HHS Public Access

Author manuscript

*Clin Sports Med*. Author manuscript; available in PMC 2018 October 01.

Published in final edited form as:

*Clin Sports Med*. 2017 October ; 36(4): 671–686. doi:10.1016/j.csm.2017.05.003.

# Female Athlete Triad: Future Directions for Energy Availability and Eating Disorder Research and Practice

**Nancy I. Williams, Sc.D., FACSM**,

Department of Kinesiology, Women's Health and Exercise Laboratories, Room 108 Noll Laboratory, Penn State University, University Park, PA 16802, Phone: 814-865-1346, Fax: 814-865-4602

**Siobhan M. Statuta, MD**, and

University of Virginia Health System, Department of Family Medicine, P.O. Box 800729, Charlottesville, VA 22908

**Ashely Austin, MD**

University of Virginia Health System, Department of Family Medicine, P.O. Box 800729, Charlottesville, VA 22908, Phone: 434-924-5348, Fax: 434-243-1473

## Synopsis

Despite over three decades of research on the Female Athlete Triad, research gaps remain. Although low energy availability (EA) is the key etiological factor in the Triad and the pathways to low EA are varied, its effects can be modified by several factors. As such, a more individualized approach to identifying and treating low EA is warranted. Accurate screening, diagnosis, and treatment of disordered eating (DE) remains a challenge, however, recent techniques combined with novel educational and behavior interventions prove promising. Recently published practice based guidelines have helped to translate Triad science and should improve as they are refined. Our goal in this paper is to identify the current state of research and distinguish areas that require further investigation.

### Keywords

Low energy availability; eating disorders; female; athlete; exercise; female athlete triad

## Introduction

In the 1990's, the concept of the Female Athlete Triad was introduced, drawing attention to a syndrome of three tightly interrelated conditions: disordered eating (DE), amenorrhea, and

Corresponding author: Nancy I. Williams, Sc.D., Department of Kinesiology, Women's Health and Exercise Laboratories, Room 108 Noll Laboratory, Penn State University, University Park, PA 16802, niw1@psu.edu, Phone: 814-865-1346, Fax: 814-865-4602.

Disclosure Statements

NI Williams has nothing to disclose

osteoporosis [1]. The definition of the Triad was revised in 2007 to its current meaning to include one or more of the following three components:

1. Low energy availability (EA)(with or without DE)
2. Menstrual dysfunction
3. Low bone mineral density (BMD)

This also includes the continuum between healthy and unhealthy states for each of the three elements [2]. Several seminal studies in the 1980's [3–5] piqued the interest of clinicians and researchers alike, and almost four decades of research now serves as the foundation for our understanding of this complex medical condition. The existence of the Triad is widespread, with prevalence varying by sport. Sports that demand high energy expenditure, a lean physique, and/or an aesthetic component carry the greatest incidence [6]. The clinical, behavioral, and physiological consequences of the Triad are extensive and include clinical eating disorders and DE, osteopenia, transient infertility, dyslipidemia, impaired endothelial function [7–9], performance-related issues such as stress fractures [10–12], fatigue, and decrements in competitive performance [13]. Much progress has been made in our understanding of the underlying behaviors and physiology of these conditions [14–16] as well as the creation of practical recommendations for prevention, screening, treatment, and return to play [2 17 18]. However, many gaps still exist in the literature as well as in the translation of research into practice. The purpose of the article is to highlight future potential directions for research by drawing attention to areas in the Triad literature that require clarification. From there, this data may be applied to the clinical setting for more evidence-supported interventions. For recent reviews on the Female Athlete Triad, the reader is referred to other sources [19–24].

## Low Energy Availability- Gaps and Clarifications

EA has been more of a research focus since the 2007 American College of Sports Medicine Position Stand [2] on the Female Athlete Triad emphasized the critical role of EA(with or without DE) in the etiology of the Triad. Current knowledge on the underlying mechanism of exercise-related menstrual disorders has been informed by prospective studies in nonhuman primates [25] and previously untrained women [26 27]. These have shown that aerobic exercise, in combination with caloric restriction, can induce menstrual disturbances. Menstrual function is restored when energy intake (EI) (and, in turn, energy availability (EA)) is increased during periods of exercise. This demonstrates a causal role of low EA in the induction, as well as the vital role it plays in the reversal of exercise associated menstrual disturbances [25 28]. EA also plays an important role in maintaining skeletal health in exercising women. This is evidenced by its association with altered bone parameters independent of estrogen status [29 30], and by the dysregulation of important bone related hormones when Triad conditions are present [31–34].

While the causal role of low EA in the development of Triad conditions is well supported, several issues deserve consideration. The elegant studies of Loucks et al. are frequently cited to support a particular calculation of EA that represents the difference between the total

calories consumed as food and the caloric expenditure of exercise, normalized for fat free mass (ffm). [35]

$$\frac{(daily\ dietary\ intake\ (kcals) - daily\ exercise\ energy\ expenditure\ (kcals))}{ffm\ (kg)}$$

Short-term (5 day) reductions in EA below a threshold of 30 kcals/kg ffm per day have been found to slow the normal pulsatile release of luteinizing hormone (LH) from the anterior pituitary gland- a proxy indicator of hypothalamic gonadotropic-releasing hormone (GnRH) secretion [36]. A slowing of LH pulse frequency is, in turn, associated with delays in folliculogenesis, luteal phase shortening, and more severe menstrual disturbances [25 37–39]. This reduced LH pulse frequency occurs regardless of whether EA is reduced via diet, exercise, or a combination of the two [35]. When EA is considered to be the energy required to support a body's basic physiological processes, it becomes clear that effects will be extensive as levels decline. Specifically, once EA is reduced below the 30 kcals/kg ffm per day threshold mentioned above, some of the observed metabolic alterations include reduced serum concentrations of glucose, triiodothyronine, insulin, insulin like growth factor-1, and elevations in growth hormone and cortisol [35 36].

The previous studies demonstrate the importance of EA in the modulation of LH pulse frequency with reductions associated with subclinical menstrual disturbances [40 41] and amenorrhea [42 43]. Yet, much more needs to be clarified to further our understanding. For example, the precise magnitude of reduction in EA, or rather, the degree of energy deficit associated with the initial disruption of ovarian function has not been directly demonstrated through experimentation. Although we know an EA below the threshold of 30 kcals/kg ffm per day modifies LH pulsatility and metabolism, we do not know what magnitude of change in LH pulsatile dynamics is associated with the induction of menstrual disturbances as this has not been prospectively evaluated in humans. In a recent randomized trial, Williams et al. demonstrated that luteal phase defects, oligomenorrhea, and anovulation were induced by energy deficits ranging from −22% to −42% of baseline energy needs (−470 kcals to −810 kcals below initial energy requirements) [27]. These outcomes provide practical information about the magnitude of caloric deficiency resulting in exercise-associated menstrual disturbances. However, the assessment of actual energy balance is difficult and expensive to calculate. Furthermore, it varies significantly as it adjusts in attempts to conserve energy, restore energy balance, and stabilize weight [44]. As such, future studies should address the magnitude of change in EA as calculated by Loucks et al. associated with the induction of menstrual disturbances. To that end, a preliminary report by Lieberman et al. [45 46] demonstrates that EA is linearly related to the risk of menstrual disturbances and that there exists no clear threshold below which ovarian function is disrupted. Rather, as EA drops below 30 kcal·ffm$^{-1}$, the risk of a menstrual disturbance increases above 50% [45]. Regardless of the level of EA at which reproductive function is disrupted, current recommendations [47] to maintain EA at approximately 45 kcal·ffm$^{-1}$ are supported. It is clear, however, that a need exists for additional research about energy thresholds as well as identify easier, less expensive methods to calculate energy deficits.

One future research area to prioritize includes the validation of repeated assessments using field measures of EA that accurately reflect the EA calculations of Loucks, et al. [47]. These calculations could additionally be used to explore new methods of monitoring energy status such as repeated measurements of BMI, weight loss, and percent body fat. Traditionally, these data have been difficult to interpret given the variability of the Triad athlete presentations. For example, a single measurement of body weight and/or BMI may reveal overt undernutrition and chronic energy deficiency if values are < 85% of expected body weight, BMI < 17.5 $kg \cdot m^2$, or if an adolescent's BMI is < $50^{th}$ percentile [44]. However, if these measures do not reveal an energy deficit, additional, more reliable measures are required to determine low EA. The ideal biomarker of energy status would be one that can be accurately and objectively measured to scale and is reflective of compensatory adaptations to chronic energy deficiency (i.e., body weight can remain stable even when EA is low). Furthermore, to enhance its applicability, the biomarker would need to be reflective of changes in EA over the same time frame that is associated with changes in ovarian function. One such biomarker, triiodothyronine, may fit this criteria. Because body weight stability can be observed despite a low EA state [48], physiological signs of energy conservation should be assessed such as blood concentrations of total triiodothyronine and measures of the ratio of actual to predicted resting metabolic rate [17]. Future studies would ideally determine the reliability and validity of any EA biomarker.

As noted above, the current lack of reproducibility in the methods of assessing energy deficiency has limited the widespread implementation of EA assessments. Another potential approach to diagnosing low EA is to target the more qualitative assessment of eating behaviors and attitudes to identify the factors related to the under consumption of energy relative to energy expenditure. This may be an alternative approach to quantifying energy intake and or energy expenditure in athletes who are under-consuming food due to conscious restriction. In support of this, studies have documented significant associations between drive for thinness, cognitive restraint, and EA [49 50]. Regardless of the approach, it is important that any measurements demonstrate acceptable levels of sensitivity and specificity if particular cut-offs for indicators of EA are used in decision making rules for individual athletes. A recent review by Joy and Nattiv [51] provided a foundation of information regarding the clinical assessment and management of eating disorders (ED) and DE in athletes upon which future studies can expand.

## Etiology of Low Energy Availability

Appropriate treatment of low EA as it relates to the induction of menstrual and bone sequelae requires an understanding of how and why EA is low. What is the pathway to low EA? As described in the Female Athlete Triad Coalition Consensus Statement [44], there are four distinct pathways to low EA:

- disordered eating
- clinical eating disorder
- intentional weight loss without disordered eating
- inadvertent undereating

As such, screening and treatment strategies need to target these individual pathways. If the etiology of low EA involves disordered eating (DE), medical attention and nutrition education are warranted. A clinical eating disorder (ED) should trigger medical, psychological, and nutritional education interventions, each with monitored components. Similarly, weight loss without DE should also involve nutritional education. A less well understood basis for low EA is inadvertent undereating, which presumably occurs when caloric intake does not meet energy expenditure needs in the absence of conscious restriction of food intake. The extent to which inadvertent undereating contributes to the Triad is currently unclear. Possible explanations for inadvertent undereating could include practical and logistic challenges such as access to and or affordability of food and beverages. Unfortunately, the prevalence of these issues has not been well documented. The physiological suppression of hunger in response to the intensity or volume of exercise has been demonstrated in prospective studies and therefore appetite is not considered a reliable indicator of energy requirements in endurance sports [48 52 53]. There exist many questions regarding inadvertent undereating as there is not much data currently available. In fact, the majority of studies of female athletes with Triad conditions provide evidence of DE in the form of body image disturbances, measures of restrictive food intake, or pathogenic weight control behaviors in association with menstrual disturbances and low bone mass [14 54 55]. A recent report on the prevalence of individual and combined Triad conditions from over 65 studies found that the prevalence of clinical ED and DE ranged from 0 – 48% and 7.1 – 89.2%, respectively. Future studies need to document the extent to which, and the mechanisms whereby, inadvertent undereating contributes to low EA associated with the Triad.

## Gynecological Age

The importance of low EA as a causal factor in Triad conditions has been established. Yet, the individual variation in the susceptibility to low EA may be attributable to factors that modify the relation between EA, ovarian disruption, and/or bone metabolism. A critical factor that has not been addressed in Triad literature is gynecological age, i.e., the difference between one's chronological age and the age of menarche. The natural prevalence of menstrual disturbances decreases with advancing age until the time of perimenopause [56]. In a variety of species, the impact of various stressors on the reproductive axis also decreases as reproductive opportunity decreases [57]. Evidence for the effects of gynecological age were reported by Loucks et al. who showed that decreases in LH pulsatility caused by low EA (EA < 10 kcal/kg FFM), were dependent on gynecological age. They noted that subjects whose gynecological age ranged from 14–18 years did not experience a decrease in LH pulse frequency whereas those with a gynecological age of 5–8 years did [58]. Gynecological maturity was also cited as a factor in the prospective 12 month marathon training study by Rogol et al. who reported that no significant changes occurred in any LH pulse parameter in women that were 17.8 ± 0.9 years post menarche [59]. However, this study was criticized because the exercising women began the intervention with some indications of exercise-induced menstrual disturbances at the outset which may have prevented the ability to see the effects of the yearlong training [60]. In a prospective study comprised of women aged 25–40 years who participated in an exercise training program

combined with caloric restriction to achieve modest weight loss, few disruptions in menstrual regularity occurred [61]. Taken together, these studies indicate that the risk of developing menstrual disturbances in association with exercise may decline with advanced gynecological age. This is an important translational finding as practitioners should take gynecological age into account when assessing the risk of exercise-associated menstrual disturbances and determining the need for female athlete triad prevention strategies in gynecologically mature athletes.

## Genetics

An individual's genetics may contribute to one's susceptibility to functional hypothalamic amenorrhea (FHA). Caronia et al.[62] reported that in a sample of 55 women with FHA, seven had heterozygous mutations associated with hypothalamic hypogonadism, where mutations to the following genes were found: fibroblast growth factor receptor-1, the Kallmann syndrome 1 sequence, prokineticin receptor 2, and the GnRH receptor. No such mutations were found in 422 control subjects with normal menstrual cycles. Each of the affected genes serve unique and significant roles. The Kallmann syndrome 1 sequence gene and the prokineticin receptor 2 gene both play a key role in the migration of GnRH-secreting neurons [63 64]. The fibroblast growth factor receptor-1 gene determines differentiation, migration, and maintenance of GnRH secreting neurons [65]. Lastly, the GnRH receptor gene encodes the receptor that GnRH binds to on the gonadotrophs [66]. Consequently, individuals with FHA may possess defects important to GnRH secretion and regulation therefore making their hypothalamic pituitary ovarian function vulnerable to stress-induced dysfunction (i.e. low EA). More research is necessary to determine the extent to which genetic factors may contribute to menstrual cycle disturbances in exercising women of all ages.

## Psychological Factors

Although much of the Triad condition relates to states of low EA, it is well documented that psychological and social stress can impact reproductive function in humans and animals [67–75]. Despite this fact, specific Triad literature lacks the recognition that exercise-associated menstrual disturbances are a subtype of this stress-induced reproductive disruption paralleling anorexia, DE, bulimia, and other psychosocial stressors. It is likely that these exercise-induced menstrual disturbances involve elements of psychosocial stress, as metabolic and psychosocial stressors co-exist in everyday life and are difficult to tease apart. Even though clinical eating disorders such as anorexia and bulimia are considered stress-related disorders, the singular focus on the energy availability aspects of these psychiatric disorders as the primary mechanism underlying reproductive dysfunction ignores the potential contribution of additional suppressive effects of neuroendocrine pathways associated with psychogenic factors against a background of energy deficiency. In fact, synergistic effects of a combination of metabolic and psychosocial stressors on the disruption of menstrual function have been demonstrated in a monkey model [76]. Bethea et al. have extended these findings to show that individual differences in stress sensitivity to the aforementioned multi-stress paradigm are associated with alterations in central neurotransmitter systems [77 78].

Similar to these animal experiments, additional studies in humans by Berga et al. have demonstrated that women with FHA have psychological phenotypes suggestive of high stress responsiveness [79 80]. These women commonly display dysfunctional attitudes, difficulty coping with daily hassles, a higher dependence on interpersonal relationships, higher incidence of past psychiatric disorders, and subclinical symptoms of depression and anxiety. A randomized controlled trial utilizing cognitive behavioral therapy to treat the aforementioned abnormal psychological profiles in women with FHA demonstrated that women who received the therapy had an ovarian recovery rate of 87.5% versus a 25.0% recovery rate in the control group who received no treatment [81]. A follow up study detailed the neuroendocrine changes that accompanied the recovery of ovarian function [82]. To highlight the importance of psychosocial change in the recovery of ovarian function, it is important to note that Berga's work represented exercising women with menstrual disturbances who continued to exercise as a part of therapy in addition to employing behavioral change techniques to make healthy adjustments to dietary intake.

The mechanism underlying the effects of psychosocial stressors on menstrual function is commonly thought to be the stress-induced activation of the HPA axis [83], but the actual neuroendocrine mechanisms that suppress GnRH neuronal activity remain unclear [84–86]. A challenging aspect in identifying these mechanisms is that psychogenic stressors are often associated with metabolic stress because food intake is reduced. In many stress studies, this is overlooked, as food intake and body weight changes are often not quantified or reported. Loucks and Redman [87] have explored this conundrum concluding that the underlying mechanism whereby psychogenic stressors act to suppress the reproductive axis is through their impact on energy balance. The challenge of teasing out psychogenic versus metabolic factors associated with menstrual disturbances in exercising women has also been addressed [88 89]. Future research should comprehensively examine the role of psychosocial factors in the development and reversal of Triad conditions.

Given the complexity of how the key role of low EA in the etiology of menstrual disturbances may be modified by factors such as gynecological age, genetics, and psychogenic stress, it is important that future research explores the relative importance of these effects. Other fertile areas for research include the impact of racial and cultural differences on an individual's susceptibility to stress-induced reproductive disturbances. Although progress has occurred regarding the application of research findings to the development of recommendations for athletes and sports medicine practitioners, the move toward more "precise" and "personalized" medicine should foster evidence-based approaches to Triad prevention and treatments that incorporate a more comprehensive understanding of physiological and psychosocial influences on menstrual function and/or bone metabolism.

## Female Athlete Triad and Relative Energy Deficiency in Sport (RED-S)

As Triad research evolves, it is important to keep in focus that the primary physiological and clinical presentations of the Triad continue to be low EA with or without DE, menstrual disturbances, and low bone mass. These are the medical conditions that clinicians and practitioners have deemed clinically important enough to warrant treatment and prevention

strategies. Secondary physiological and clinical consequences of the Triad have also been documented in exercising women including alterations in metabolism [90–93], lipid profiles [8 94], cardiovascular function [94–96], and bone stress injury [97 98]. These changes are mechanistically linked to the Triad related to the chronic hypoestrogenic state and long-term low energy availability [97]. Recently, the International Olympic Committee (IOC) described their version of this concept of low EA in the acronym “RED-S” or “relative energy deficiency in sport”. They emphasize a broader impact that low EA may have on additional physiological systems outside of the hypothalamic pituitary gonadal axis and bone [99]. The juxtaposition of this recent IOC statement with existing Triad literature has generated debate and confusion [18 100 101]. The authors propose that “relative energy deficiency” (which is not quantitatively defined) is a common problem for both females and males in sport, and that its effects are widespread across a variety of organ systems [99]. The RED-S concept broadly considers all bodily processes that may be affected by relative energy deficiency equally and depicts the relation between low EA and each aspect of physiology as a direct association. In contrast, previous research and position stands on the Triad have repeatedly documented specific physiological effects associated with low energy availability [2 97] (i.e. alterations in metabolic hormones) and those associated with clinical eating disorders (i.e. gastrointestinal disturbances (GI)). These outcomes represent part of the primary underlying mechanism of energy conservation (as in the case of hormonal changes) as well as the secondary disturbances (the GI disturbances) in relation to the principal components of the female athlete triad. Notably, the prevalence, severity, and clinical importance of secondary effects of chronic low EA, such as those seen in immune and vascular function, have not yet been irrefutably established. In the meantime, clinicians and practitioners should not lose sight of the established clinical importance and treatment recommendations associated with the Female Athlete Triad as more data regarding other body systems are explored. Future research should include direct comparisons of the validity of approaches used for risk stratification, treatment, and return to play in recent consensus statements [44 99]. More research on the effects of low EA in males, individuals of different abilities, and individuals from different racial backgrounds is highlighted by Mountjoy et al. [99].

## Male Athlete Triad

Interest in whether a parallel to the female athlete triad occurs in male athletes has increased recently [102]. Clinically, this is a challenge to ascertain, as outward reproductive manifestations are difficult to identify and may require sperm and fertility testing as well as tracking of hormone levels. Testosterone levels are, indeed, affected by physical activity with levels shifting in response to time/duration of exercise, endurance versus resistance-trained sport, and age [103]. Testosterone production can also decrease in overtraining or conditions of decreased EA [104]. Manifestations on bone health are less clear as available studies have been in small samples across varying ages and sports. For endurance athletes, levels of sex hormones tend to be reduced [105], and values outside of the normal range have been associated with impaired bone health [102]. However, Ackerman et al. [106] showed that levels of estradiol were positively correlated with higher BMD, raising questions regarding the possible effects of estradiol on bone health in males beyond that of

the classically considered testosterone. Currently, parallels between male and female athletes in low energy conditions are being further examined. There clearly exists a dearth of information, requiring further research in areas such as nutritional deficits in male athletes and vitamin supplementation in regards to bone health. Applying conclusions found in the female population to males is not substantiated and caution should be encouraged. Future research should highlight the magnitude of change in EA that is associated with clinical and physiological sequelae in both reproductive and bone health outcomes in male athletes, especially considering that it may prove to be different than what has been found in female athletes.

## Eating Disorders and Disordered Eating

Most cases of the Female Athlete Triad involve low EA that results from the conscious restriction of food intake that occurs along a continuum of severity. As Joy et al. noted in a recent extensive review [51], the concern surrounding eating behaviors in athletes is pressing, as the rates of EDs in the general population is on the rise for individuals in their late teenage 19 years [107] and are high among elite adolescent athletes [108]. Added attention to certain research gaps would assist with demystifying this entity and would allow for earlier identification of potential problematic athlete cases, as this is thought to be crucial for the recovery process [109]. There exists no simple method for detection of EDs and although questionnaires exist, studies indicate that a clinical interview is the best option among elite athletes and controls [108]. More recent screening approaches that complement established ED inventories [110 111] or interview strategies [112] includes the Brief ED in Athletes Questionnaire (BEDA-Q) [113]. An alternative is the LEAF-Q (Low Energy Availability in Females Questionnaire) which has been shown to predict overall Triad risk independent of whether DE is present [114]. Future studies should continue to focus on fast and accurate ways to screen and diagnose DE/ED in female athletes with attention towards the effectiveness of these approaches on an individual basis. Regarding the specificity of methodological approaches, Bratland-Sanda's review effectively points out that previously-proposed risk factors need to be scientifically validated by showing a clear, causal relationship through more prospective, largescale, and longer-term studies [115]. There is also exists a need for an extensive literature review of general, as well as more specific risk factors across sport and gender including weight cycling and dieting pressures, personality traits, early sport-specialization, history of injury, and sport regulations (especially in those emphasizing leanness) [115]. Suggested risk factors need to be scientifically validated by showing a cause-and-effect relationship. By having a more sport specific checklist of confirmed items to watch for, a more timely recognition and management process can be initiated [115]. Besides refining the checklist of risk factors and improving diagnosis, intervening through education shows potential value in the prevention of eating pathologies. Recent advancements regarding educational interventions targeting adolescent athletes and coaches to prevent the development of ED [116 117] should be a focus going forward.

Another area of potential investigative focus is the clarification of when an athlete identified to be on the ED-DE spectrum is too ill for sport participation. How soon will the recovering athlete be well enough to resume sport, and to what degree? These are clinical conundrums encountered regularly without any clear or applicable consensus. The current consensus

statements are beneficial but do not address specifics as basic as warranted exercise limitations upon identification of an ED. Delineation of more explicit sport-specific guidelines would offer invaluable guidance to those healthcare providers overseeing athlete recovery and safe guidance back into sport. Similarly, completion of a simple yet thorough review of current clinical practices by these care providers would provide applicable and real-life data to help identify the direction of future position statements/guidelines. A review would also help in refining previously published strategies for Triad risk stratification [44]. With actual data illustrating the real-world clinical successes and hurdles, providers and care teams can focus on more efficient and practical approaches to the athlete.

## Applying Triad Science and Clinical Judgment to Inform Clearance and Return to Play Decisions

Although the scientific underpinnings and epidemiology of the Female Athlete Triad have been well explored in the literature and updated position stands are available, a gap in practice based applications of Triad science still exists. This is arguably the most difficult step in addressing public health issues, and as such, represents a gap in the area of the Female Athlete Triad. The recent Female Athlete Triad Coalition Consensus Statement [44] provided the first comprehensive effort to provide clinicians and practitioners with recommendations for clearing athletes for competition and returning them to play. As a joint effort among leading scientists, physicians, nutritionists, and other sports medicine experts, the statement advances the field of Triad research because it provides an evidence based approach to risk stratification including an easy to use algorithm for incorporating Triad related risk factors into decision making processes for clearance and return to play of individual athletes. Another approach i.e., “Red Light, Yellow Light, Green Light” has been developed by Sundgot-Borgen et al. and is described in Mountjoy et al. [99]. These guidelines are based on scientific evidence and must be used in the context of clinical judgment while considering “decision modifiers” such as the type of sport, the timing during the season, the position played, etc. These approaches represent the translation of Triad science into practice at an organizational and policy level, with recent reports confirming that these recommendations are being implemented and adapted [118 119]. Future research should include refinements and modifications to these algorithms that improve their sensitivity and specificity.

## Summary and Conclusions

Research on the Female Athlete Triad has spanned several decades. Despite this, there still exist many gaps in the research. Low EA is the key factor in the etiology of the Triad, but the impact of low EA on reproductive function can be modified by gynecological age, psychological factors, genetics, and likely many other factors. As such, a more individualized approach to diagnosing and treating low EA is warranted and more research is necessary to improve the measurement of EA and how these measurements are incorporated into decisions regarding clearance and return to play. The difficulties of diagnosing and treating the increasing number of athletes with DE also represent key challenges in Triad research going forward. Screening instruments that are validated for use

in individual athletes are needed as are effective educational and behavioral interventions applied to both coaches and athletes for the prevention and treatment of ED and DE. Recently published guidelines for determining Triad risk stratification and providing guidance for clearance and return to play represent a critical step in the advancement of an evidence based translation and need to be refined and validated going forward. It is critical that sports medicine practitioners and researchers continue to work together with these challenges in mind to achieve the goal of reducing the prevalence of the Female Athlete Triad.

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## Key points

The impact of low EA on reproductive function can be modified by gynecological age, psychological factors, and genetics. As such, a more individualized approach to diagnosing and treating low EA is warranted.

In practice, the accurate measurement of EA (in combination with the difficulties of diagnosing and treating the increasing number of athletes with DE) represent key challenges in Triad research going forward.

Recently published guidelines for determining Triad risk stratification including guidance for clearance and return to play represent a critical step in the advancement of evidence based translation, but need to be refined and validated moving forward. It is critical that sports medicine practitioners and researchers work together to achieve this goal which, in turn, will more effectively reduce the prevalence of the Female Athlete Triad.