# Female Athlete Triad

## Future Directions for Energy Availability and Eating Disorder Research and Practice

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**KEYWORDS**

- Low energy availability • Eating disorders • Female • Athlete • Exercise
- Female Athlete Triad

**KEY POINTS**

- The impact of low energy availability (EA) on reproductive function can be modified by gynecologic 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 disordered eating) 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.

### INTRODUCTION

In the 1990s, the concept of the Female Athlete Triad was introduced, drawing attention to a syndrome of 3 tightly interrelated conditions: disordered eating (DE), amenorrhea, and osteoporosis.[1] The definition of the Triad was revised in 2007 to its current meaning to include 1 or more of the following 3 components:

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

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This also includes the continuum between healthy and unhealthy states for each of the 3 elements.[2] Several seminal studies in the 1980s[3–5] piqued the interest of clinicians and researchers alike, and almost 4 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 physiologic 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 and in the translation of research into practice. The purpose of this article is to highlight future potential directions for research by drawing attention to areas in the Triad literature that require clarification. From there, these 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 a focus of Triad research 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.[3,26] 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, EA) is increased during periods of exercise. This demonstrates a causal role of low EA in the induction and the vital role it plays in the reversal of exercise-associated menstrual disturbances.[25,27] 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,[28,29] and by the dysregulation of important bone-related hormones when Triad conditions are present.[30–33]

Although the causal role of low EA in the development of Triad conditions is well supported, several issues deserve consideration. The elegant studies of Loucks and colleagues[34] 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).

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

Short-term (5-day) reductions in EA below a threshold of 30 kcal/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.[35] A slowing of LH pulse frequency is, in turn, associated with delays in folliculogenesis, luteal phase shortening, and more severe menstrual disturbances.[25,36–38] This reduced LH pulse frequency occurs regardless of whether EA is reduced via diet, exercise, or a combination of the two.[34] When EA is considered to be the energy required to support a body's basic physiologic