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## Reproductive Disorders among Female Athletes: Causes, Pathomechanism, and Treatment Approaches – A Literature Review

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**Abstract**

**Background.** Female sport participation has grown rapidly, outpacing sex-specific clinical guidelines and leaving reproductive disorders under-recognised and often normalised as a marker of training success. The conceptual model has shifted from the narrow Female Athlete Triad to the broader Relative Energy Deficiency in Sport (RED-S) framework, yet diagnostic delay remains common.

**Aim.** To synthesise current literature on the spectrum of reproductive disorders in female athletes and provide a clinically actionable framework for aetiology, diagnosis, and management.

**Material and methods.** A narrative review of original studies, randomised controlled trials, systematic reviews, meta-analyses, and consensus statements from the International Olympic Committee, American College of Sports Medicine, Endocrine Society, and American Society for Reproductive Medicine.

**Results.** Low energy availability (LEA) was identified as the primary trigger of GnRH suppression, with effects on skeletal, cardiovascular, metabolic, and psychological health. Diagnosis requires a systematic algorithm; management is based on nutritional and psychological intervention, with transdermal hormonal replacement as a last resort. Widespread oral contraceptive prescription is a counterproductive practice masking the underlying energy deficit.

**Conclusions.** Reproductive dysfunction in female athletes reflects a systemic energy-conservation response, not an isolated gynaecological finding. Closing the diagnostic gap requires screening integration, education, and a cultural shift away from normalising amenorrhea in sport.

**Key words:** relative energy deficiency in sport, low energy availability, functional hypothalamic amenorrhea, female athlete triad

**Abbreviations:** ACSM-American College of Sports Medicine; AMH-Anti-Müllerian Hormone; BMD-Bone Mineral Density; CAT-Clinical Assessment Tool; CBT- Cognitive Behavioural Therapy; CRH - Corticotropin-Releasing Hormone; DHEA-S- Dehydroepiandrosterone Sulphate; DXA-Dual-Energy X-ray Absorptiometry; EA- Energy Availability; FFM- Fat-Free Mass; FHA- Functional Hypothalamic Amenorrhea; FSH-Follicle-Stimulating Hormone; GnRH- Gonadotropin-Releasing Hormone; HPA-axis- Hypothalamic-Pituitary-Adrenal axis; IGF-1-Insulin-like Growth Factor-1; IOC- International Olympic Committee; LDL- Low-Density Lipoprotein; LEA- Low Energy Availability; LEAF-Q - Low Energy Availability in Females Questionnaire; LH- Luteinizing Hormone; LPD - Luteal Phase Deficiency; MRI-Magnetic Resonance Imaging; OCP - Oral Contraceptive Pill; PCOS - Polycystic Ovary Syndrome; PYY- Peptide YY; RED-S - Relative Energy Deficiency in Sport; rT3 - Reverse Triiodothyronine; T3 - Triiodothyronine; T4 -Thyroxine; TSH - Thyroid-Stimulating Hormone.

## **1. Introduction**

### **1.1 Female Athletes in Sport**

Over the past decades, the percentage of women in competitive sports has grown exponentially, which has brought to light gender-specific health disorders. Yet this expansion in participation has not been matched by a proportional expansion in evidence-based sports-medicine physiology and training science for women. Female athletes, who are constantly under pressure to improve performance, endure enormous physical strain and, in some disciplines, maintain a specific body weight, have become more prone to chronic energy deficits. Historically, guidelines have been based on the research conducted predominantly in male cohorts, leaving clinicians without sex-specific algorithms for diagnosing and managing the reproductive health consequences of intensive training in women.

### **1.2 Changing the paradigm: From the Female Athlete Triad to RED-S**

Over the past three decades, the clinical concept of exercise-associated reproductive dysfunction has undergone substantial revision. The construct now known as the Female

Athlete Triad was first articulated by an American College of Sports Medicine (ACSM) task force in 1992 and formalised in the 1997 ACSM Position Stand, which described a three-node syndrome of disordered eating, amenorrhea, and osteoporosis in physically active girls and women [1]. This original approach was criticised for its reliance on categorical, end-stage clinical endpoints, thereby excluding athletes with milder, subclinical presentations from timely recognition. The 2007 ACSM Position Stand update addressed this limitation directly, replacing the categorical "disordered eating" node with the continuum construct of low energy availability (LEA) and reframing all three components: energy availability, menstrual function, and bone mineral density as a sliding spectrum rather than fixed disease states [2].

However, the most significant conceptual advance came in 2014, when an International Olympic Committee (IOC) expert working group introduced the term Relative Energy Deficiency in Sport (RED-S), replacing the Triad with a subset of a more extensive, multisystem syndrome. The 2014 IOC Consensus Statement expanded the model beyond bone and reproductive health to highlight the consequences of limited energy availability on immune, cardiovascular, and gastrointestinal function, metabolic rate, haematological status, growth and development, and psychological health. It broadened it to include male and female athletes [3]. In 2018, the IOC presented its first consensus, with an accumulated evidence base and clinical guidance for sports physicians [4], and in 2023, a further, substantially revised statement was published that introduced a 4-class severity classification model and an updated Clinical Assessment Tool (CAT) for making decisions on returning to play based on risks [5].

### **1.3 Amenorrhea as a Marker of Success**

A persistent obstacle to early diagnosis is the perception of menstrual dysfunction within sporting culture itself. Studies were carried out to identify the cause of the underreporting of amenorrhoea by female athletes. It was found that normalisation by medical practitioners, coaches, and fellow athletes was the single most frequently mentioned barrier, with amenorrhea often informally viewed as a marker of training success rather than as a clinical warning sign [6]. This normalisation compounds a separate, well-documented pattern of underreporting: menstrual cycle disturbances are known to go unreported by exercising women at rates approaching approximately 40% in some groups [6], and in athletes from lean-build disciplines specifically, roughly one in five has indicated they would not report amenorrhea even if it occurred [8]. The consequence of this combination of normalisation and underreporting is a diagnostic delay that allows the underlying energy deficit and its musculoskeletal, cardiovascular, and metabolic consequences to progress before clinical intervention occurs.

This pattern should be understood as a feature of embedded sport culture and inadequate athlete/coach education, rather than as a failure of individual clinical vigilance.

## **2. The Clinical Manifestations of Reproductive Disorders among Sportswomen**

### **2.1 Epidemiology**

Without counting pregnancy, lactation, and menopause, amenorrhea affects approximately 3–4% of women in the general population [9]. In comparison with this baseline, the athletic population shows a markedly elevated and highly heterogeneous burden of menstrual dysfunction. A review of 48 studies across Olympic disciplines found reported prevalence of menstrual disorders ranging from 0% to 61%. The highest rates were observed in the following disciplines: for secondary amenorrhea in cycling (56%) and triathlon (40%), and for oligomenorrhea in boxing (55%) and rhythmic gymnastics (44%) [10]. A subsequent systematic review confirmed this rate range. However, it was noted that most data came from self-reported cycle histories rather than hormonally confirmed ovulatory status, which likely understates the true prevalence of menstrual disturbances in other sports disciplines [11]. Importantly, more recent data indicate that these manifestations are not limited to endurance and aesthetic disciplines. In a survey of 584 elite German athletes across 64 sports, lifetime oligomenorrhea reached 74% and lifetime secondary amenorrhea 40%, with primary amenorrhea specifically elevated in aesthetic sports. In contrast, rates of oligomenorrhea and secondary amenorrhea did not differ significantly by discipline category, thus indicating the demand for systematic gynaecological screening among elite athletes across sport types, not only lean-build or endurance sportswomen [12].

### **2.2 The Pathological Course Reproductive Suppression**

#### **2.2.1 Delayed Menarche**

At the earliest point of the continuum, delayed menarche is clinically defined as the absence of menarche by age 15 in the presence of otherwise normal secondary sexual development [9]. When intensive training begins before menarche, the resulting energy deficit can delay the onset of hypothalamic-pituitary-ovarian activation required for pubertal cycling, thereby postponing menarche beyond this threshold. Because the peripubertal and adolescent years constitute the main window for bone mineral accumulation toward peak bone mass, a delayed onset of ovarian estrogen exposure during this window carries consequences for lifelong skeletal trajectory that extend well beyond the reproductive system itself.

#### **2.2.2 Luteal Phase Deficiency and Anovulation as Early Subclinical Manifestations**

The subclinical stage of the process is arguably the most diagnostically significant, precisely because it is invisible to standard diagnostic methods, such as self-report of menstrual cycle

length. Luteal phase deficiency (LPD) is clinically defined by a luteal phase length of  $\leq 10$  days, arising from inadequate progesterone secretion, inadequate progesterone duration, or endometrial progesterone resistance [13]. Athletes can present with shortened luteal phases and blunted progesterone output while continuing to report bleeding at seemingly regular intervals. The described pattern led to the identification of LPD as the most common menstrual disturbance affecting the largest proportion of physically active women, precisely because it produces no evident change in reported cycle length [14]. Studies of recreational runners using hormonal markers have directly confirmed this pattern, revealing luteal phase deficiency, even in runners who did not report menstrual irregularity [15]. The 2010 research showed a high prevalence of both subtle (anovulation, LPD) and severe (oligomenorrhea, amenorrhea) menstrual disorders among exercising women overall based on daily urinary and serum hormone sampling rather than self-report, highlighting that self-reported "eumenorrhea" is an unreliable representation for ovulatory competence, and that ambulatory basal body temperature charting, mid-luteal serum progesterone, or urinary pregnanediol glucuronide assays are required to detect this tier of dysfunction in athlete screening programs [7].

### **2.2.3 Oligomenorrhea and Functional Hypothalamic Amenorrhea**

At the other end of the continuum, oligomenorrhea is defined as fewer than nine menstrual cycles per year, or a cycle length over 35 days, while secondary amenorrhea is defined as the absence of menses for three or more consecutive months in a previously regularly cycling individual [9]. When amenorrhea in an athlete after diagnostic research cannot be attributed to an organic, anatomic, or pharmacologic cause, the resulting diagnosis is Functional Hypothalamic Amenorrhea (FHA), a diagnosis of exclusion by definition rather than a probable one.

### **2.3 Hyperandrogenism and PCOS-like Phenotypes in Power and Strength Sport**

Another observation noted among female athletes is that elevated androgen concentrations and ovarian morphology typical of PCOS have been documented. A case-control study of Olympic-level competitors found functional hyperandrogenism common enough across disciplines [16]. That phenomenon could potentially explain a considerable portion of reproductive dysfunction in this subgroup, independent of the energy-deficient FHA mechanism [16]. Elevated free and total testosterone have positive effects on muscle mass, power output, and recovery capacity. Yet at the same time, the presented hyperandrogenic, often combined with ovulation disturbances, phenotype meets the same Rotterdam diagnostic criteria (any two of oligo/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasound) used to diagnose PCOS in any non-athlete group. It carries the same

associated long-term metabolic risk profile of insulin resistance, dyslipidemia, and endometrial hyperplasia risk from chronic anovulatory unopposed estrogen exposure [17]. The clinical significance of this phenomenon is that this phenotype must be actively distinguished from FHA at initial presentation: both may present with oligomenorrhea or amenorrhea, but the two require opposite first-line management logic (energy repletion for FHA and metabolic and androgen-directed management for the hyperandrogenic phenotype), making the differential diagnostic algorithm essential.

### **3. Aetiology and Pathomechanisms**

#### **3.1 Low Energy Availability**

The primary trigger responsible for the entire spectrum of reproductive disturbances is Low Energy Availability (LEA). Energy Availability (EA) is formally defined as  $EA = (Energy\ Intake - Exercise\ Energy\ Expenditure) / Fat-Free\ Mass\ (FFM)$ , expressed in kcal/kg FFM/day. EA is the energy remaining for all other physiological processes after subtracting the metabolic cost of exercise from dietary intake. The critical threshold for reproductive disturbance was established in a controlled study, in which the EA of regularly menstruating, habitually sedentary women was experimentally manipulated for five days across a range of 10, 20, 30, and 45 kcal/kg lean body mass per day; luteinizing hormone (LH) pulse frequency began to decline and pulse amplitude to rise specifically below the 30 kcal/kg/day threshold, establishing this value as the cutoff separating energy-replete from energy-deficient reproductive physiology [18]. This experimental paradigm did not require disordered eating or intentional caloric restriction to produce the hormonal disturbance: EA was manipulated directly through a combination of controlled diet and imposed exercise load. This distinction has direct clinical relevance: LEA arising from deliberate restriction and LEA from insufficient intake relative to an athlete's training regime, meaning that the diagnostic approach to LEA shouldn't be influenced by the presence or absence of an eating disorder [19].

#### **3.2 Suppression of Pulsatile GnRH Secretion**

The following reproductive consequence of sustained LEA is the disruption of the pulsatile secretion of Gonadotropin-Releasing Hormone (GnRH) from arcuate and preoptic hypothalamic neurons. Because GnRH pulse frequency and amplitude directly govern the secretory pattern of LH and FSH from the anterior pituitary, a collapse in pulsatility prevents the mid-cycle LH surge required to trigger ovulation [18]. Follicular development consequently stops at an early stage, and ovarian estradiol secretion falls, causing the biochemical cascade that results in hypogonadotropic hypogonadism with hypoestrogenism, which is a clinical manifestation of low EA, HPA-axis activation, and gastrointestinal starvation.

### **3.3 The Neuroendocrine Signalling Cascade**

#### **3.3.1 Adipokine Signalling: Leptin and Kisspeptin**

The principal metabolic mechanism of this cascade operates through the adipokine leptin and its downstream target, hypothalamic kisspeptin neurons of the arcuate nucleus. Kisspeptin is the immediate upstream trigger for GnRH neurons. In a state of adequate energy balance, adipose-derived leptin provides a metabolic signal that sustains kisspeptin output. Decreased fat mass and declining circulating leptin under conditions of LEA remove this drive, thereby suppressing kisspeptin expression and GnRH pulse secretion [20]. It is worth noting that leptin does not appear to be a sufficient signal on its own: experimental restoration of leptin in animal models to normal basal levels during negative energy balance has not fully restored kisspeptin or LH secretion, indicating that leptin operates as one necessary but not as a single sufficient stimulus [21].

#### **3.3.2 Cortisol and CRH**

A second component of suppression pathomechanisms operates through the hypothalamic-pituitary-adrenal (HPA) axis. Combined psychological and physical training stress activates corticotropin-releasing hormone (CRH) neurons. The 2022 study shows that CRH exerts direct inhibitory input onto GnRH neurons via the central CRH receptor, independent of the leptin-kisspeptin pathway described above, while cortisol elevation exerts additional suppressive effects at both the pituitary and ovarian levels [22]. Because this pathway operates through distinct receptors and neural circuits from the leptin-kisspeptin axis, the combination of energetic and psychological stress in an athlete's training environment should be viewed as a synergistic factor for suppression of the reproductive axis.

#### **3.3.3 Gastrointestinal Starvation Signals: Ghrelin and PYY**

A third arm involves peripheral gastrointestinal hormones that signal acute nutrient status directly to the hypothalamus. Fasting ghrelin, a hormone that rises during negative energy balance, is elevated in physically active women with menstrual disturbances relative to their eumenorrheic, energy-replete counterparts [23]. Peptide YY (PYY), a gut peptide released in response to caloric intake, shows the same directional disturbance: in a controlled comparison of sedentary ovulatory, exercising ovulatory, and exercising amenorrheic women, fasting PYY was elevated specifically in the exercising amenorrheic group and correlated with indices of subclinical disordered eating. Experiments on animal models show that elevated PYY suppresses GnRH-mediated LH secretion and, combined with ghrelin's suppressive effect,

PYY further reinforces reproductive shutdown as part of an energy-sparing neuroendocrine mechanism [24].

### **3.3.4 Thyroid Axis Adaptation**

The fourth arm of adaptation focuses on the thyroid axis and metabolic rate. LEA can be responsible for the induction of a low-T3 (euthyroid sick) syndrome characterised by suppressed serum triiodothyronine with low to normal thyroid-stimulating hormone (TSH) and free T4 as a result of preferential peripheral deiodination toward biologically inactive reverse T3 rather than active T3 [25]. Furthermore, low-T3 syndrome occurs at the same EA threshold that disrupts LH pulsatility [25]. This indicates that reproductive suppression and metabolic rate downregulation are not independent events but co-occurring outputs of the same underlying energy-conservation mechanisms. At the same time, it provides clinicians with a peripherally measurable biomarker (suppressed free T3) that tracks the severity of the same energy-deficient state responsible for reproductive disturbances.

### **3.4 Synthesis: Metabolic Triage and the Hierarchy of Biological Function**

The four arms of this cascade are best understood as coordinated expressions of a single underlying adaptive logic. In a state of LEA, energetic investment is prioritised toward processes vital for immediate survival, with long-term investments such as growth, immune function, and reproduction down-regulated first and restored only once immediate survival needs are secured [26]. Applied to exercising females, this hierarchy-of-function logic explains why energetically expensive but not acutely survival-critical processes such as reproductive function, bone remodelling, and thyroid-driven metabolic rate are suppressed under LEA. In contrast, cardiac and central nervous system functions are preserved [19].

## **4. Diagnostic Approaches in Sports Endocrinology**

### **4.1 Structured Clinical Screening Instruments**

Systematic screening is the first point in the diagnostic algorithm, and there are two validated instruments in sports endocrinology practice. The Low Energy Availability in Females Questionnaire (LEAF-Q) is a 25-item, sport-specific self-report tool assessing three symptom domains: injury history, gastrointestinal function, and menstrual function. The threshold of 8 was established as the risk-stratification cutoff, indicating an athlete likely to be affected by persistent LEA or Triad-related pathology [27]. Validation work in Norwegian elite endurance athletes reported a sensitivity of 78% and a specificity of 90% against this cutoff, supporting its clinical utility as a first-line screening instrument. However, its developers themselves cautioned that further data would be required before extending its use beyond endurance and

aesthetic-sport populations. Complementing the LEAF-Q's symptom-based screening, the RED-S Clinical Assessment Tool (CAT) in its 2023 iteration provides team physicians with a four-tier risk-stratification framework that consolidates clinical, biochemical, and historical risk factors to guide sport-participation clearance decisions [5]. Used together, these two instruments allow for the selection of athletes who demand closer evaluation, with the RED-S CAT then guiding the clinical decision on whether continued training and competition can proceed, be modified, or must be paused.

#### **4.2 The Laboratory Algorithm: Excluding Organic Pathology**

Because Functional Hypothalamic Amenorrhea is, by definition, a diagnosis of exclusion, the clinical practice guideline on FHA outlines a systematic laboratory algorithm to rule out organic and anatomic causes of amenorrhea before an FHA diagnosis can be made [28]. Serum beta-hCG should be obtained first, as pregnancy remains the single most common cause of secondary amenorrhea in any reproductive-age individual and must be excluded before further diagnostic workup. LH, FSH, and estradiol are assessed next: a pattern of low or low-normal gonadotropins accompanied by low estradiol is consistent with the hypogonadotropic hypogonadism characteristic of FHA, whereas an elevated FSH in the presence of low estradiol instead points toward primary ovarian insufficiency and indicates a distinct diagnostic pathway. Serum prolactin is measured to exclude a prolactin-secreting pituitary adenoma, since hyperprolactinemia independently suppresses GnRH secretion and can result in amenorrhea that mimics FHA. TSH, free T4, and free T3 are assessed together, rather than TSH alone, to distinguish primary thyroid disease from the LEA-induced low-T3 pattern. It is important to obtain TSH, free T3, and free T4, since TSH can remain within the normal range even as free T3 is meaningfully suppressed by energy deficiency. The guideline further recommends obtaining total testosterone and DHEA-S in patients with clinical signs of hyperandrogenism, with 17-hydroxyprogesterone added if late-onset congenital adrenal hyperplasia is suspected, to differentiate FHA from the hyperandrogenic phenotype. Finally, Anti-Müllerian Hormone, alongside basal or day-3 FSH, is used to exclude Premature Ovarian Insufficiency, an especially important parameter in athletes whose age profile or family history raises suspicion of early ovarian depletion.

#### **4.3 Pelvic Imaging: Differentiating FHA from PCOS Morphology**

Transvaginal or transabdominal ultrasound provides additional information. FHA is typically characterised by a thin endometrial stripe and small, multifollicular or quiescent-appearing ovaries with reduced antral follicle counts and low stromal echogenicity, typical for the hypoestrogenic, low-gonadotropin-drive state. This differs directly from the ultrasound

phenotype specified by the Rotterdam consensus criteria for PCOS, which include increased ovarian volume and a peripherally arranged, "string of pearls" pattern of small follicles with increased stromal volume [17]. Pituitary MRI is not indicated routinely but should be obtained when neurological symptoms such as headache or visual field defects are present, when prolactin is markedly elevated, or when the biochemical picture remains ambiguous after the laboratory algorithm above. FHA can be diagnosed only after organic and anatomic pathology, including a pituitary mass lesion, has been actively excluded rather than presumed absent [28].

#### **4.4 Bone Health Assessment via DXA**

Dual-Energy X-ray Absorptiometry (DXA) screening should be considered mandatory in any athlete presenting with six or more months of menstrual dysfunction, a history of bone stress injury, or confirmed LEA. In premenopausal individuals, Z-scores, not T-scores, must be used to interpret DXA results, as T-scores are based on a postmenopausal population and would systematically misclassify bone status in young premenopausal athletes. The current Official Positions of the International Society for Clinical Densitometry, a Z-score of  $-2.0$  or lower is classified as "below the expected range for age," and individuals meeting this threshold combined with clinical fracture risk factors should be considered at the osteoporosis-range end of the spectrum, while Z-scores above  $-2.0$  but still below  $-1.0$  in the presence of clinical risk factors should be flagged as low bone mineral density for age and indicate close monitoring and nutritional intervention [29].

### **5. Consequences of Chronic LEA**

#### **5.1 Skeletal Fragility and Failure to Achieve Peak Bone Mass**

Chronic hypoestrogenism removes the restraining influence of estrogen on osteoclast activity, so that resorption outpaces formation. As adolescence and young adulthood constitute the primary window for accruing Peak Bone Mass, menstrual dysfunction during this period can produce a bone deficit which persists even after menses resume. The clinical consequence of this deficit is measurable and substantial: in a cohort of adolescent and young adult athletes, lifetime fracture prevalence was 47% among oligo/amenorrheic athletes compared with 25.7% among eumenorrheic athletes and 12.5% among non-athletic controls, a difference driven overwhelmingly by stress fractures specifically (32% versus 5.9% versus 0%, respectively), and fractured amenorrheic athletes showed lower bone mineral density Z-scores than their non-fractured amenorrheic peers [30]. The anatomic pattern of injury reinforces this mechanism: a study of track and field athletes found that bone stress injuries of the femoral neck, pubic bone, and sacrum specifically were associated with prolonged recovery time, and that female athletes with oligomenorrhea or amenorrhea sustained bone stress injuries of significantly higher MRI

severity grade than their eumenorrheic teammates [31]. Together, this evidence supports that bone stress injuries are not caused by training load alone, but also by a hypoestrogenic, energy-deficient state responsible for the reproductive disturbances.

## **5.2 Cardiovascular Risk and Endothelial Dysfunction**

Estrogen also presents a normal vasoprotective effect through upregulation of endothelial nitric oxide synthase and the resulting nitric oxide-mediated vasodilation, which preserves healthy endothelial function. This effect is measurably lost in a state of chronic hypoestrogenism. In a classic case-control study, endothelium-dependent brachial artery dilation was reduced to 1.08% in athletes with amenorrhea compared with 6.44% in oligomenorrheic athletes and 6.38% in eumenorrheic athletes, establishing a strong association between athletic amenorrhea and impaired endothelial function [32]. A more recent systematic review and meta-analysis of 18 observational studies confirmed this pattern at a larger scale, finding that physically active women with secondary amenorrhea had significantly lower flow-mediated dilation alongside higher total cholesterol, LDL cholesterol, and triglycerides than their eumenorrheic counterparts, in addition to lower basal heart rate and blood pressure [33]. The combination of decreased dilation and an adverse lipid shift in a lean, fit young athlete is a genuine clinical paradox: it represents a vascular risk profile similar to that of a postmenopausal, estrogen-deficient woman, despite the athlete's youth and cardiorespiratory fitness. This underlines cardiovascular risk in this population, and it must be assessed without interference from body fitness or training regimen.

## **5.3 Declines in Athletic Performance and Body Composition**

LEA directly compromises the training adaptations, resulting in impaired training response. Even short-term, severe energy restriction suppresses muscle protein synthesis and reduces fat-free mass growth despite continued high protein intake and ongoing resistance training. Alongside measurable reductions in circulating insulin-like growth factor (IGF-1), compromised protein turnover in collagen-rich connective tissues, such as tendon and ligament, is observed, plausibly explaining the connection between chronic LEA and elevated soft-tissue injury risk [34]. The same source of evidence documents direct impairments in power output, sprint performance, and endurance capacity under conditions of short-term severe LEA, despite the reductions in body mass that athletes and coaches often intend such restriction to produce, questioning the widespread assumption in weight-sensitive sports that a lower body mass reliably improves the power-to-mass ratio and performance [34]. Drained glycogen stores in muscle, a further consequence of chronic energy restriction, intensify this effect by limiting the capacity for repeated high-intensity activity. Therefore, the cumulative cost of LEA in athletes

attempting to lean out for competition can offset or exceed any hypothetical benefit gained from reduced body mass.

## **5.4 Psychological Health**

The final consequence domain is psychological, and the literature here supports a co-occurring rather than strictly unidirectional relationship between reproductive suppression and mood disturbance. A systematic review and meta-analysis of psychological aspects in FHA found that depression and disordered eating attitudes, especially an elevated drive for thinness, were the psychological variables most consistently associated with the condition; in addition, affected women showed higher rates of anxiety, sleep disturbance, dysfunctional attitudes, and alexithymia relative to eumenorrheic comparison groups [35]. An earlier cross-sectional study quantified this association directly, finding that women with FHA scored significantly higher than eumenorrheic controls on standardised depression and anxiety measures, with correspondingly large effect sizes, alongside significantly lower sexual function scores [36]. Rather than treating this association as evidence that psychological distress causes amenorrhea, or that amenorrhea causes distress, the more defensible interpretation draws on the shared neuroendocrine mechanisms. Because HPA-axis activation and CRH signalling directly suppress GnRH pulsatility [22] and also modulate mood-regulating circuitry, HPA-axis dysregulation provides a credible common upstream mechanism capable of producing both the reproductive and psychological findings.

## **6. Multidisciplinary Clinical Management and the Contraceptive Fallacy**

### **6.1 Debunking the Oral Contraceptive Pill**

The widespread prescription of a combined oral contraceptive pill (OCP) to an athlete presenting with amenorrhea is one of the most persistent and evidence-contradicted practices in sports gynaecology, and the critique against it can be built on two explicit grounds. First, the withdrawal bleeding produced during the placebo interval of a combined OCP is a pharmacologic hormone-withdrawal bleed, not evidence of restored endogenous ovulatory function. Because it produces the outward appearance of a normal cycle, it provides false clinical reassurance that can allow the underlying LEA and its sequelae to progress undetected. Second, the pharmacokinetic reasoning explains why the OCP fails to address the bone consequences of that underlying deficit even when it is recognised: unlike physiological ovarian estradiol, which is secreted directly into the systemic circulation, oral ethinyl estradiol undergoes extensive hepatic first-pass metabolism, which suppresses hepatic IGF-1 synthesis and alters IGF-1 binding protein production [37]. Since IGF-1 is a principal anabolic driver of osteoblastic bone formation, this hepatic suppression is directly consequential. A 12-month

randomised clinical trial in 121 oligo-amenorrheic athletes found that transdermal estradiol produced significantly greater gains in lumbar spine and femoral neck bone mineral density than either a combined OCP or no estrogen treatment at all, with the OCP showing essentially no BMD advantage over untreated controls [38]. This finding is the clearest available evidence that the OCP fails at the one outcome it is often prescribed to protect, while simultaneously normalising the athlete's cycle and delaying recognition of the energy deficit. This critique is directed specifically at reflexive OCP use to "fix" amenorrhea in this population, not at hormonal contraception generally, which remains appropriate and effective for its primary indication in athletes who are not using it as a substitute for addressing LEA.

## **6.2 First-Line Management: Restoring Energy Availability**

Reversing the underlying energy deficit is the only intervention capable of correcting the pathophysiology of the described disorders, and it should be positioned as first-line regardless of the athlete's progression. A 12-month randomised controlled trial in 76 exercising women with oligomenorrhea or amenorrhea found that a daily energy intake increase of just 330 kcal (approximately 18% above baseline) was sufficient to induce menstrual recovery, accompanied by modest increases in body weight, fat mass, and total T3 indicative of improved metabolic status [39]. This finding gives clinicians and dietitians a concrete, achievable target, which is clinically important given the perfectionism and fear of weight gain frequently present in this population. This intervention should be delivered by a sports dietitian working within the interdisciplinary team, with a supervised reduction in training volume or intensity where appropriate, and with staged monitoring of resumption of menses, weight and body composition trajectory, and interval reassessment using the LEAF-Q and RED-S CAT instruments.

## **6.3 Second-Line Management: Psychological and Behavioural Intervention**

Cognitive Behavioural Therapy (CBT), including forms designed specifically for eating disorders and disordered eating psychopathology, addresses the perfectionism, body-image distortion, fear of weight gain, and athlete-identity-linked control behaviours that frequently underlie or perpetuate LEA independent of caloric intake alone. The evidence for this approach is not purely theoretical. In a randomised controlled trial, women with FHA assigned to CBT targeting these problematic attitudes were substantially more likely to resume ovarian activity than those assigned to observation alone [40]. A follow-up randomised trial from the same group demonstrated that CBT also decreased hypercortisolaemia and the neuroendocrine and metabolic disturbances characteristic of FHA. In contrast, the observation group showed no such improvement [41]. These outcomes are directly relevant to the HPA-axis mechanism and psychological comorbidity, as CRH-mediated HPA-axis activation independently suppresses

GnRH pulsatility. An intervention that reduces psychological stress reactivity has a plausible direct neuroendocrine pathway to reproductive recovery, not only an indirect effect via improved eating behaviour. For this reason, psychological intervention should run alongside nutritional rehabilitation from the outset of treatment, rather than being introduced only after nutritional measures have been given time to work in isolation.

#### **6.4 Third-Line Management: Wise Hormonal Replacement**

Hormonal therapy should be reserved as a last resort, initiated only when an adequate nutritional rehabilitation has failed to restore menses and bone density parameters continue to decline, a signal of particular urgency in adolescents and young adults still within the peak-bone-mass accrual window [28]. When these conditions are met, the preferred treatment is transdermal 17-beta-estradiol delivered by patch or gel, paired with cyclic oral micronised progesterone to provide endometrial protection against unopposed estrogen exposure. Because transdermal estradiol bypasses hepatic first-pass metabolism entirely, it avoids the IGF-1 suppression that compromises the oral OCP's ability to protect bone, and this advantage is precisely what was demonstrated clinically in the randomised trial showing transdermal estradiol's superior BMD outcomes over combined OCP therapy in oligo-amenorrhoeic athletes [38]. Hormonal replacement at this stage should be understood as compensating for ongoing hypoestrogenism while nutritional and psychological interventions continue to manage the underlying energy deficit.

### **7. Conclusions**

#### **7.1 Reproductive Dysfunction as the Tip of the Iceberg**

This review presents evidence that menstrual and reproductive disturbances in female athletes should not be managed in isolation. Rather, the larger picture should be taken into account, as they reflect a systemic energy-saving program that compromises skeletal, cardiovascular, metabolic, and psychological health. The mechanistic cascade behind it is the same cascade responsible for the failure to achieve peak bone mass, the endothelial dysfunction and adverse lipid shift, the impaired training response, and the depression and anxiety. Treating amenorrhea as a standalone complaint will result in missing the larger picture. The subclinical point of this continuum, luteal phase deficiency and anovulation occurring beneath apparently regular self-reported cycles, likely represents a significantly underdiagnosed group at risk because clinical and research screening have formerly relied on self-reported cycle regularity as a marker for ovulatory competence, which is unreliable.

## **7.2 Recommendations for Sports Governing Bodies and Practice**

Formal integration of RED-S and LEAF-Q screening into pre-participation and periodic health evaluations, rather than leaving such screening to clinician discretion, would close much of the existing diagnostic gap and is consistent with the risk-stratification infrastructure already proposed at the international consensus level [5]. The education programs on menstrual disturbances and energy deficiency for coaching staff are equally necessary, given the evidence that normalisation of amenorrhea by coaches and peers, rather than individual clinical oversight failure, is a leading driver of diagnostic delay [6]. Ultimately, these measures require an institutional cultural shift away from treating amenorrhea as a marker of training success or desirable leanness, and toward treating it as the early-warning clinical sign this review has shown it to be.

## **7.3 Future Research Directions**

The long-term reversibility of bone mineral deficits following adolescent-onset FHA remains incompletely characterised, particularly regarding whether microarchitectural deficits remain despite full weight and menstrual restoration, a gap explicitly identified in the most recent review of this literature [42]. Optimal duration and dosing protocols for transdermal hormonal therapy in athletes remain to be established through dedicated trials in athlete populations. Finally, validated, low-cost biomarkers capable of detecting subclinical ovulatory disruption in field or team-based settings, without requiring the daily urinary hormone sampling used in research contexts, would meaningfully close the gap of the subclinical underdiagnosis problem.

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