC HYPOGONADISM IN MALE ATHLETES

Several levels of evidence are available to demonstrate that acute exercise and high-volume endurance exercise have suppressive effects on the HPG axis resulting in hypogonadotropic hypogonadism. This is demonstrated by (1) suppressed testosterone (T) and luteinizing hormone (LH) concentrations and pulse frequency, particularly in athletes engaged in lean sports,[61–65] after acute bouts of prolonged exercise[38,66–68] with T concentrations often, but not always, observed within the subclinical to clinical range; (2) decreased T and responsiveness of gonadotropins to gonadotropin-releasing hormone (GnRH) stimulation in male athletes after exercise training[40,69,70]; (3) negative changes in spermatogenesis in high-volume male athletes and in men after exercise training[69,71–74]; and (4) self-report data of decreased libido and sexual desire in men participating in high-volume exercise training.[61,75] The levels of evidence available to support the occurrence of hypogonadotropic hypogonadism in male athletes include (1) acute responses to prolonged exercise, (2) cross-sectional reports, and (3) RCTs. This evidence is summarized in Tables 1 and 2.

Which Specific Reproductive Hormones Are Suppressed in Male Athletes?

Ironman events (ultra-endurance running and cycling races of ~160-1200 km) provide evidence for the effects of acute responses to prolonged, strenuous exercise on the suppression of the HPG axis in men, as characterized by reduced serum total T concentrations,[38,66–68] often in the subclinical[67] and clinically low range,[38,66] and reduced LH.[62,67] In cross-sectional reports where athletes, primarily runners, are compared with controls, suppression of total T and mean LH is also observed, although T is often still within the normal range. For example, runners training >80 km/wk had lower total T and lower LH mean pulse amplitude and mean area under the 6 hours LH curve compared with controls.[62] However, male marathoners (running 125-200 km/wk) demonstrated decreased LH pulse frequency and amplitude and suppressed LH responsiveness to GnRH, but no difference in T levels compared with healthy controls.[70] In other studies of male runners with a lower training load of ~80 km/wk, no changes in LH pulse frequency were observed when compared with controls.[62,76] Other cross-sectional

TABLE 1. Testosterone Concentrations in Exercising Men

Study	Pre	Post	Recovery	Control	Significance
Short-term restricted EA					
Koehler et al[14]					
Caloric restriction only	18.72 ± 1.42	17.44 ± 2.95			None
Caloric restriction + exercise	18.27 ± 1.59	15.46 ± 3.33			None
Acute exercise bouts					
Kraemer et al[64]					
Runners	12.32 ± 1.41	6.96 ± 1.01			Pre vs post
Cyclists	13.81 ± 1.30	5.59 ± 1.53			Pre vs post
Kupchak et al[65],*	14.9	10.9	13.5		Pre vs post Post vs recovery
Hooper et al[66],*	14	15	10		Pre vs recovery
Multistressor environment					
Friedl et al[38],†	16.3 ± 1.6	2.2 ± 0.9	19.3 ± 3.2		Pre vs post Post vs recovery
Cross-sectional					
McColl et al[60],*	17.3 ± 1.0	13.8 ± 1.0		29.4 ± 1.0	Pre vs post
MacConnie et al[68]	21.5 ± 1.1			20.8 ± 1.2	None
Wheeler et al[63],*	24.0			28.0	Runners vs controls
Hooper et al[59]	9.2 ± 0.8			16.2 ± 1.2	Runners vs controls
RCT					
Safarinejad et al[67]					
High intensity	13.2 ± 0.4	8.6 ± 0.4	13.8 ± 0.4		Pre vs post
Moderate intensity	13.4 ± 0.4	11.4 ± 0.4	13.7 ± 0.4		None

Data presented as mean ± SEM or mean. The units of total testosterone are expressed as nmol/L.
** Approximations of concentrations based on available data and figures; SEM not available.*
† Subset of completers who had a follow-up visit during recovery.

TABLE 2. Semen Characteristics in a Subset of Studies of Exercising Men

Study	Volume (mL)	Concentration (10^6/mL)	Total No. (10^6)	Normal Forms (%)
Vaamonde et al[72]				
Active controls	3.2 ± 0.9	61.0 ± 23.0	191.8 ± 73.4	15.2 ± 1.2
Water Polo	3.4 ± 1.3	58.0 ± 24.4	196.6 ± 85.4	9.7 ± 3.0*
Triathletes	2.9 ± 0.9	48.2 ± 14.7*	141.3 ± 58.0*	4.7 ± 2.2*
De Souza et al[69]				
High-mileage runners	4.1 ± 01.7	88.5 ± 49.0*	352.0 ± 230.4	40.9 ± 6.6
Low-mileage runners	3.5 ± 1.8	127.2 ± 96.6	317.8 ± 249.9	46.4 ± 13.2
Controls	2.5 ± 1.6	175.5 ± 78.7	375.9 ± 186.1	47.0 ± 10.4
Safarinejad et al[67]				
High intensity				
Pre	2.7 ± 1.4	66.2 ± 14.6	196 ± 32.6	19.2 ± 2.6
Running	2.7 ± 1.6	35.4 ± 4.2*	106 ± 20.8*	14.6 ± 2.4*
Recovery	2.7 ± 1.7	64.8 ± 4.2	188 ± 31.2	18.8 ± 2.1
Moderate intensity				
Pre	2.8 ± 1.3	64.4 ± 14.4	197 ± 32.4	19.3 ± 2.7
Running	2.8 ± 1.1	56.8 ± 3.6*†	161 ± 31.4*†	16.2 ± 2.1
Recovery	2.7 ± 1.4	62.8 ± 3.4	191 ± 30.4	18.4 ± 1.8

Data presented as mean ± SD.
** Significantly different from controls/pre.*
† Significantly different from high-intensity group at same time point.

reports observe similar findings,[63,65] except for 1 study in endurance-trained men averaging >450 min/wk of training (in running, swimming, and cycling) that failed to observe any differences in LH pulse frequency or amplitude compared with sedentary controls.[64]

A limitation to the interpretation of these studies is that energy status and/or EA is not typically quantified in studies of reproductive function. Presumably, the effects of the acute prolonged bouts of exercise and cross-sectional comparisons of male athletes and nonathletes were associated with energy deficiency/reductions in EA, subsequently translated to suppression of the HPG axis. One such example in long-distance runners reported that EA of runners (27.2 kcal/kg lean body mass [LBM]/d) was lower than that observed in controls (45.4 kcal/kg LBM/d) in concert with subclinically low T.[61] In another study of a 54-hour 1230-km ultra-endurance cycling event, athletes averaged an energy deficit of 2468 kcal/24-hour and demonstrated severe reductions in serum T in the clinically low range.[38] Further research that carefully documents EA changes after acute exercise and chronic training is necessary to clearly identify whether prolonged endurance exercise has suppressive effects on the male reproductive axis in the absence of energy deficiency/low EA.

Other prospective data supporting a link between energy status and reproductive function in men are the aforementioned 8-week multistressor studies of Army Rangers.[40] As a reminder, 1 group of Rangers was exposed to extreme energy restriction with deficits averaging ~1100 kcal/d and experienced reduced mean LH concentration and severely reduced concentrations of T (4.5 ± 3.9 nmol/L), which fell below the normative range and within a clinically low range (see Table 3 for normal to clinically low T concentrations in men[77–83]) at the completion of the 8-week training regimen.[40] However, in the group of Rangers receiving supplemental calories (400 kcal), suppression of both T and LH was avoided. Moreover, at completion of the training, recovery of both T and LH concentrations was observed within 1 week in the group who did not receive supplemental calories,[40] providing strong support for the notion that reproductive hormone concentrations are dependent on energy status.[1,2]

Evidence in support of hypogonadotropic hypogonadism also comes from RCTs, including both short-term and long-term training studies. To date, 1 long-term RCT of 286 previously sedentary men consisted of exercise training at either moderate-intensity (60% maximal oxygen uptake [$\dot{V}O_2$max]) or high-intensity (80% $\dot{V}O_2$max) treadmill running 5 d/wk for 120 minutes each session for 60 weeks, followed by a period of 36 weeks of light training to assess recovery.[69] Both serum total and free T concentrations decreased by week 12, sex hormone-binding globulin increased in both training groups, and serum LH and follicle-stimulating hormone (FSH) decreased significantly only in the high-intensity group.[69] Notably, serum T levels were suppressed to concentrations in the subclinical "gray zone" in both groups. In all cases, hormone concentrations improved to their pre-exercise concentrations during the 36-week recovery period.[69] These studies, however, did not include assessments of energy and/or metabolic status.

As discussed in the EA section, 2 short-term (4-5 days) experiments[13,14] are available to date that have manipulated EEE and EI to induce low EA in men in a manner similar to the studies conducted by Loucks et al[4] in women. In both short-term experiments, EA was manipulated to a deficit level of 15 kcal/kg FFM/d, a level generally considered severely low, and was compared with a replete energetic level of 40 to 45 kcal/kg FFM/d. Only 1 study assessed T concentration and failed to report significant reductions after 4 to 5 days of exposure to EA of 15 kcal/kg FFM/d.[14] This is surprising given that EA of 15 kcal/kg FFM/d is very low and well below the threshold

TABLE 3. Classification of Testosterone Concentrations in Men*

Total Testosterone Concentration	Classification
<8 nmol/L (<230 ng/dL)	Clinical testosterone deficiency
8-12 nmol/L (230-350 ng/dL)	Subclinical gray zone
>12 (>350 ng/dL)	Likely normal

*These ranges are found in multiple guidelines, including those from the International Society for Sexual Medicine, the British Society for Sexual Medicine, and the European Association of Urology, and are widely referenced in the literature.[75–79] However, the Endocrine Society and the American Urological Association recommend a single cutoff value of <10.4 nmol/L or 300 ng/dL.[80,81]

level of 30 kcal/kg FFM/d, where Loucks et al[4] proposed that perturbations to reproductive function in women are observed. These data suggest that, in men, perturbations to reproductive function may require reductions in energy status that are more severe (ie, lower than 15 kcal/kg FFM/d) or more prolonged and at very strenuous levels of exertion. Further testing is warranted to clarify this point. Several,[68] but not all,[61,63] investigators have observed elevated concentrations of serum cortisol in association with reductions in T.

It is notable that the suppressed total T concentrations observed in male athletes often fall within the "gray zone" of 8 to 12 nmol/L or 230 to 350 ng/dL. Cutoff values for "low T" are presented in Table 3, and the clinical significance of this subclinical "gray zone" has been debated.[68,80] In Ironman athletes, only 4 of 22 male athletes had T concentrations in the clinically low range (<8 nmol/L) at baseline, with ~one-third of athletes being clinically low immediately postrace.[68] Only in 100-mile ultramarathon events, such as the Alaska Iditarod Trail Run[66] and the Western States Ultramarathon,[67] were T levels more frequently found to be clinically low, although some athletes had concentrations within the subclinical "gray zone."

Evidence Level B. High-volume exercise training and bouts of acute prolonged exercise in men can be associated with reductions in T concentration, often falling into the subclinical "gray zone" and sometimes into the clinically low zone.

What Are the Effects of Exercise or Energy Deficiency/Low Energy Availability on Spermatogenesis?

The evidence available to support that exercise perturbs spermatogenesis in male athletes includes cross-sectional reports and short-term and long-term RCTs. Results are summarized in Table 2. It is important to note that these studies do not differentiate between the effects of exercise alone and effects of energy deficiency/low EA on spermatogenesis. Cross-sectional reports of semen quality in trained men indicate that high-volume training is associated with impaired semen quality. Elite triathletes training for an Ironman competition (122.6 ± 62.7 min/session, 9.9 ± 1.8 sessions/wk) had lower sperm concentration, total sperm number, and percent normal forms compared with both recreationally (60 minutes per session, 3×/wk) and competitively active athletes (90 minutes per session, 5×/wk).[74] High-mileage runners (108.0 ± 4.5 km/wk) had decreased sperm motility, increased number of immature sperm, and decreased sperm penetration of bovine cervical mucus compared with moderate-distance runners (54.2 ± 3.7 km/wk) and controls, although all groups were within the normal range for T.[71]

Two RCTs, 1 short-term (2 weeks) study and 1 long-term (60 weeks) study, provide evidence that exercise training has an effect on semen quality. In the long-term RCT, 60 weeks of high-intensity exercise resulted in reduced sperm density, motility, and morphology; only sperm concentration and motility were affected in the moderate-intensity group.[69] In both groups, changes in semen parameters coincided with a decrease in T from the normal range to the subclinical "gray zone." In a 2-week trial in which 16 healthy men were randomized to either a nonexercising control group or an exercising group undergoing maximal cycle ergometer tests 4 d/wk, the exercising group had reduced ejaculate volume, sperm concentration, sperm number, and percent normal morphology compared with baseline and were significantly different from controls immediately postintervention but remained within the normal T range.[73]

A "volume-threshold effect" was proposed in the 1990s to explain how high exercise training volumes translate to effects on spermatogenesis and the reproductive axis. Based on the evidence that moderate-mileage running (40-60 km/wk) was not associated with alterations to T or sperm quality and the reproductive profiles of these runners were similar to that of the nonrunning sedentary control group, it was concluded that decreased T and abnormal semen profiles are likely related to an inability to meet energy needs during periods of high EEE, that is, periods of high-volume exercise training.[71,72]

What Are the Effects of Exercise or Energy Deficiency/Low Energy Availability on Libido?

Current evidence suggests that T and sexual function are often related.[84] In a cross-sectional study of long-distance runners who had low EA (27.2 ± 12.7 kcal/kg FFM/d) and reduced T concentrations, trained men reported higher Aging Male Symptoms (AMS) scores compared with nonactive controls.[61] The AMS scale is a clinical tool developed to assess health-related quality of life in men and is scored such that symptoms are scaled on a 1 to 5 point scale, with higher scores indicating greater severity.[85] An adapted version of the AMS, combined with questions from the Androgen Deficiency in the Aging Male questionnaire and the Sexual Desire Inventory, was used to develop a survey on exercise training habits and libido in 1077 recreationally and competitively active men >18 years old. Survey results indicate that training intensity and duration were highly associated with libido such that participants with lower training intensities and/or training durations were more likely to have a high/normal libido.[75] Therefore, it is suggested that endurance-trained male athletes may have decreased libido.[75]

Evidence Level C. Endurance-trained men may have decreased libido compared with their non–endurance-trained counterparts. Future research designed to test the independent effects of exercise and energy deficiency/low EA, as well as the relationship between hypothalamic

hypogonadism and changes in libido, is warranted to further understand the mechanisms associated with reduced libido in male athletes.

With Increased Energy Intake, What Are the Patterns of Recovery for Reproductive Hormones?

Reproductive perturbations in exercise-trained men (Army Rangers) and male athletes are mostly reversible after a period of refeeding or termination of the exercise stressor. Several examples are available including acute exercise studies,[38,67] the Army Ranger study,[40] and a large RCT.[69] After a 100-mile trail run, endurance-trained runners with suppressed T and LH concentrations showed evidence of recovery of both T and LH within 24 and 48 hours postrun to approximate prerace values.[67] No reference to the energy status of the runners during the run or during recovery was discussed. Energy status was assessed, however, after a 54-hour 1230-km ultra-endurance cycling event where the athletes were in an energy deficit that averaged 2468 kcal/24 hour.[38] Serum T concentrations, which were in the normal range in all but 1 athlete prerace, fell below the clinically low cutoff. Values remained below the clinically low cutoff (in this study reported at <8 nmol/L or 230 ng/dL) in 10 of 14 athletes even after 12 hours of recovery with ad libitum food intake. Recovery beyond 12 hours was not reported and presumably would have been associated with additional recovery if refeeding to restore energy balance was pursued, given the evidence below.

Two longitudinal investigations provide good evidence of recovery of hypogonadotropic hypogonadism. In the Army Ranger training study, where the soldiers were exposed to severe energy restriction concomitant with other stressors, the clinically low T concentrations in many of the soldiers were restored to prestressor baseline concentrations largely within 1 week of refeeding and reversal of the energy deficit.[40] Furthermore, in the soldiers who were refed extra calories midway through the 8-week program, reductions in T were prevented. After the 60-week RCT of treadmill running, serum LH, FSH, T, and free T significantly increased by the 12-, 24-, and 36-week recovery time points.[69] Notably, recovery of these parameters occurred faster in the moderate-volume training group compared with the high-volume training group.[69]

Recovery of the HPG axis in the aforementioned examples is likely due to an increase in central drive to the hypothalamus secondary to an increase in food intake. Refeeding male monkeys after 24 hours of fasting-induced suppression of LH pulse frequency and reduced T was associated with increased LH pulse frequency and T concentrations such that LH, FSH, and T progressively increased as the size of the refeed meal increased.[1,3] In young men, the suppression of LH pulsatile release and T levels noted after 48 hours of fasting was not observed in a fed state.[2] These data suggest that undernutrition presenting as an energy deficiency or low EA, even for short periods, can suppress functioning of the reproductive axis, perhaps in a dose–response manner.

Evidence Level A. Recovery of reproductive hormones in exercising men with reproductive hormone perturbations is evident on refeeding or removal of the exercise stressor.

What Are the Patterns of Spermatogenesis Recovery With Increased Energy Availability?

To date, few examples of recovery of spermatogenesis are available in the literature, but the examples available are well-executed studies and provide evidence for relatively prompt recovery. After the 60-week RCT of treadmill running, there was a significant and positive correlation observed between sperm concentration, motility, and morphology with duration of recovery time such that improved recovery of semen parameters was associated with increased duration of the recovery period.[69] After the 2-week trial in which 16 healthy men were randomized to either a nonexercising control group or an exercising group, ejaculate volume, sperm concentration, sperm number, and percent normal morphology were restored to pretraining values within 72 hours.[73]

Evidence Level B. Recovery of spermatogenesis occurs promptly on removal of the exercise stimulus.

BONE HEALTH IN MALE ATHLETES

What Is the Evidence for Poor Bone Health in Male Athletes Affected by the Triad?

Similar to female athletes,[86,87] impaired bone health and a higher risk of BSI have been reported among male athletes engaged in sports emphasizing leanness.[88,89] Low bone mineral density (BMD) has been reported in distance runners[90–94] and in athletes engaged in sports associated with low-impact loading patterns, such as cyclists,[95–98] jockeys,[99–101] and swimmers.[102,103] This correlates well with sports demonstrating higher rates of energy deficiency/low EA among male athletes. Specific risk factors for low BMD in male athletes have been identified and include energy deficiency/low EA, low body weight (<85% expected), hypogonadism, average weekly running mileage >30, and previous stress fracture.[91] Of note, evidence available for poor bone health in male athletes is largely limited to cross-sectional studies,[90–92,96,98–101,104–109] case series,[110] and a few prospective observational studies.[88,94,95,97,111,112]

Evidence Level B. Numerous prospective observational studies, cross-sectional studies, and case series have provided evidence for impaired bone health and higher risk of BSI among male athletes engaged in sports emphasizing leanness. Energy deficiency/low EA and low BMD have also been reported in distance runners and athletes engaged in sports associated with low-impact or nonimpact loading patterns.

Which Groups of Male Athletes Are Most Likely to Have Low Bone Mineral Density?

A large proportion of lean-sport athletes meet criteria for low BMD.[90–93,95,96,98,99,101,106,107] For example, 20 to 30 years old elite long-distance runners have lower spine and hip BMD compared with soccer players and lower spine BMD compared with nonathlete controls.[90] Similarly, male adolescent endurance runners have lower spine BMD Z-scores compared with athletes participating in ball or power sports.[91] Spine BMD Z-scores of ≤ -1.0 or ≤ -2.0 have been reported in 21% to 23.5% and 3.9% to 4.0%, respectively, of adolescent male runners compared with 5.6% and 0% of nonrunner athletes.[91,93] Tam et al[92] reported that 40% of 15 elite Kenyan runners versus none of the nonrunner controls had spine BMD Z-

scores of <-2.0. Even more concerning, Fredericson et al[90] reported spine T-scores between -1.0 and -2.5 in 40% of elite distance runners. In a case series of male athletes (mostly runners) with BSI, including stress reactions and cortical stress fractures, 43% had BMD Z-scores of <-1.0. Athletes with a history of trabecular-rich BSIs had a 4.6-fold increased risk for low BMD compared with those with cortical-rich BSIs.[106]

Data for low-impact or nonimpact sports (such as cycling and jockeying) are variable. Some,[95,96,107,108] but not all, studies[98] have reported lower femoral neck and/or spine BMD in cyclists compared with either recreationally active[107] or sedentary controls.[108] Penteado et al[98] reported Z-scores of ≤-2.0 in 32% of competitive cyclists, whereas Nichols et al[96] reported low BMD in 66% of master cyclists versus only 42% of nonathlete controls. Compared with controls, male jockeys have lower total body, lumbar spine, and femoral neck BMD,[99,100] with 29% of 79 male flat jockeys and 13% of 69 male jump jockeys having spine BMD Z-scores ≤-2.0.[101]

Evidence Level B. The prevalence of low BMD is greater in men participating in leanness or low-impact or nonimpact loading sports compared with men participating in nonleanness sports.

What Is the Impact of Training Load and Cross-Training on Bone at Specific Sites?

In runners, spine BMD is not associated with training load or biomechanical variables, such as running distance per week or knee/joint stiffness, suggesting that this specific site is susceptible to both metabolic and energetic factors,[92] such as hypogonadism and energy deficiency/low EA observed in Triad-affected male athletes. By contrast, femoral BMD is more strongly related to training load, suggesting an influence of loading patterns.[92] This is supported by the finding that elite distance runners had higher BMD than controls at the calcaneus, likely in response to loading patterns.[90] It is possible that high-impact loading mitigates or attenuates effects of energy restriction on bone health to some extent at certain sites. For example, high-magnitude loading, characteristic of resistance training, is associated with higher spine and hip BMD in competitive male cyclists.[113] In addition, male master cyclists who began resistance training or participating in impact exercise during a 7-year follow-up period lost significantly less spine and femoral neck BMD than those who did not engage in such activities.[97] Similarly, runners with a high training volume who also engaged in resistance training had greater total body, hip, and spine BMD than runners who did not engage in such training.[105]

Evidence Level B. In male athletes, hypogonadism and energy deficiency/low EA exert a strong influence on lumbar spine BMD, whereas training load exerts a strong influence on femoral BMD. High-impact loading may mitigate the effect of energy restriction on bone health at certain sites to a variable extent.

What Is the Relationship of Bone Health to Energy Status in Male Athletes?

Although most investigations of bone health in male athletes did not assess energy status, endurance and weight-class athletes, as previously discussed, are groups reported to have energy deficiency/low EA, supporting the link between energy status and bone health. In a group of 6 competitive male cyclists with BMD Z-scores of ≤-1.0, EA ranged from a mean of 19 to 22 kcal/kg FFM/d across a season.[28] In adolescent male runners, low BMI and the belief that being thinner improves performance were associated with lower BMD Z-scores,[93] demonstrating the link between energy deficiency/low EA and low BMD.

Limited longitudinal and interventional data exist regarding the effect of energy restriction on BMD in male athletes. BMD decreased significantly over a 9-month competitive cycling season at the total hip and femoral neck in a cohort of 14 amateur cyclists competing at the state and regional level, whose training volume averaged 13.5 h/wk, and remained below baseline values 3 months postseason.[95] By contrast, another longitudinal study found no change in BMD in cyclists across the summer racing season, attributed in part to the short duration of high-intensity training (or low EA) over the study period.[111] In a comparison among male power athletes ($n = 27$), endurance athletes ($n = 31$), and nonathlete controls ($n = 27$), there was a greater increase in lumbar spine BMD in power athletes compared with endurance athletes and controls over a 12-month observational period.[114] As such, effects on bone may differ with weight-bearing exercise, and because limited data are currently available, more research is warranted.

Evidence Level C. Observational studies in adolescent and young adult endurance and weight-class athletes provide most evidence linking energy deficiency/low EA and low BMD. More research is needed.

How Are Bone Strength, Geometry, and Structure Affected in Exercising Men With Low Bone Mineral Density?

Reports on bone strength, geometry, and structure in exercising men with low BMD and BSI are not available to date. Reports on bone geometry in oligo/amenorrheic female athletes and female patients with anorexia nervosa, both populations negatively affected by chronic energy deficiency, indicate that there is a decrease in trabecular volumetric density and number, an increase in trabecular spacing, and an increase in cortical thinning.[115] Despite weight-bearing exercise in oligo/amenorrheic athletes, changes in microarchitecture and bone strength result in an increased risk of stress fracture.[116] Moreover, oligo/amenorrheic athletes have a lower radial and tibial fracture failure load when compared with nonathletes, supporting the notion that they are at an increased risk of fractures.[117] Studies assessing microarchitecture and strength estimates in at-risk male athletes are essential to further understand the effects of energy deficiency/low EA on bone health in male athletes. Notably, in male athletes deemed healthy, high-resolution peripheral quantitative computed tomography has indicated that participation in high-impact, bone-loading sports such as alpine skiing and soccer (compared with nonimpact swimmers) is protective and improves bone geometry in trabecular bone and yields a higher failure load.[104] Interestingly, in a 2-dimensional imaging study, male athletes with a history of BSI at trabecular-rich sites, such as the pelvis, femoral neck, and calcaneus, had a 4.6-fold increased risk for low BMD compared with male athletes with BSI at cortical-rich sites, such as the tibia, fibula, femur, metatarsal, and tarsal navicular bones.[106] In addition, in a 5-year prospective study of male and female collegiate runners, runners with BSIs of predominantly trabecular skeletal sites had a significantly prolonged time to return to sport compared with runners with BSI at cortical sites.[94]

Evidence Level C. Male athletes with a history of BSI at trabecular-rich sites have demonstrated an increased risk for low BMD compared with male athletes with BSI at cortical-rich sites. More research is needed assessing how bone geometry, structure, and strength are affected in the high-risk male athlete with low BMD.

What Is the Risk for Bone Stress Injury and Stress Fractures in Male Athletes?

Investigations of BSI and stress fractures in men have largely focused on male runners[88,89,94] and men during military training.[109,112] In a prospective study, as many as 27% of male runners sustained at least 1 BSI over 1.9 years.[88] Bone stress injury may be predicted by a version of the Female Athlete Triad Cumulative Risk Assessment score that was modified for male athletes. This risk assessment included a score for low EA, low BMI, previous BSI, and low BMD values.[88] Consistent with this, an increased rate of stress fractures was associated with a history of a previous fracture and repetitive participation in competitive running seasons in adolescent male runners.[89] Importantly, in adolescent male athletes,[91] the cumulative effect of an increasing number of risk factors increased the prognosis for poor bone health, similar to female athletes.[86,87] This highlights the importance of identifying Triad risk factors and underscores the importance of screening for risk factors to avoid the sequelae of poor bone outcomes.[118]

Studies from the military are also informative, and in male Royal Marine military recruits, those with stress fractures had a lower spine, hip, and total body BMD compared with recruits without stress fractures.[109] Interestingly, pQCT measures of bone geometry and estimated strength were lower in recruits with versus those without stress fractures.[109] Among male military cadets in the United States, a risk of stress fracture was higher in cadets with smaller tibial cortical area, lower tibial BMC, and smaller femoral neck diameter.[112] These data demonstrate BMD and geometry characteristics that may predispose males to stress fractures during basic military training.

Evidence Level B. A previous history of BSI is associated with an increased rate of BSI in collegiate and adolescent male runners. There is a cumulative effect of an increased number of Male Athlete Triad risk factors on prognosis for bone health in the male athlete.

Is There Evidence for Reversibility of Low Bone Mineral Density or Improved Bone Geometry After a Period of Refeeding?

To date, there is no evidence available for the reversibility of low BMD or improved bone geometry in response to refeeding in male athletes or exercising men.

What Is the Relationship of Energy Deficiency/Low Energy Availability to Bone Marker Metabolism in Men?

Bone turnover markers are influenced by periods of energy restriction that result in energy deficiency/low EA in women and serve as proxy indicators of bone health.[13,119] There are few reports available to date regarding bone turnover markers in male athletes. An abrupt short-term 3-day exposure to energy restriction of approximately 50% of estimated energetic needs, while running for 60 minutes on a treadmill, was compared with 3 days of the same 60 minutes of treadmill running in an energy balanced condition.[119] Energy restriction during training resulted in a 15% decrease in a bone formation marker (N-terminal pro-peptide of procollagen [P1NP]) associated with a 17% decrease in IGF-1, whereas no changes were observed in bone resorption markers (urinary N-terminal telopeptide and deoxypyridinoline).[119] This suggests that in an energy deficient environment, bone formation is suppressed as is the anabolic stimulus for bone growth. By contrast, another experiment that involved 5 days of restricted (15 kcal/kg FFM/d) versus replete (45 kcal/kg FFM/d) EA, achieved by both energy restriction and treadmill running, resulted in no significant effect on markers of bone formation or resorption in men.[13] This is in contrast to a study in women that demonstrated a 13% reduction in bone formation (P1NP) and a 19% increase in bone resorption (sCTx) after energy restriction.[13] As such, more research is necessary to determine the specific volume of energy deficits or low EA that renders an effect on bone marker metabolism in men.

Evidence Level C. There are limited studies assessing the effect of energy deficiency/low EA on markers of bone turnover in the male athlete, and results are mixed. More research is needed.

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS REGARDING ENERGY AVAILABILITY, REPRODUCTIVE FUNCTION, AND BONE HEALTH

In summary, observational studies, field-based studies, and a limited number of short-term laboratory experiments support that (1) there is evidence of energy deficiency/low EA in subsets of male athletes, particularly those who participate in leanness sports, (2) the magnitude and duration of energy deficiency/low EA required for metabolic hormone alterations to be observed in males is likely more severe than that required in females, (3) a specific threshold value of "low" EA, below which metabolic alterations are induced, is not currently supported or identified, and (4) the pathways to low EA can vary as in women, however, the psychological underpinnings of disordered eating and associated eating behaviors may vary between sexes. Much additional work is still necessary, including head-to-head comparisons of male and female physiological responses to energy deficiency/low EA and comparisons of the psychological and behavioral drivers of energy deficiency/low EA.

A summary of the findings with respect to reproductive function includes findings from a review of acute studies of exercise in male athletes, cross-sectional reports of male athletes, and longitudinal and RCT evidence that demonstrate (1) exercise-associated suppression of reproductive hormone concentrations, impaired semen quality, and decreased libido, that is (2) likely related to an energy deficient metabolic environment, and which (3) recovers promptly after removal of the exercise stressor. Strong evidence supports that hypogonadotropic hypogonadism is a major component of the Male Athlete Triad, although additional work is needed to identify the independent effects of energy deficiency (as opposed to high training volume) on reproductive function in exercising men.

With respect to bone, cross-sectional, case-series, and a few prospective studies suggest that male athletes engaged in sports emphasizing leanness have (1) impaired bone health

and (2) a higher risk of BSI. Much additional work is necessary to identify the effects of energy deficiency on bone turnover and bone microarchitecture. Evidence statements from "The Male Athlete Triad: A Consensus Statement from the Female and Male Athlete Triad Coalition, Part I: Definition and Scientific Basis" are listed in Table 4.

CALLS FOR RESEARCH AND GAPS IN KNOWLEDGE

1. Experimental evidence testing the direct effects of energy deficiency/low EA on reproductive and bone outcomes and on libido.
2. Validation of easy-to-use techniques to assess objective measures of metabolic hormones and/or metabolic rate for the purposes of determining whether an individual can be deemed "energy deficient" in the absence of observable changes in body weight.
3. Research on the psychological and behavioral underpinnings of disordered eating in male athletes.
4. Research on the magnitude of energy deficiency/low EA that is causally related to physiological outcomes and whether any threshold exists below which effects are induced.
5. Research on the underlying mechanisms and timeframe over which restored energetic status leads to recovery of reproductive and bone outcomes.
6. Research on whether the underlying mechanisms for induction of Triad effects differ from those of recovery.
7. Research on physiologic perturbations associated with low EA and effects on performance.
8. Research on the long-term effects of the Male Athlete Triad on health and performance.

Part 2 of the Female and Male Athlete Triad Consensus Statement, "The Male Athlete Triad: Diagnosis, Treatment, and Return-to-Play," also in this issue, includes best practice strategies for screening, diagnosing, and managing the Male Athlete Triad conditions. It is through this translation research that we can develop a best practice model for management and

TABLE 4. Summary of Evidence Statements

Evidence Statements	Evidence Level
Severe energy deficit (~1100 kcal/d) in men is associated with alterations in metabolic hormones indicative of metabolic compensation. Data are not consistent for alterations in these hormones in states of low EA (defined as <15 kcal/kg FFM/d).	B
Exercising men, particularly those in leanness sports, are at risk for developing disordered eating/eating disorders; however, the specific eating behaviors may differ from those observed in exercising women.	C
High-volume exercise training and bouts of acute prolonged exercise in men can be associated with reductions in T concentration, often falling into the subclinical "gray zone" and sometimes into the clinically low zone.	B
Endurance-trained men may have decreased libido compared with their non–endurance-trained counterparts. Future research designed to test the independent effects of exercise and energy deficiency/low EA, as well as the relationship between hypothalamic hypogonadism and changes in libido, is warranted to further understand the mechanisms associated with reduced libido in male athletes.	C
Recovery of reproductive hormones in exercising men with reproductive hormone perturbations is evident on refeeding or removal of the exercise stressor.	A
Recovery of spermatogenesis occurs promptly on removal of the exercise stimulus.	B
Numerous prospective observational studies, cross-sectional studies, and case series have provided evidence for impaired bone health and higher risk of bone stress injury among male athletes engaged in sports emphasizing leanness. Energy deficiency/low EA and low BMD have also been reported in distance runners and athletes engaged in sports associated with low-impact or nonimpact loading patterns.	B
The prevalence of low BMD is greater in men participating in leanness or low-impact or nonimpact loading sports compared with men participating in nonleanness sports.	B
In male athletes, hypogonadism and energy deficiency/low EA exert a strong influence on lumbar spine BMD, whereas training load exerts a strong influence on femoral BMD. High-impact loading may mitigate the effect of energy restriction on bone health at certain sites to a variable extent.	B
Observational studies in adolescent and young adult endurance and weight-class athletes provide most evidence linking energy deficiency/low EA and low BMD. More research is needed.	C
Male athletes with a history of bone stress injuries at trabecular-rich sites have demonstrated an increased risk for low BMD compared with male athletes with bone stress injuries at cortical-rich sites. More research is needed assessing how bone geometry, structure, and strength are affected in the high-risk male athlete with low BMD.	C
A previous history of BSI is associated with an increased rate of BSI in collegiate and adolescent male runners. There is a cumulative effect of an increased number of Male Athlete Triad risk factors on prognosis for bone health in the male athlete.	B
There are limited studies assessing the effect of energy deficiency/low EA on markers of bone turnover in the male athlete, and results are mixed. More research is needed.	C

return-to-play for male athletes with one or more components of the Male Athlete Triad and improve health outcomes.

Male Athlete Triad Roundtable in conjunction with the 64th Annual ACSM Meeting 2017

Co-Chairs: Michael Fredericson, MD, Aurelia Nattiv, MD

Invited Expert Panelists that Reviewed Evidence Statements: Michelle T. Barrack, PhD, RD, Graeme Close, PhD, Mary Jane De Souza, PhD, Elizabeth Joy, MD, MPH, Karsten Koehler, PhD, Madhusmita Misra, MD, MPH, Aimee Shu, MD, Adam Tenforde, MD, Nancy Williams, ScD

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