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Low Energy Availability as a Key Factor in Exercise-induced Menstrual Dysfunction – a Narrative Review

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Abstract

Background. Low energy availability (LEA) is increasingly recognized as a key factor underlying physiological disturbances in physically active women, particularly exercise-induced menstrual dysfunction (EIMD). Energy imbalance may impair reproductive function via neuroendocrine disruption of the hypothalamic–pituitary–gonadal axis.

Aim. This narrative review synthesizes current evidence on LEA as an etiological factor in EIMD, focusing on physiological mechanisms, spectrum of menstrual disorders, and clinical implications within the Female Athlete Triad and Relative Energy Deficiency in Sport (RED-S) frameworks.

Material and methods. A targeted literature search was conducted in PubMed and supplemented by Google Scholar. Over twenty key publications were selected based on relevance and scientific quality, including consensus statements, reviews, and original experimental and observational studies.

Results. LEA was identified as the primary driver of EIMD, acting through suppression of gonadotropin-releasing hormone pulsatility and reduced luteinizing hormone secretion. These changes lead to menstrual disturbances ranging from luteal phase defects and anovulation to oligomenorrhea and functional hypothalamic amenorrhea. Evidence suggests that energy deficiency, rather than exercise itself, is the main causal factor. LEA also underlies broader syndromes such as the Female Athlete Triad and RED-S, affecting multiple physiological systems beyond reproduction.

Conclusions. LEA is a key modifiable factor in prevention and management of EIMD. Early identification and restoration of adequate energy availability are essential to prevent short- and long-term health consequences in physically active women.

Keywords: low energy availability; menstrual dysfunction; female athlete; RED-S; functional hypothalamic amenorrhea

1. Introduction

Regular physical activity is widely acknowledged as a key component of a healthy lifestyle; however, excessive training combined with inadequate energy intake may lead to adverse health outcomes in women.

Low energy availability (LEA) is increasingly recognized as a primary mechanism underlying a spectrum of physiological disturbances in physically active women, particularly exercise-induced menstrual dysfunction. Energy availability (EA) refers to the amount of dietary energy remaining for physiological functions after accounting for energy expended during exercise. When energy intake is insufficient to meet both exercise demands and basic physiological needs, LEA occurs, leading to disruptions in multiple systems, including the reproductive axis [1, 2].

Exercise-induced menstrual dysfunction (EIMD), encompassing conditions such as luteal phase defects, oligomenorrhea, and functional hypothalamic amenorrhea, is commonly observed among athletes and physically active women. The prevalence of such disorders varies depending on the population and type of activity but may affect up to 50% of exercising women in certain high-risk groups [3, 4, 5]. These disturbances are not merely benign adaptations but reflect underlying neuroendocrine dysregulation, particularly involving suppression of the hypothalamic–pituitary–gonadal (HPG) axis.

The pathophysiological link between LEA and menstrual dysfunction is primarily mediated through alterations in metabolic and hormonal signals, including leptin, insulin, cortisol, and ghrelin, which influence gonadotropin-releasing hormone (GnRH) pulsatility. Reduced GnRH secretion leads to decreased luteinizing hormone (LH) pulsatility, ultimately impairing ovarian function [6, 7]. Importantly, these changes can occur even in the absence of clinically apparent eating disorders, highlighting that LEA may be unintentional and driven by mismatched energy intake and expenditure [8].

LEA is also the cornerstone of broader syndromes such as the Female Athlete Triad and Relative Energy Deficiency in Sport (RED-S), both of which extend beyond reproductive dysfunction to affect bone health, metabolism, immunity, and cardiovascular function [4, 9]. The conceptual shift from the Female Athlete Triad to RED-S reflects a more comprehensive understanding of the systemic consequences of inadequate energy availability [4].

Given the increasing participation of women in competitive and recreational sports, understanding the role of LEA in the development of menstrual dysfunction is essential for early identification, prevention, and management of these conditions. This paper aims to synthesize current evidence on the mechanisms linking LEA with exercise-induced menstrual disturbances and to highlight clinical implications for athletes and healthcare providers.

2. Aim

The aim of this narrative review is to synthesize current evidence on low energy availability (LEA) as a key etiological factor in exercise-induced menstrual dysfunction (EIMD). Particular emphasis is placed on the physiological mechanisms linking LEA to disturbances of the hypothalamic–pituitary–gonadal (HPG) axis, the spectrum of menstrual disorders observed in physically active women, and the clinical implications within the frameworks of the Female Athlete Triad and Relative Energy Deficiency in Sport (RED-S). Additionally, this review seeks to highlight the importance of early identification and management of LEA to prevent long-term health consequences.

3. Materials and Methods

This narrative review is based on a targeted literature search conducted in the PubMed database and supplemented by Google Scholar to ensure comprehensive coverage of relevant publications. The search focused on studies examining the relationship between low energy availability (LEA), exercise, and menstrual dysfunction in physically active women.

Key search terms included combinations of the following: “low energy availability”, “energy availability”, “exercise-induced menstrual dysfunction”, “functional hypothalamic amenorrhea”, “Female Athlete Triad”, and “Relative Energy Deficiency in Sport (RED-S)”.

The selection of literature was guided by relevance to the topic, scientific rigor, and contribution to the understanding of underlying physiological mechanisms and clinical outcomes. Particular emphasis was placed on review articles, consensus statements, and selected original studies addressing the effects of energy deficiency on the hypothalamic–pituitary–gonadal (HPG) axis and menstrual function.

A total of over twenty publications were included in the final analysis. These comprised review articles, consensus statements, and selected original studies, including experimental and

observational research, providing both mechanistic and clinical perspectives on the effects of low energy availability. Older experimental studies were included where appropriate to illustrate foundational physiological mechanisms, particularly in relation to luteinizing hormone pulsatility and neuroendocrine adaptations to energy deficiency.

Inclusion criteria were as follows:
(i) studies involving physically active women or female athletes,
(ii) research addressing energy availability and/or menstrual function,
(iii) articles published in peer-reviewed journals in English.

Exclusion criteria included:
(i) studies not involving human subjects,
(ii) publications not directly related to the interaction between energy availability and reproductive function,
(iii) non-peer-reviewed sources.

Given the narrative nature of this review, no formal quality assessment or meta-analysis was performed. Instead, the selected literature was analyzed and synthesized to provide a coherent overview of the role of low energy availability in exercise-induced menstrual dysfunction.

4.1 Definition of energy availability

Energy availability (EA) is defined as the amount of dietary energy remaining for physiological processes after subtracting the energy expended during exercise. It is typically expressed relative to fat-free mass (FFM) and calculated as:

$$\text{EA} = \frac{\text{Energy Intake} - \text{Exercise Energy Expenditure}}{\text{Fat-Free Mass}}$$

EA is considered a more physiologically relevant metric than energy balance, as it reflects the energy available to sustain essential biological functions, including thermoregulation, cellular maintenance, growth, and reproduction [1, 4].

In healthy adult women, an EA of approximately 45 kcal/kg FFM/day is regarded as optimal for maintaining normal physiological function. In contrast, low energy availability (LEA) is typically defined as EA below 30 kcal/kg FFM/day, a threshold below which disruptions in endocrine and metabolic processes are likely to occur [4, 9]. It is important to note that these

thresholds are not absolute and may vary between individuals depending on factors such as training status, age, and genetic predisposition [9].

A key distinction must be made between EA and overall energy balance. An individual may be in energy balance (stable body weight) while still experiencing LEA, due to compensatory physiological adaptations such as reductions in resting metabolic rate or alterations in hormonal function. Therefore, EA provides a more sensitive indicator of physiological stress than body weight or caloric balance alone [1].

LEA can arise from intentional behaviors, such as dietary restriction, or unintentionally, when energy intake fails to match high exercise energy expenditure. Importantly, LEA does not necessarily require the presence of an eating disorder, and it is frequently observed in athletes without clinically diagnosed disordered eating [8].

Accurate assessment of EA in free-living individuals remains challenging. Measurement of energy intake is prone to underreporting, while exercise energy expenditure is difficult to quantify precisely, particularly in athletes with variable training loads. Additionally, the estimation of FFM introduces further variability. As a result, EA is often approximated rather than directly measured, which should be considered when interpreting research findings [6, 9].

4.2 Pathophysiology

Low energy availability (LEA) induces a range of adaptive physiological responses aimed at conserving energy for vital functions. One of the primary systems affected is the hypothalamic–pituitary–gonadal (HPG) axis, which is highly sensitive to energy status. Disruption of this axis is the central mechanism underlying exercise-induced menstrual dysfunction [1, 4, 10].

At the hypothalamic level, LEA leads to suppression of gonadotropin-releasing hormone (GnRH) pulsatility. This process is mediated by energy-sensing pathways involving key metabolic hormones such as leptin, insulin, ghrelin, and cortisol. Reduced energy availability decreases circulating leptin levels, reflecting diminished energy stores, which in turn signals an unfavorable metabolic environment for reproduction. Leptin plays a permissive role in reproductive function, and its reduction contributes to inhibition of GnRH secretion [7, 8].

In parallel, increased ghrelin and cortisol levels further reinforce this inhibitory effect on hypothalamic function. Ghrelin, an orexigenic hormone elevated during energy deficiency, has

inhibitory effects on the reproductive axis, while chronic elevations in cortisol—associated with physiological stress—also contribute to suppression of GnRH pulsatility [7]. Additionally, alterations in kisspeptin signaling, a key regulator of GnRH neurons, are implicated in mediating the effects of LEA on reproductive function, although the exact mechanisms remain an area of ongoing research [10].

At the pituitary level, reduced GnRH pulsatility results in decreased frequency and amplitude of luteinizing hormone (LH) pulses [1, 9]. This disruption is critical, as normal LH pulsatility is required for follicular development and ovulation. Consequently, impaired LH secretion leads to inadequate ovarian stimulation. This phenomenon has been well documented in experimental studies of energy deficiency [2, 11].

At the ovarian level, these upstream disturbances translate into impaired folliculogenesis, reduced estradiol production, and anovulation. Clinically, this manifests as a spectrum of menstrual disturbances, ranging from subtle luteal phase defects to complete functional hypothalamic amenorrhea [6].

Importantly, these reproductive adaptations are part of a broader energy-conserving response. LEA is also associated with suppression of other endocrine axes, including reductions in triiodothyronine (T3) and insulin-like growth factor 1 (IGF-1), as well as decreased resting metabolic rate. These systemic changes further reflect the body's prioritization of essential survival processes over reproduction [1, 9].

Notably, the onset and severity of these alterations are influenced not only by the absolute level of energy availability but also by the duration of LEA and individual susceptibility. Even short-term reductions in EA can lead to measurable changes in LH pulsatility, highlighting the rapid responsiveness of the reproductive axis to energy deficiency [1].

4.3 Menstrual disorders

Menstrual disturbances associated with low energy availability (LEA) exist along a continuum, ranging from subtle subclinical abnormalities to complete cessation of menses. These disorders are collectively referred to as exercise-induced menstrual dysfunction (EIMD) and reflect varying degrees of disruption within the hypothalamic–pituitary–gonadal (HPG) axis [1, 6].

Recent evidence further supports the association between LEA and the full spectrum of menstrual disturbances observed in physically active women [6, 12]. At the milder end of this spectrum are luteal phase defects (LPD), which are characterized by insufficient progesterone production or shortened luteal phases despite apparently regular menstrual cycles. These disturbances often go unrecognized clinically but may indicate early impairment of ovarian function due to altered luteinizing hormone (LH) pulsatility [7]. Anovulatory cycles may also occur despite normal cycle length, indicating that regular menstruation does not necessarily reflect normal reproductive function.

With increasing severity of energy deficiency, more overt menstrual irregularities develop, including oligomenorrhea, defined as menstrual cycles longer than 35 days. This condition reflects more pronounced suppression of GnRH and LH pulsatility, resulting in inconsistent or absent ovulation [9].

At the most severe end of the spectrum lies functional hypothalamic amenorrhea (FHA), defined as the absence of menstruation for at least three months in the absence of organic pathology. FHA represents a state of profound neuroendocrine suppression, in which GnRH pulsatility is significantly reduced or absent, leading to markedly decreased secretion of LH and follicle-stimulating hormone (FSH), and consequently low estradiol levels [1, 6].

Importantly, these menstrual disturbances are reversible and are considered functional adaptations to inadequate energy availability rather than structural pathology. However, prolonged hypoestrogenism associated with chronic LEA has significant clinical consequences, including impaired bone mineral density, increased risk of stress fractures, and potential long-term effects on cardiovascular and reproductive health [4, 9].

The prevalence of menstrual disorders is particularly high in athletes participating in sports emphasizing leanness or endurance, where the mismatch between energy intake and expenditure is common [5]. Notably, LEA-related menstrual dysfunction can occur even in the absence of clinically diagnosed eating disorders, underscoring the importance of considering energy availability as an independent risk factor [8].

Overall, menstrual dysfunction in physically active women should be viewed as an early clinical marker of underlying LEA and systemic physiological stress, rather than a benign or expected consequence of training.

4.4 LEA and exercise-induced dysfunction

Although menstrual disturbances have long been associated with high levels of physical activity, current evidence indicates that low energy availability (LEA), rather than exercise per se, is the primary etiological factor underlying exercise-induced menstrual dysfunction (EIMD) [1, 4].

Early observations suggested that intensive training directly suppressed reproductive function. However, subsequent research has demonstrated that when adequate energy intake is maintained, even high training loads do not necessarily lead to menstrual disturbances. This has shifted the paradigm from an “exercise-induced” to an “energy deficiency–induced” model of reproductive dysfunction [1].

Experimental studies have provided strong support for this concept, demonstrating that disruptions in reproductive function are primarily driven by energy deficiency rather than exercise itself [3]. Short-term reductions in energy availability, even over a period of a few days, have been shown to disrupt luteinizing hormone (LH) pulsatility, independent of exercise volume [9]. Conversely, restoration of adequate energy intake can normalize neuroendocrine function, even in the presence of continued training. These findings underscore the sensitivity of the hypothalamic–pituitary–gonadal (HPG) axis to energy status rather than physical activity alone [13].

LEA contributes to EIMD through a cascade of metabolic and hormonal adaptations, including decreased leptin and insulin levels, increased ghrelin and cortisol concentrations, and subsequent suppression of gonadotropin-releasing hormone (GnRH) secretion. These changes collectively impair ovarian function, leading to the spectrum of menstrual disturbances described previously [7].

Importantly, LEA may occur both intentionally, as in the case of dietary restriction aimed at improving body composition or performance, and unintentionally, when increased training demands are not matched by adequate nutritional intake. This is particularly relevant in endurance and aesthetic sports, where high energy expenditure and pressure to maintain low body weight frequently coexist [5, 14].

Furthermore, the development of menstrual dysfunction is influenced by the duration and severity of LEA, as well as individual susceptibility. Not all women exposed to similar training

loads and energy deficits develop EIMD, suggesting variability in physiological resilience and adaptive capacity [6].

Taken together, these findings highlight that exercise acts primarily as a contributing factor by increasing energy expenditure, while LEA represents the primary underlying cause of reproductive dysfunction. Recognizing this distinction is essential for both prevention and management, as interventions should focus on restoring adequate energy availability rather than reducing physical activity alone.

4.5 Female Athlete Triad and RED-S

Low energy availability (LEA) constitutes the central underlying mechanism of broader clinical syndromes affecting physically active individuals, most notably the Female Athlete Triad and Relative Energy Deficiency in Sport (RED-S). These frameworks provide a more comprehensive understanding of the multisystem consequences of chronic energy deficiency [1, 4].

The Female Athlete Triad, originally described as the interrelationship between disordered eating, menstrual dysfunction, and low bone mineral density, has evolved into a spectrum model in which each component may vary in severity. LEA is now recognized as the primary driver of all three components, with or without the presence of clinically diagnosed eating disorders [15, 16, 17]. Within this model, menstrual dysfunction reflects disruption of the hypothalamic–pituitary–gonadal (HPG) axis, while impaired bone health results from chronic hypoestrogenism and reduced anabolic signaling [9, 18].

However, the Triad framework has limitations, particularly its focus on female athletes and a relatively narrow set of clinical outcomes. To address these limitations, the International Olympic Committee (IOC) introduced the concept of RED-S, which expands the scope to include both sexes and a wider range of physiological systems affected by LEA [4].

RED-S encompasses impairments in metabolic rate, menstrual function, bone health, immunity, protein synthesis, and cardiovascular health, among others. In this model, reproductive dysfunction is one of several clinical manifestations of inadequate energy availability rather than an isolated condition [4, 9, 19]. This broader perspective highlights that LEA induces systemic adaptations aimed at conserving energy, often at the expense of non-essential functions such as reproduction and long-term tissue maintenance.

Importantly, both the Female Athlete Triad and RED-S emphasize that LEA may occur across a wide spectrum of athletes, including those who appear healthy and maintain normal body weight. This is particularly relevant in clinical practice, as reliance on body mass index (BMI) alone may fail to identify individuals at risk [8].

From a clinical standpoint, the integration of these models underscores the importance of early detection of LEA and its consequences. Menstrual dysfunction should be viewed not only as a reproductive issue but also as a marker of broader physiological dysregulation with potential long-term health implications, particularly for bone health and overall metabolic function [9, 18].

In summary, the Female Athlete Triad and RED-S frameworks provide essential context for understanding exercise-induced menstrual dysfunction as part of a systemic response to inadequate energy availability, reinforcing the need for a multidisciplinary approach to prevention, diagnosis, and management.

4.6 Clinical implications

Recognition of low energy availability (LEA) as the primary driver of exercise-induced menstrual dysfunction (EIMD) has important implications for clinical practice, particularly in the fields of sports medicine, gynecology, and primary care. Early identification of LEA is essential, as menstrual disturbances often represent one of the first clinically observable signs of underlying physiological dysregulation [1, 9, 20].

A key challenge in clinical settings is that LEA frequently occurs in individuals who do not meet criteria for eating disorders and may present with normal body weight. As a result, reliance on body mass index (BMI) or outward appearance alone is insufficient for identifying at-risk individuals. Instead, clinicians should adopt a comprehensive approach that includes detailed assessment of dietary intake, training load, menstrual history, and psychosocial factors, particularly in athletes exposed to sport-specific pressures related to body composition and performance [5, 8, 16].

Menstrual irregularities, including oligomenorrhea or amenorrhea, should not be considered a normal consequence of intensive training and should always warrant clinical evaluation. They should prompt evaluation for LEA and related conditions such as the Female Athlete Triad or RED-S. Early recognition is critical, as prolonged hypoestrogenism may lead to decreased bone

mineral density and increased risk of stress fractures, particularly in adolescent and young adult athletes [18]. These effects are particularly concerning due to their potential long-term impact on skeletal health and peak bone mass acquisition.

Management of LEA-related menstrual dysfunction should primarily focus on restoring adequate energy availability. This is typically achieved through a combination of increased energy intake, modification of exercise load, or both. Nutritional rehabilitation remains the cornerstone of treatment, with the goal of achieving an energy availability sufficient to support normal physiological function. Importantly, non-pharmacological interventions should be prioritized, as hormonal therapies (e.g., oral contraceptives) may mask symptoms without addressing the underlying cause [1, 9].

A multidisciplinary approach is often required, involving physicians, dietitians, psychologists, and coaches. Education plays a critical role in both prevention and treatment, particularly in athletic populations where misconceptions about body weight and performance may contribute to the development of LEA [14].

Finally, screening strategies should be incorporated into routine care of physically active women, especially those participating in high-risk sports. Standardized tools and questionnaires may assist in identifying individuals at risk, although their sensitivity and specificity vary. Continued research is needed to improve early detection methods and to establish evidence-based guidelines for management [21].

In summary, effective management of exercise-induced menstrual dysfunction requires recognition of LEA as the underlying cause and implementation of targeted interventions aimed at restoring energy balance and preventing long-term health consequences.

5. Conclusions

Low energy availability (LEA) is the central mechanistic factor underlying exercise-induced menstrual dysfunction. Current evidence consistently indicates that menstrual disturbances in physically active women represent a physiological adaptation to insufficient energy supply rather than a direct consequence of exercise itself [1, 4].

The spectrum of menstrual disorders associated with LEA ranges from subtle luteal phase defects to oligomenorrhea and functional hypothalamic amenorrhea, reflecting progressive

impairment of neuroendocrine regulation [6]. These disturbances should be interpreted as early clinical markers of systemic energy deficiency rather than isolated gynecological conditions.

The broader frameworks of the Female Athlete Triad and Relative Energy Deficiency in Sport (RED-S) further emphasize that LEA affects multiple physiological systems beyond reproduction, including bone health, metabolism, and cardiovascular function [4, 9]. This underscores the need for a holistic clinical approach rather than a solely reproductive focus.

Effective prevention and management require early identification of at-risk individuals and restoration of adequate energy availability through nutritional and training interventions. Importantly, menstrual dysfunction should not be normalized in athletic populations, as prolonged LEA may lead to significant short- and long-term health consequences [8, 18].

In conclusion, LEA should be regarded as the primary target in both the understanding and management of exercise-induced menstrual dysfunction, with multidisciplinary strategies essential for preserving reproductive and overall health in physically active women.

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