QUALITY IN SPORT

eISSN 2450-3118 · Open Access · Peer-reviewed

apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

Cite as: POLAK, Mateusz, GRANDOS, Jakub, HUSZCZA, Beata, OLEJNIK, Agnieszka, GRONEK, Konrad, KACZMAREK, Julia, GAJEWSKI, Dominik, GŁOWACKA, Zuzanna, KOŚCIOŁEK, Kinga and KOSOWSKI, Mateusz. Beyond the HPG Axis in Relative Energy Deficiency in Sport: Sleep, Psychological Health, and Neuroendocrine Adaptation in Athletes — A Structured Narrative Review. Quality in Sport. 2026;57:72608. https://doi.org/10.12775/QS.2026.57.72608

ARTICLE TIMELINE

Received: 25.05.2026. Revised: 30.05.2026. Accepted: 31.05.2026. Published: 20.06.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines: Medical Sciences; Health Sciences.

Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Nauki medyczne; Nauki o zdrowiu.

Beyond the HPG Axis in Relative Energy Deficiency in Sport: Sleep, Psychological Health, and Neuroendocrine Adaptation in Athletes — A Structured Narrative Review

Mateusz Polak, ORCID https://orcid.org/0009-0003-1904-7044
Email: mat.polak@icloud.com
Provincial Hospital in Poznan – Greater Poland Specialist Center Poland

Jakub Grandos, ORCID https://orcid.org/0009-0002-5859-6532
Email: kuba.grandos@gmail.com
St. John of God Brothers Hospitaller Hospital in Lodz, Poland

Beata Huszcza, ORCID https://orcid.org/0009-0002-2168-5275
Email: beata.huszcza@stud.umed.lodz.pl
Municipal Medical Centre of Karol Jonscher in Lodz, Poland

Agnieszka Olejnik, ORCID https://orcid.org/0009-0002-4180-5270
Email: agnieszkv.olejnik@gmail.com
Independent Public Healthcare Institution MSWiA in Lodz, Poland

Konrad Gronek, ORCID https://orcid.org/0009-0008-7812-5432
Email: konrad.gronek1999@gmail.com
Independent Public Healthcare Institution MSWiA in Lodz, Poland

Julia Kaczmarek, ORCID https://orcid.org/0009-0006-3781-3066
Email: julia.kaczmarek@stud.umed.lodz.pl
Independent Public Healthcare Institution MSWiA in Lodz, Poland

Dominik Gajewski, ORCID https://orcid.org/0009-0003-8611-4518
Email: dominik.gajewski@stud.umed.lodz.pl
St. John of God Brothers Hospitaller Hospital in Lodz, Poland

Zuzanna Głowacka, ORCID https://orcid.org/0009-0006-6594-1444
Email: zglowacka15@gmail.com
Independent Public Healthcare Institution MSWiA in Lodz, Poland

Kinga Kościołek, ORCID https://orcid.org/0009-0000-6235-7513
Email: kinga.kosciolek@stud.umed.lodz.pl
Independent Public Healthcare Institution MSWiA in Lodz, Poland

Mateusz Kosowski, ORCID https://orcid.org/0009-0001-0811-5326
Email: mateusz.kosowski03.08@gmail.com
Independent Public Healthcare Institution in Koło, Poland

* Corresponding author: Mateusz Polak, e-mail: mat.polak@icloud.com

Abstract

Background: Relative Energy Deficiency in Sport (REDs) is increasingly viewed as a multisystem condition related to problematic low energy availability (LEA), but evidence beyond reproductive and skeletal outcomes is uneven.
Objective: To synthesize evidence on sleep, psychological health and neuroendocrine adaptation in athletes with LEA/REDs, while distinguishing established mechanisms from emerging domains.
Methods: A structured narrative review with evidence mapping was conducted in PubMed/MEDLINE; 61 full-text records were assessed and synthesized across predefined domains.
Findings: The strongest support concerns endocrine and metabolic adaptation, including thyroid, adipokine, somatotropic and stress-axis pathways, although individual markers remain non-specific. Sleep and psychological outcomes are clinically relevant but rely mainly on heterogeneous, mostly observational evidence. Direct neurocognitive evidence in athletes with well-characterized LEA/REDs remains insufficient.

Conclusions: REDs should be considered beyond an HPG-centered framework, but claims regarding sleep, psychological health and neurocognition require caution. Future studies should combine objective LEA characterization, longitudinal designs, endocrine profiling, sleep assessment and direct cognitive testing.
Keywords: Relative Energy Deficiency in Sport; REDs; low energy availability; sleep; mental health; neuroendocrine adaptation; athletes; Female Athlete Triad

1. Introduction

Relative Energy Deficiency in Sport (REDs; previously abbreviated RED-S in earlier literature) describes impaired physiological and/or psychological functioning in athletes exposed to problematic low energy availability (LEA). LEA occurs when dietary energy intake is insufficient to support the combined energetic demands of exercise and essential physiological processes. The concept evolved from the Female Athlete Triad, which historically emphasized the interrelationship among LEA, menstrual dysfunction and impaired bone health [1-5].

This historical focus on the HPG axis and skeletal health is scientifically justified. Experimental and clinical Triad research has provided some of the strongest evidence linking energy deficiency with reproductive suppression and bone-related outcomes. However, the Triad and REDs frameworks should be viewed as complementary rather than competing. The Triad remains a robust foundation, whereas REDs extends the conceptual scope to male athletes and broader physiological and psychological domains [1,5,26].

The key unresolved issue is not simply that sleep, psychological health and neuroendocrine adaptation have been addressed separately, but that these domains are supported by unequal types and strengths of evidence. Contemporary consensus statements and endocrine reviews support the concept of problematic LEA as a systemic neuroendocrine and metabolic stressor, yet the evidence varies across organ systems, outcomes, populations and study designs [1,6-8]. No single longitudinal study has simultaneously measured LEA, sleep physiology, neuroendocrine profiling and psychological outcomes in athletes. A credible synthesis must therefore distinguish well-supported endocrine and metabolic adaptations from less established or hypothesis-generating domains.

Recent reviews have begun to address non-reproductive consequences of REDs. Angelidi et al. provided a comprehensive endocrine synthesis of REDs manifestations across multiple axes [6], and the 2023 IOC consensus formally extended the framework beyond the HPG axis to psychological and performance domains [1]. The present review builds on this literature by integrating sleep, psychological health and neuroendocrine adaptation within a single evidence-mapped framework, identifying areas of convergent versus divergent evidence, and explicitly mapping where direct evidence remains insufficient — most notably for neurocognitive outcomes [40,42,48].

Sleep and psychological health are clinically relevant because they may act both as correlates and modifiers of LEA. Sleep integrates recovery, autonomic regulation, immune function, endocrine signaling and psychological resilience. Psychological symptoms, body image concerns, restrictive eating, exercise dependence and sport-specific pressures may contribute to LEA risk, arise during REDs, or reinforce maladaptive behavioral cycles. Therefore, these

domains should be interpreted through a bidirectional systems model rather than a simple downstream-effect model [14-17].

Neurocognitive and neurotransmitter-related pathways are addressed in this review only as emerging considerations. The available full-text evidence base does not yet support strong title-level claims about objective cognitive impairment in athletes with well-characterized LEA/REDs. These domains are therefore discussed as future research priorities, particularly for executive function, attention, working memory, reaction time, decision-making and pacing [29,32,40,42].

This structured narrative review aims to synthesize evidence on sleep-related, psychological and neuroendocrine consequences and correlates of LEA/REDs in athletes, with particular attention to extra-gonadal mechanisms and to the distinction between well-supported, emerging and hypothesis-generating domains. Its central contribution is to integrate sleep, psychological health and neuroendocrine adaptation as interconnected consequences and correlates of problematic LEA, while preserving proportionality between evidence strength and claims.

2. Methods

Review design and search strategy

This article was designed as a structured narrative review with evidence mapping. It was not planned as a formal systematic review or meta-analysis. The aim was to synthesize heterogeneous clinical, mechanistic and framework-oriented evidence on sleep, psychological health and neuroendocrine adaptation in athletes exposed to LEA or REDs. The primary search was conducted in PubMed/MEDLINE on 13 May 2026 using MeSH terms and title/abstract keywords related to REDs/RED-S, LEA, the Female Athlete Triad, athletes or sport, cognition, sleep, psychological health and neuroendocrine mechanisms. No publication-date restriction was applied to the topic. The full PubMed/MEDLINE search strategy is provided in Appendix A.

Although the use of a single primary database limits coverage, this was a deliberate scoping decision for a structured narrative rather than systematic review. To partially offset this limitation, reference lists of included consensus statements and major reviews were checked for additional sources.

Eligibility, screening and full-text assessment

English-language primary research articles, reviews, consensus statements and case reports were eligible if they addressed LEA, REDs or Triad-related energy deficiency in athletes or physically trained populations and contributed to at least one review domain: sleep, psychological health, neuroendocrine/metabolic adaptation, screening/diagnosis, or emerging performance-regulation and neurocognitive considerations. Conference abstracts and dissertations were excluded. The selection process is summarized in Figure 1. The PubMed/MEDLINE search identified 362 records. After title/abstract screening, reclassification of initially misclassified records and functional categorization by review domain, 329 records remained in the evidence map. Of these, 68 records were selected as essential for full-text assessment. Sixty-one full texts were obtained and assessed; seven records could not be retrieved and were not used to support central claims, detailed mechanistic interpretation or clinical implications.

Evidence extraction and synthesis

For each assessed full text, we extracted study design, population, exposure or REDs construct, outcomes, key findings, limitations, likely manuscript use and qualitative citation strength. Evidence was synthesized narratively across predefined domains rather than pooled statistically because studies differed substantially in population, sport type, sex, age, exposure definition, outcome measurement and design. Formal risk-of-bias scoring was not conducted. Instead, each full-text record was assigned a qualitative citation-strength rating during evidence mapping, and claims were interpreted according to evidence type: primary athlete data were prioritized for specific findings, review and consensus sources were used for framework-level interpretation, and mechanistic evidence was presented as biological plausibility rather than proof of clinical causality.

Figure 1. Literature-selection workflow.

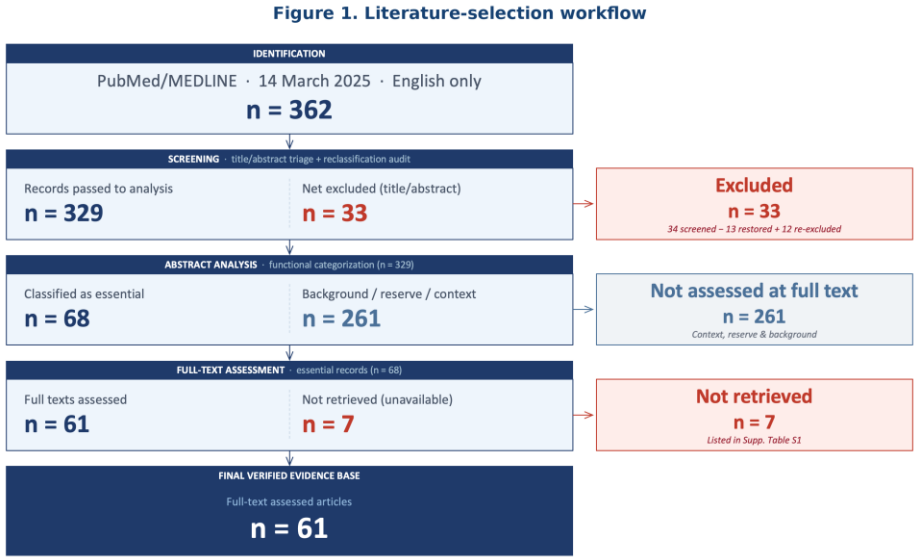


Table 1. Key terms and interpretative framework used in this review.

Term	Working definition in this review	Interpretative note
Energy availability (EA)	Dietary energy intake minus exercise energy expenditure, normalized to fat-free mass [38,39].	Used as a research construct; measurement error is common in free-living athletes.
Low energy availability (LEA)	A state in which energy available for physiological function is reduced after exercise expenditure [38,39].	Not all short-term LEA is necessarily pathological.
Problematic LEA	LEA that is prolonged and/or severe enough to impair physiological or psychological functioning [1].	Main exposure of interest in REDs.
Relative Energy Deficiency in Sport (REDs)	A clinical framework describing health and performance consequences related to problematic LEA [1,26].	This manuscript uses REDs, consistent with the 2023 IOC consensus terminology.
Female Athlete Triad	Historical framework linking LEA, menstrual dysfunction and impaired bone health [2-4].	Treated as complementary to REDs, not obsolete.

Term	Working definition in this review	Interpretative note
Evidence-mapped synthesis	Narrative synthesis in which sources are categorized by domain, evidence type, relevance and citation strength.	Used here instead of formal risk-of-bias scoring.

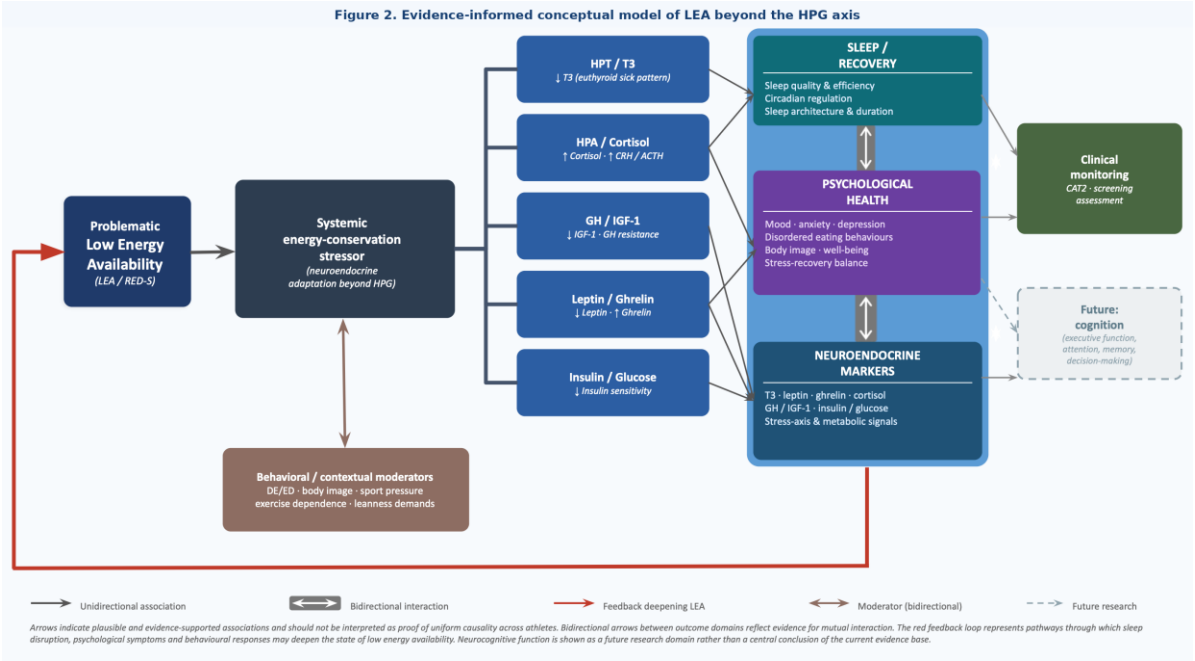
3. LEA as a systemic stressor: conceptual framework

The core argument flow of this review is: LEA may act as a systemic stressor; systemic stress can induce endocrine and metabolic adaptation; these adaptations may interact with sleep and psychological health; and the clinical implication is that REDs assessment should be multidisciplinary and context-sensitive. This argument flow is deliberately cautious: it is a systems framework, not a claim that LEA uniformly causes sleep disturbance or mental health symptoms in all athletes.

Problematic LEA differs from short-term or adaptable reductions in energy availability. Contemporary REDs frameworks distinguish between adaptable LEA, which may be brief and reversible, and problematic LEA, which is prolonged and/or severe enough to impair physiological or psychological functioning [1]. The threshold concept of energy availability — originally defined as approximately 30 kcal/kg fat-free mass/day for reproductive disruption in women — provides a quantitative framework for operationalizing LEA in research and clinical contexts [38,39]. This distinction is important for athletes because short-term body-composition manipulation or intensified training may not have the same risk profile as chronic underfueling [52,60].

From a mechanistic perspective, endocrine responses to LEA appear to involve coordinated energy-conservation pathways, including thyroid, adipokine, somatotropic and stress-axis signaling, rather than isolated HPG suppression alone [6-9]. These pathways provide biological plausibility for links among LEA, sleep, mood, recovery and performance regulation, but they do not establish uniform causality [41,53,58-60,62].

Figure 2. Evidence-informed conceptual model of LEA as a systemic neuroendocrine stressor beyond the HPG axis.



The figure depicts problematic LEA/REDs as an upstream exposure that may interact with coordinated neuroendocrine adaptation pathways beyond the HPG axis and with three interconnected outcome domains: sleep/recovery, psychological health and neuroendocrine markers. Bidirectional arrows reflect plausible mutual interaction, whereas the feedback loop represents pathways through which sleep disruption, psychological symptoms and behavioral responses may deepen LEA. Neurocognitive function is shown as a future research domain rather than a central conclusion. Arrows indicate plausible, evidence-informed associations and should not be interpreted as proof of uniform causality across athletes.

4. Neuroendocrine and metabolic adaptation beyond the HPG axis

Contemporary endocrine reviews describe REDs as involving multiple neuroendocrine axes beyond the gonadal axis, including thyroid, adrenal and somatotropic pathways, as well as adipokines and metabolic fuels [6-8]. This evidence supports a coordinated neuroendocrine adaptation model, provided the manuscript distinguishes candidate markers from validated diagnostic tests.

The HPT axis is among the most clinically relevant pathways beyond the HPG axis. Reduced triiodothyronine (T3) has been reported in several LEA contexts and may reflect energy-conserving metabolic adaptation rather than primary thyroid disease [6-9,20,41,53,58,59,62]. However, T3 is influenced by acute energy intake, training load, illness, menstrual status and assay timing. It should therefore be interpreted as a contextual marker rather than a standalone diagnostic indicator.

Leptin, ghrelin, insulin and glucose-related pathways provide another mechanistic layer. Leptin is responsive to energy availability and adipose tissue status and may signal energy sufficiency to central neuroendocrine systems [6,7]. Ghrelin and other appetite-regulating pathways may also be relevant. However, their clinical role in REDs diagnosis or treatment remains investigational.

The GH/IGF-1 axis and bone-related markers link energy deficiency to growth, tissue repair and skeletal adaptation. Endocrine reviews and athlete biomarker studies support the relevance of IGF-1 and related anabolic pathways, but responses vary by sex, sport, energy-deficit duration and measurement context [6-9,18-24,34,41,53,58,60,62].

The HPA axis and cortisol require particularly cautious wording. Cortisol may reflect training stress, psychological stress, energy deficiency, sleep disruption, illness or sampling conditions. Thus, cortisol should not be interpreted as a specific REDs marker. Its value is mainly conceptual: it illustrates how energy deficiency, stress physiology and recovery processes may overlap.

Table 2. Neuroendocrine pathways beyond the HPG axis relevant to REDs.

Pathway/domain	Candidate markers or constructs	Evidence type in this review	Main interpretation	Key caveat
HPT/thyroid axis	T3, thyroid-hormone adaptation	Primary athlete data + endocrine reviews [6-9,20,41,53,58,59,62]	Reduced T3 may reflect energy-conserving metabolic	T3 is non-specific and influenced by acute intake,

Pathway/domain	Candidate markers or constructs	Evidence type in this review	Main interpretation	Key caveat
			adaptation in LEA/REDs contexts.	illness, training load and assay timing.
Adipokine/appetite signaling	Leptin, ghrelin, insulin/glucose	Mechanistic reviews + experimental/observational evidence	Leptin and related metabolic signals provide biological plausibility linking energy stores with central endocrine adaptation.	Not validated as diagnostic markers; ghrelin/leptin patterns vary by population and timing.
GH/IGF-1 and tissue repair	GH, IGF-1, bone turnover markers	Mechanistic reviews + athlete biomarker studies [6-9,18-24,34,41,53,58,60,62]	Somatotropic adaptations may link LEA with growth, repair and skeletal consequences.	Effects are heterogeneous and overlap with training stress and maturation.
HPA/stress axis	Cortisol, ACTH-cortisol signaling, stress-recovery state	Review-level and observational evidence	HPA-axis activity may interact with energy deficiency, sleep and psychological stress.	Cortisol is highly context-sensitive and cannot be interpreted as LEA-specific.
HPG overlap	Menstrual function, testosterone, estradiol, libido	Strong Triad/REDs framework + primary data [2-4,26]	HPG suppression remains a central established pathway but	The review focus is beyond HPG; HPG evidence should be

Pathway/domain	Candidate markers or constructs	Evidence type in this review	Main interpretation	Key caveat
			is not the only endocrine domain relevant to REDs.	used as foundation rather than entire model.
Neurotransmitter-related pathways	Serotonin, dopamine, reward/motivation pathways	Indirect and hypothesis-generating only	Potentially relevant to mood, motivation and performance regulation.	Current REDs-specific athlete evidence is insufficient for strong pathway claims.

5. Sleep-related consequences of LEA and REDs

The available evidence on sleep in LEA/REDs converges on one moderately supported association: lower energy availability tends to coincide with poorer subjective sleep quality, whereas findings on sleep architecture and circadian regulation remain inconsistent and methodologically heterogeneous. Sleep is biologically plausible as both an outcome and mediator in REDs because it integrates recovery, autonomic regulation, endocrine signaling, immune function and psychological resilience.

Evidence from athlete cohorts using objective or semi-objective sleep assessment suggests that lower energy availability or LEA risk may be associated with poorer sleep quality, altered sleep architecture or greater sleep debt in some samples [10-13,47,54,55]. However, evidence strength varies substantially with the measurement approach. Studies using objective methods such as portable polysomnography or actigraphy report the most concrete sleep-architecture changes but are limited by small samples and short observation windows [11,13]. Questionnaire-based studies provide larger cohorts but capture predominantly subjective sleep quality and are susceptible to recall and reporting bias [12,47,54].

This methodological heterogeneity — combined with the sensitivity of sleep to training load, competition timing, travel, injury, illness, caffeine use, mood and dietary composition — means that any observed association between LEA and sleep outcomes should be interpreted as associative rather than causal. Broader systems-level data reinforce this complexity: Olympic cohort findings showed interdependence among LEA, sleep quality, mental health symptoms, stress-recovery and illness, and REDs consensus and mental health reviews position sleep within a wider athlete-health context [1,14,17].

Three patterns emerge from the synthesis. First, the association between lower energy availability and poorer subjective sleep quality is the most consistent signal across populations

and designs. Second, effects on sleep architecture and circadian regulation are heterogeneous and understudied, with no convergent direction across studies. Third, the directionality of the sleep-LEA relationship is unresolved: sleep disruption may be a consequence of LEA, a contributor to it through hormonal dysregulation, or both. Taken together, these patterns support treating sleep as an interacting recovery and regulatory domain within REDs rather than a proven downstream consequence of LEA. Future studies should combine objective sleep assessment with prospective LEA characterization and endocrine profiling to clarify directionality.

6. Psychological health and behavioral pathways

Evidence on psychological health in LEA/REDs consistently points to bidirectional associations rather than a linear downstream effect. Anxiety, depressive symptoms, body image concerns and disordered eating may co-occur with LEA, but the strongest empirical signals come from cross-sectional designs that cannot establish causal direction. The IOC mental health subgroup review describes early psychological indicators associated with problematic LEA, including mood changes, fatigue and psychological conflict, and more severe outcomes such as reduced well-being, elevated anxiety, depressive symptoms and eating disorders [14]. This framing supports integrating psychological assessment into REDs screening.

Two interrelated outcome clusters emerge from the primary athlete literature. The first concerns anxiety and depressive symptoms: Olson et al. reported that Triad risk score was associated with moderate and severe depression and anxiety symptoms in women, with the strongest links to low EA and disordered eating/eating disorder components [15], and additional cohort data link LEA risk with elevated anxiety, depressive symptoms or impaired stress-recovery [17,43,44,49,55]. The second cluster concerns body image and eating behaviors: LEA risk co-occurs with body dissatisfaction, disordered eating constructs and exercise dependence in collegiate, endurance and dance contexts [16,23,48]. Separating these clusters clarifies where the evidence is strongest and where it remains diffuse.

These findings should not be read as evidence that LEA directly causes depression or anxiety. Restrictive eating, body image concerns, exercise dependence, pressure for leanness and excessive training can contribute to LEA, while LEA and its downstream health consequences may in turn worsen mood, fatigue and stress responses. The most defensible interpretation is therefore a bidirectional reinforcement model in which affective symptoms, disordered eating constructs and LEA may mutually sustain one another.

Psychological screening, body image evaluation, assessment of eating behaviors and exercise dependence, and monitoring of stress-recovery status should be integrated into an interdisciplinary workup. Age-, sex- and sport-specific contexts remain important — particularly in adolescent athletes, male endurance athletes, aesthetic and dance disciplines, ultra-endurance sports, tactical athlete populations and team sports [33-36,46,47,54,56,61].

7. Integrative synthesis: how sleep, psychological health and neuroendocrine adaptation interconnect

The three domains reviewed above are not independent. Sleep, psychological health and neuroendocrine adaptation interact bidirectionally, and understanding their crosstalk is essential for conceptualizing problematic LEA as a systemic stressor rather than a collection of isolated deficits.

Neuroendocrine pathways provide the mechanistic backbone linking LEA to sleep and psychological outcomes. Cortisol-related stress-axis activity may plausibly contribute to sleep disruption, but this should be interpreted as mechanistic plausibility rather than direct REDs-specific causal evidence [6,8,58,60]. Reduced triiodothyronine, which accompanies energy-conserving metabolic adaptation in LEA, is associated with fatigue, mood disturbance and impaired recovery — all of which may worsen sleep quality and reinforce psychological symptoms [6,7]. Leptin, responsive to energy availability and adipose signaling, influences reproductive neuroendocrine function and may also relate to mood and central satiety signaling; however, REDs-specific athlete evidence remains insufficient for strong pathway claims [6].

Sleep, in turn, modulates neuroendocrine function and psychological resilience. Athlete cohort data support co-occurrence among LEA risk, poorer sleep quality, psychological symptoms and illness burden, but they do not directly establish sleep-restriction physiology as a REDs mechanism [17]. Mechanistic overlap between sleep, stress-axis activity and metabolic signaling remains biologically plausible and should be interpreted as contextual rather than direct causal evidence [58,60]. Together, these data support a plausible bidirectional model in which LEA risk, sleep disruption and stress-axis activation may reinforce one another, while remaining insufficient to prove a uniform causal sequence in athletes.

Psychological health intersects with both neuroendocrine and sleep domains through behavioral and physiological pathways. Anxiety and depressive symptoms may reduce sleep quality through increased arousal and rumination, while disordered eating behaviors and body image concerns may contribute to LEA. Conversely, the fatigue and mood disturbance that accompany chronic energy deficiency may reinforce restrictive eating, exercise compulsion and avoidance of help-seeking. These links are best interpreted as interacting pathways rather than a single linear causal chain.

Importantly, evidence for these connections is unevenly distributed across domains. Neuroendocrine mechanisms are supported by primary biomarker and review-level evidence. Sleep effects are supported by cohort data with limited causal inference. Psychological interactions are supported by associative evidence and theoretical models derived from related clinical populations. The absence of longitudinal studies simultaneously measuring LEA, sleep physiology, neuroendocrine profiles and psychological outcomes in athletes represents both a major limitation and a productive target for future research.

8. Emerging considerations: performance regulation and neurocognition

Performance regulation and neurocognition remain emerging rather than established REDs domains. The available corpus contains plausible mechanisms and indirect performance-regulation evidence, but few direct studies using objective cognitive testing in athletes with well-characterized LEA or REDs [40,42]. Indirect evidence relevant to cognitive restraint, performance regulation or iron-related pathways was retained as contextual support rather than central evidence [29,32].

Performance claims also require caution. A systematic review and meta-analysis on overreaching and LEA markers found that underperforming athletes may present with markers of LEA, but underperformance can also occur in the absence of LEA, and LEA markers do not uniformly explain performance outcomes [30,31]. A male combat-sport case report further

suggests that severity, duration and patterning of LEA matter, but single-case data cannot define generalizable thresholds [18].

The safest conclusion is that performance regulation and neurocognition should be discussed as emerging considerations. Future studies should directly assess executive function, attention, working memory, reaction time, decision-making and pacing while objectively characterizing LEA/REDs and controlling for sleep, mood, training load, iron status and illness.

Table 3. Domain-level evidence synthesis.

Evidence domain	Main sources/evidence type	Overall support	Safe synthesis statement	Manuscript role
Framework and clinical assessment	IOC RED-S/REDs consensus; Triad consensus; CAT2 application study; critical reviews [1-5,26,27]	Strong for framework; moderate for clinical validation	REDs is best framed as problematic LEA with multisystem consequences, but evidence strength varies by domain.	Introduction, Methods rationale, screening/diagnosis.
Neuroendocrine adaptation	Endocrine reviews; biomarker studies; short-term LEA studies; male athlete evidence [6-9,18-24,34,41,53,58-60,62]	Moderate-to-strong	LEA is associated with coordinated endocrine/metabolic adaptation beyond isolated HPG suppression.	Mechanistic backbone and conceptual figure.
Sleep and recovery	Objective sleep studies, athlete cohorts, tactical-athlete data, recovery/illness evidence, consensus context [10-13,17,47,54,55]	Moderate	LEA risk may be associated with poorer sleep outcomes, but causal direction remains uncertain.	Sleep section and research priorities.
Psychological health	IOC mental health review; anxiety/depression/Triad studies; body image and DE/ED studies [14-17,23,43,44,48,49,55]	Moderate	Psychological symptoms, DE/ED constructs and LEA-related risk factors appear bidirectionally interrelated.	Psychological health section and clinical interpretation.

Evidence domain	Main sources/evidence type	Overall support	Safe synthesis statement	Manuscript role
Performance regulation	Overreaching/LEA markers review; conceptual performance-regulation evidence; case data [18,25,29-31,40,60]	Mixed	Performance effects are context-dependent and should not be interpreted as uniform or deterministic.	Cautionary discussion and future directions.
Neurocognition	Indirect conceptual evidence; cognition-focused reviews and missing essential cognitive papers not assessed at full text [29,32,40,42]	Low/insufficient	Direct neurocognitive effects remain insufficiently tested and should remain a future research gap.	Short subsection and future directions, not title-level claim.

9. Clinical and practical implications, screening and diagnosis

The clinical framework domain is well supported by contemporary consensus and validation work. The historical Triad position stand and 2014 Triad consensus provide the foundation for screening, treatment and return-to-play decision-making in female athletes [2-4]. The 2014 IOC REDs consensus broadened this framework to male and female athletes and to multiple physiological systems [26]. The 2023 IOC REDs consensus further refined definitions, introduced adaptable versus problematic LEA, and updated clinical assessment recommendations [1]. Updated Triad guidance and sport-specific consensus work further emphasize the need to adapt assessment to age, sex and sport context [52,56].

The IOC REDs Clinical Assessment Tool version 2 (CAT2) is the most relevant contemporary clinical framework for severity and risk stratification. Its application across more than 200 elite athletes supports its clinical relevance, showing associations between higher-risk categories and indicators such as amenorrhea, bone stress injury history, lower T3, lower testosterone and lower BMD [27]. Nevertheless, CAT2 should be interpreted as structured risk stratification rather than a standalone diagnostic test.

Screening tools such as LEAF-Q and LEAM-Q are useful for triage but have limitations. Questionnaire-based screening may identify athletes requiring further assessment [28,50], but REDs remains a clinical diagnosis requiring history, examination, laboratory interpretation, sport context and exclusion of alternative causes. Implementation also requires education of clinicians and school- or sport-health personnel; survey data from public health nurses working with elite sport schools suggest that knowledge of REDs/LEA may remain limited even among professionals in athlete-support settings [63]. Endocrine markers may support interpretation but should not be used in isolation.

For clinicians and sport-health teams, the practical implication is not to screen every athlete with an exhaustive endocrine panel, but to broaden the clinical interview and monitoring framework. Sleep quality, recovery, mood, body image, eating behaviors, exercise dependence, injury history, menstrual or libido changes, bone stress injury history and training-load context should be considered together. This integrated approach is particularly important when athletes present with recurrent illness, fatigue, unexplained performance decline, bone stress injury, menstrual dysfunction or psychological distress.

10. Limitations and future directions

This review has several limitations. First, although the workflow was structured and auditable, it was not a formal systematic review. The search was primarily PubMed/MEDLINE-based and may have missed relevant studies indexed only in sport science, psychology, nutrition or sleep databases, such as SPORTDiscus, PsycINFO, Scopus or Web of Science. Grey literature and trial registries were not systematically searched, and formal risk-of-bias scoring was not conducted.

Second, the evidence base is heterogeneous. Included studies varied in population, sex, age, sport type, training level, LEA definition, REDs framework, assessment tool and outcome measure. Many studies relied on self-reported dietary intake, self-reported sleep or questionnaire-based LEA risk rather than objective energy-availability measurement.

Third, causal inference is limited. Much of the psychological and sleep-related evidence is cross-sectional, and many associations are vulnerable to confounding by disordered eating, body dissatisfaction, injury, illness, training load, competition timing and stress-recovery state.

Fourth, several mechanistic pathways are supported by plausible physiology and review-level synthesis but remain incompletely established in free-living athletes. This is particularly true for neurotransmitter-related mechanisms and direct neurocognitive outcomes.

Fifth, the evidence base disproportionately represents female athletes and endurance or aesthetic sports. Male athletes, team-sport athletes, power athletes and recreational populations are underrepresented, which limits the generalizability of conclusions across sex, sport type and training level.

Future work should use longitudinal and experimental designs that define LEA severity, duration, frequency and recovery periods; integrate objective sleep assessment with energy availability and endocrine measures; assess psychological pathways using validated tools; and directly test executive function, attention, working memory, reaction time, decision-making and pacing in athletes with objectively characterized LEA/REDs.

Future studies should also validate sex-specific and sport-specific thresholds and screening tools, distinguish adaptable short-term LEA from prolonged or severe problematic LEA, and evaluate interdisciplinary interventions that combine nutrition, training modification, psychological care, sleep optimization and return-to-play decision-making [45,51,52]. Digital or virtual-reality approaches remain speculative and should be evaluated only as hypothesis-generating adjuncts; current publications in this area are preliminary communications rather than controlled trials [37].

Measurement development is also needed. Feasible endocrine-marker approaches, including minimally invasive or capillary blood methods, require validation before being applied as clinical REDs tools [57]. Biomarker panels should be evaluated against clinically meaningful

outcomes and structured risk/severity tools rather than interpreted as standalone diagnostic tests [57,58].

Table 4. Evidence limitations and future research priorities.

Claim area	Risk of overclaiming	Allowed wording	Avoid wording	Future direction/action
Sleep	Moderate-high	LEA risk has been associated with poorer sleep outcomes in some athlete cohorts.	LEA causes sleep disruption.	Combine objective sleep assessment with EA, endocrine and recovery measures.
Mental health	High	Psychological symptoms, DE/ED and LEA-related risk factors may interact bidirectionally.	LEA causes depression or anxiety.	Use longitudinal designs controlling for DE/ED, body image, injury and training load.
Endocrine biomarkers	High	Markers such as T3, leptin, IGF-1 and cortisol may support contextual assessment.	These markers diagnose REDs.	Validate marker panels and measurement methods against clinically meaningful outcomes and CAT2 categories [57,58].
Performance	High	Performance implications appear context-dependent and may vary by LEA severity and duration.	LEA always impairs performance.	Separate adaptable short-term LEA from prolonged/severe problematic LEA [30,31].
Neurocognition	High	Direct neurocognitive outcomes	LEA impairs cognition in athletes.	Test executive function, attention,

Claim area	Risk of overclaiming	Allowed wording	Avoid wording	Future direction/action
		remain insufficiently tested.		working memory, reaction time and decision-making in characterized LEA/REDs [29,32].
Serotonin/dopamine	High	Neurotransmitter-related pathways remain plausible but insufficiently characterized.	REDs dysregulates serotonin/dopamine.	Only discuss as hypothesis-generating unless direct REDs evidence becomes available.
Clinical tools	Moderate	CAT2 supports structured risk/severity stratification within broader clinical assessment.	CAT2 definitively diagnoses REDs.	Evaluate CAT2 prospectively across sex, sport and performance-level strata [27,28].

11. Conclusions

Problematic low energy availability in athletes should not be conceptualized as a primarily reproductive or skeletal exposure. Current evidence supports a broader model in which LEA acts as a systemic neuroendocrine and behavioral stressor, with evidence strength differing substantially across domains.

Endocrine and metabolic adaptations provide the strongest mechanistic foundation for this broader view. Sleep and psychological health are clinically relevant and increasingly supported, but much of the evidence remains observational and vulnerable to confounding. Neurocognitive outcomes and neurotransmitter-specific mechanisms remain important future research areas rather than established conclusions.

The most defensible conclusion is therefore cautious: REDs should not be understood solely as a reproductive or skeletal health problem, but neither should every proposed REDs consequence be treated as equally established. The value of this review lies in integrating sleep, psychological health and neuroendocrine adaptation within a single evidence-informed framework while preserving proportionality between evidence and claims. Future work should prioritize prospective designs integrating objective LEA characterization, sleep physiology, psychological assessment, endocrine profiling and direct cognitive testing.

Supplementary Materials

The supplementary material is included within this manuscript as Appendix A. Appendix A provides the complete PubMed/MEDLINE search strategy.

Author Contributions

Conceptualization, M.P. and J.G.; methodology, M.P., J.G. and B.H.; literature screening and evidence mapping, M.P., J.G., B.H., A.O. and K.G.; writing - original draft preparation, M.P., J.G., B.H., A.O., K.G., J.K., D.G., Z.G., K.K. and M.K.; writing - review and editing, M.P., J.G., B.H., A.O., K.G., J.K., D.G., Z.G., K.K. and M.K.; visualization, M.P., J.G. and D.G.; supervision, M.P. All authors have read and agreed to the submitted version of the manuscript.

Funding

This research received no external funding.

Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

No new primary dataset was generated for this review. The PubMed/MEDLINE search strategy is provided in Appendix A.

Acknowledgements

Not applicable.

Conflicts of Interest

The authors declare no conflict of interest.

Declaration of use of artificial intelligence

In preparing this work, the authors used Claude (Anthropic) for the purpose of supporting language editing, manuscript organization, formatting assistance, linguistic flow, and bibliographic consistency checks. After using this tool/service, the author(s) have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

References

1. Mountjoy M, Ackerman KE, Bailey DM, Burke LM, Constantini N, Hackney AC, et al. 2023 International Olympic Committee's consensus statement on Relative Energy Deficiency in Sport (REDs). Br J Sports Med. 2023;57:1073-1097. doi: 10.1136/bjsports-2023-106994.

2. Nattiv A, Loucks AB, Manore MM, Sanborn CF, Sundgot-Borgen J, Warren MP. The Female Athlete Triad. Med Sci Sports Exerc. 2007;39:1867-1882. doi: 10.1249/mss.0b013e318149f111.

3. De Souza MJ, Nattiv A, Joy E, Misra M, Williams NI, Mallinson RJ, et al. 2014 Female Athlete Triad Coalition Consensus Statement on treatment and return to play of the Female Athlete Triad. Br J Sports Med. 2014;48:289. doi: 10.1136/bjsports-2013-093218.
4. Joy E, De Souza MJ, Nattiv A, Misra M, Williams NI, Mallinson RJ, et al. 2014 Female Athlete Triad Coalition Consensus Statement on treatment and return to play of the Female Athlete Triad. Curr Sports Med Rep. 2014;13:219-232. doi: 10.1249/JSR.0000000000000078.
5. De Souza MJ, Strock NCA, Ricker EA, Koltun KJ, Barrack M, Joy E, et al. The path towards progress: a critical review to advance the science of the Female and Male Athlete Triad and Relative Energy Deficiency in Sport. Sports Med. 2022;52:13-23. doi: 10.1007/s40279-021-01568-w.
6. Angelidi AM, Stefanakis K, Chou SH, Valenzuela-Vallejo L, Dipla K, Boutari C, et al. Relative Energy Deficiency in Sport (REDs): endocrine manifestations, pathophysiology and treatments. Endocr Rev. 2024;45:676-708. doi: 10.1210/endrev/bnae011.
7. Elliott-Sale KJ, Tenforde AS, Parziale AL, Holtzman B, Ackerman KE. Endocrine effects of Relative Energy Deficiency in Sport. Int J Sport Nutr Exerc Metab. 2018;28(4):335-349. doi: 10.1123/ijsnem.2018-0127.
8. Dipla K, Kraemer RR, Constantini NW, Hackney AC. Relative energy deficiency in sports (RED-S): elucidation of endocrine changes affecting the health of males and females. Hormones (Athens). 2021;20(1):35-47. doi: 10.1007/s42000-020-00214-w.
9. Guisado-Cuadrado I, Recacha-Ponce P, Peinado AB, Romero-Parra N. Biochemical responses to experimentally induced short-term low energy availability in athletes: a systematic review. Scand J Med Sci Sports. 2026;36(3):e70249. doi: 10.1111/sms.70249.
10. Lundstrom EA, De Souza MJ, Conklin ME, Williams NI. Association between energy availability and sleep quality in elite female and male swimmers: a brief report. J Int Soc Sports Nutr. 2026;23:2665547. doi: 10.1080/15502783.2026.2665547.
11. Saidi O, Souabni M, Aloui A, Selmi O, Masmoudi L, Davenne D, et al. Association between low energy availability (LEA) and impaired sleep quality in young rugby players. Nutrients. 2024;16(5):609. doi: 10.3390/nu16050609.
12. Saifi A, Khanna GL, Kalpana K. Risk for low energy availability, disordered eating and sleep disturbance among female football players. Phys Act Nutr. 2024;28(3):43-51. doi: 10.20463/pan.2024.0022.
13. Thomas CT, Langan-Evans C, Germaine MG, Artukovic M, Jones H, Whitworth-Turner C, Close GL, Louis J. Case report: effect of low energy availability and training load on sleep in a male combat sport athlete. Front Sports Act Living. 2023;4:981755. doi: 10.3389/fspor.2022.981755.
14. Pensgaard AM, Sundgot-Borgen J, Edwards C, Jacobsen AU, Mountjoy M. Intersection of mental health issues and Relative Energy Deficiency in Sport (REDs): a narrative review by a subgroup of the IOC consensus on REDs. Br J Sports Med. 2023;57:1127-1135. doi: 10.1136/bjsports-2023-106867.
15. Olson EM, Miro E, Roche M, Mehta S, Sainani K, Kraus E. Mental Health Matters: a cross-sectional survey on depression and anxiety symptoms and the Female and Male Athlete Triad. Clin J Sport Med. 2023;33:368-375. doi: 10.1097/JSM.0000000000001130.

16. Fields JB, Jones MT, Kitts SE, King EL, Jagim AR. Relationships between disordered eating, body dissatisfaction, and low energy availability in women collegiate athletes. J Strength Cond Res. 2026;40:e164-e171. doi: 10.1519/JSC.0000000000005281.
17. Drew M, Vlahovich N, Hughes D, Appaneal R, Burke LM, Lundy B, et al. Prevalence of illness, poor mental health and sleep quality and low energy availability prior to the 2016 Summer Olympic Games. Br J Sports Med. 2018;52(1):47-53. doi: 10.1136/bjsports-2017-098208.
18. Langan-Evans C, Germaine M, Artukovic M, Oxborough DL, Areta JL, Close GL, Morton JP. The psychological and physiological consequences of low energy availability in a male combat sport athlete. Med Sci Sports Exerc. 2021;53:673-683. doi: 10.1249/MSS.0000000000002519.
19. Mikkonen R, Hackney AC, Hulmi J, Isola V, Ahtiainen J, Ihalainen J. Hormone profiles after planned low energy availability exposure in naturally menstruating and hormonal contraceptive using physique athletes. Eur J Sport Sci. 2025;25(12):e70076. doi: 10.1002/ejsc.70076.
20. Black K, Slater J, Brown RC, Cooke R. Low energy availability, plasma lipids, and hormonal profiles of recreational athletes. J Strength Cond Res. 2018;32(10):2816-2824. doi: 10.1519/JSC.0000000000002540.
21. Lee S, Kuniko M, Han S, Oh T, Taguchi M. Association of low energy availability and suppressed metabolic status in Korean male collegiate soccer players: a pilot study. Am J Mens Health. 2020;14(6):1557988320982186. doi: 10.1177/1557988320982186.
22. McGuire A, Warrington G, Walsh A, Byrne T, Doyle L. Measurement of energy availability in highly trained male endurance athletes and examination of its associations with bone health and endocrine function. Eur J Nutr. 2024;63(7):2655-2665. doi: 10.1007/s00394-024-03433-8.
23. Torstveit MK, Fahrenholtz IL, Lichtenstein MB, Stenqvist TB, Melin AK. Exercise dependence, eating disorder symptoms and biomarkers of Relative Energy Deficiency in Sports (RED-S) among male endurance athletes. BMJ Open Sport Exerc Med. 2019;5(1):e000439. doi: 10.1136/bmjsem-2018-000439.
24. Koehler K, Achtzehn S, Braun H, Mester J, Schaenzer W. Comparison of self-reported energy availability and metabolic hormones to assess adequacy of dietary energy intake in young elite athletes. Appl Physiol Nutr Metab. 2013;38(7):725-733. doi: 10.1139/apnm-2012-0373.
25. Fagerberg P. Negative consequences of low energy availability in natural male bodybuilding: a review. Int J Sport Nutr Exerc Metab. 2018;28(4):385-402. doi: 10.1123/ijsnem.2016-0332.
26. Mountjoy M, Sundgot-Borgen J, Burke L, Carter S, Constantini N, Lebrun C, et al. The IOC consensus statement: beyond the Female Athlete Triad - Relative Energy Deficiency in Sport (RED-S). Br J Sports Med. 2014;48:491-497. doi: 10.1136/bjsports-2014-093502.
27. Heikura IA, McCluskey WTP, Tsai MC, Johnson L, Murray H, Mountjoy M, et al. Application of the IOC Relative Energy Deficiency in Sport (REDs) Clinical Assessment Tool version 2 (CAT2) across 200+ elite athletes. Br J Sports Med. 2025;59:24-35. doi: 10.1136/bjsports-2024-108592.

28. Lundy B, Torstveit MK, Stenqvist TB, Burke LM, Garthe I, Slater GJ, Ritz C, Melin AK. Screening for low energy availability in male athletes: attempted validation of LEAM-Q. Nutrients. 2022;14(9):1873. doi: 10.3390/nu14091873.
29. Jurov I, Keay N, Hadzic V, Spudic D, Rauter S. Relationship between energy availability, energy conservation and cognitive restraint with performance measures in male endurance athletes. J Int Soc Sports Nutr. 2021;18:24. doi: 10.1186/s12970-021-00419-3.
30. Kuikman MA, Coates AM, Burr JF. Markers of low energy availability in overreached athletes: a systematic review and meta-analysis. Sports Med. 2022;52(12):2925-2941. doi: 10.1007/s40279-022-01723-x.
31. Logue D, Madigan SM, Delahunt E, Heinen M, Mc Donnell SJ, Corish CA. Low energy availability in athletes: a review of prevalence, dietary patterns, physiological health, and sports performance. Sports Med. 2018;48(1):73-96. doi: 10.1007/s40279-017-0790-3.
32. Petkus DL, Murray-Kolb LE, De Souza MJ. The unexplored crossroads of the Female Athlete Triad and iron deficiency: a narrative review. Sports Med. 2017;47:1721-1737. doi: 10.1007/s40279-017-0706-2.
33. Gould RJ, Ridout AJ, Newton JL. Relative Energy Deficiency in Sport (RED-S) in adolescents - a practical review. Int J Sports Med. 2023;44:236-246. doi: 10.1055/a-1960-2839.
34. Schofield KL, Thorpe H, Sims ST. Where are all the men? Low energy availability in male cyclists: a review. Eur J Sport Sci. 2021;21(11):1567-1578. doi: 10.1080/17461391.2020.1842510.
35. Robertson S, Mountjoy M. A review of prevention, diagnosis and treatment of Relative Energy Deficiency in Sport (RED-S) in artistic (synchronized) swimming. Int J Sport Nutr Exerc Metab. 2018;28(4):375-384. doi: 10.1123/ijsnem.2017-0329.
36. Billingham T, Comyns TM, Norton C, Warrington G, Sweeney G, Purtill H, Kenny IC. Relative energy deficiency in sport (REDs) in elite adult team ball sport athletes: a systematic review. J Sci Med Sport. 2026;29(5):342-350. doi: 10.1016/j.jsams.2025.12.001.
37. Herawati AA, Yusuf SLN, Ilfiandra, Irawan AW, Habibi ASY. Enhancing athlete well-being with RED-S: the potential role of virtual reality for stress management [Letter]. J Pediatr Adolesc Gynecol. 2025;38:769-770. doi: 10.1016/j.jpag.2025.09.006.
38. Loucks AB, Kiens B, Wright HH. Energy availability in athletes. J Sports Sci. 2011;29(Suppl 1):S7-S15. doi: 10.1080/02640414.2011.588958.
39. Loucks AB, Thuma JR. Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. J Clin Endocrinol Metab. 2003;88(1):297-311. doi: 10.1210/jc.2002-020369.
40. Grivas GV, Alkasasbeh WJ. Energy Availability as a Neurocognitive Regulator of Endurance Performance: Integrating Metabolic, Perceptual, and Decision-Making Mechanisms-A Narrative Review. Sports. 2026;14(4):150. doi: 10.3390/sports14040150.
41. Woronyczová J, Nováková M, Gerych D, Jurková K, Oberman Č, Bolek E, Pilný J, Vítek L. Relative energy deficiency in sport: a cross-sectional study of nutritional, biochemical and hormonal profiles in Czech female endurance athletes at risk of low energy availability. Front Sports Act Living. 2026;8:1663582. doi: 10.3389/fspor.2026.1663582.
42. Subalatha M, Rachaveti D, Amutha S, Ponpandi M, et al. A narrative review on the role of cognition, nutrition and energy availability in athletes of competitive sports to combat RED-S. PeerJ. 2025;13:e18849. doi: 10.7717/peerj.18849.

43. Scheid JL, Basile S, West SL. Low Energy Availability Risk Is Associated with Anxiety in Female Collegiate Athletes. Sports. 2024;12(10):269. doi: 10.3390/sports12100269.
44. Halioua R, Wasserfurth P, Toepffer D, Claussen MC, Koehler K. Exploring the relationship between low energy availability, depression and eating disorders in female athletes: a cross-sectional study. BMJ Open Sport Exerc Med. 2024;10(3):e002035. doi: 10.1136/bmjsem-2024-002035.
45. Rudin R, Harris L, White H, Hammond L. Exploring different interventions for Relative Energy Deficiency in Sport (REDs): A systematic review. JSAMS Plus. 2025;5:100085. doi: 10.1016/j.jsampl.2024.100085.
46. Colangelo J, Smith A, Henninger K, Buadze A, Liebrenz M. Exploring the presentation of REDs in ultra endurance sport: a review. J Eat Disord. 2025;13:1-13. doi: 10.1186/s40337-025-01381-0.
47. Eberhardt HK, Willingham B, Brisebois M, Saracino P. Low Energy Availability Prevalence, Dietary Habits, and Sleep in Female Army ROTC Cadets. Int J Exerc Sci. 2025;18(6):1030-1046. doi: 10.70252/VRDK5031.
48. Ruscheck T, Kopp C, Nieß AM, Haigis D. The Athlete's Body Image in the Context of Relative Energy Deficiency in Sport-A Scoping Review. J Funct Morphol Kinesiol. 2025;10(4):413. doi: 10.3390/jfmk10040413.
49. Gowers CR, McManus CJ, Chung HC, Jones B, Tallent J, Waterworth SP. Assessing the risk of low energy availability, bone mineral density and psychological strain in endurance athletes. J Int Soc Sports Nutr. 2025;22(1):2496448. doi: 10.1080/15502783.2025.2496448.
50. Ferraris C, de Cassya Lopes Neri L, Amoroso AP, et al. Screening tool for the identification of relative energy deficiency in Sport risk: validation of the low energy availability in female questionnaire - Italian version (LEAFQ-ITA). J Int Soc Sports Nutr. 2025;22(1):2550317. doi: 10.1080/15502783.2025.2550317.
51. Wood AN, Soundy A. Pharmacological vs. Non-Pharmacological Treatment in the Management of Relative Energy Deficiency in Sport (REDs): A Systematic Review and Meta-Analysis. Sports. 2025;13(12):453. doi: 10.3390/sports13120453.
52. Williams NI, De Souza MJ, Misra M, Nattiv A, Joy E, et al. 2025 Update to the Female Athlete Triad Coalition Consensus Statement Part 2: Clinical Guidelines for Screening, Diagnosis, Treatment, and Return to Play for Adolescents and Adults. Sports Med. 2026;56:375-427. doi: 10.1007/s40279-025-02332-0.
53. McGuire A, Warrington G, Doyle L. Prevalence of low energy availability and associations with seasonal changes in salivary hormones and IgA in elite male Gaelic footballers. Eur J Nutr. 2023;62(4):1809-1820. doi: 10.1007/s00394-023-03112-0.
54. Garron T, Klein DJ. Male Army ROTC Cadets Fail to Meet Military Dietary Reference Intakes and Exhibit a High Prevalence of Low Energy Availability and Poor Sleep Quality. J Funct Morphol Kinesiol. 2023;8(3):95. doi: 10.3390/jfmk8030095.
55. Torres-McGehee TM, Emerson DM, Moore EM, et al. Energy Availability, Mental Health, and Sleep Patterns of Athletic Trainers. J Athl Train. 2023;58(9):788-795. doi: 10.4085/1062-6050-0547.22.
56. Allen N, Kelly S, Lanfear M, Reynolds A, Clark R, Mountjoy ML, et al. Relative energy deficiency in dance (RED-D): a consensus method approach to REDs in dance. BMJ Open Sport Exerc Med. 2024;10(1):e001858. doi: 10.1136/bmjsem-2023-001858.

57. Tuma C, Thomas A, Braun H, Thevis M. Development of an LC-HRMS/MS Method for Quantifying Steroids and Thyroid Hormones in Capillary Blood: A Potential Tool for Assessing Relative Energy Deficiency in Sport (RED-S). Metabolites. 2024;14(6):328. doi: 10.3390/metabo14060328.
58. Dvořáková K, Paludo AC, Wagner A, Puda D, Gimunová M, Kumstát M. A literature review of biomarkers used for diagnosis of relative energy deficiency in sport. Front Sports Act Living. 2024;6:1375740. doi: 10.3389/fspor.2024.1375740.
59. Skrzypiec-Spring M, Kuliczkowska-Płaksej J, et al. Atypical thyroid tests in an athlete treated for hypothyroidism as the first symptom of pituitary dysfunction due to relative energy deficiency. Endocrinol Diabetes Metab Case Rep. 2024;2024(4):EDM240066. doi: 10.1530/EDM-24-0066.
60. Jeppesen JS, et al. Short-Term Severe Low Energy Availability in Athletes: Molecular Mechanisms, Endocrine Responses, and Performance Outcomes-A Narrative Review. Scand J Med Sci Sports. 2025;35:e70089. doi: 10.1111/sms.70089.
61. Skorseth P, Segovia N, Hastings B, Kraus E. Prevalence of Female Athlete Triad Risk Factors and Iron Supplementation Among High School Distance Runners: Results From a Triad Risk Screening Tool. Orthop J Sports Med. 2020;8(10):2325967120959725. doi: 10.1177/2325967120959725.
62. Kinoshita N, Uchiyama E, Ishikawa-Takata K, et al. Association of energy availability with resting metabolic rates in competitive female teenage runners: a cross-sectional study. J Int Soc Sports Nutr. 2021;18:70. doi: 10.1186/s12970-021-00466-w.
63. Heradstveit S, Garnweider-Holme L, Winje BA, Sundgot-Borgen J, Mathisen TF. Knowledge of Eating Disorders and Relative Energy Deficiency in Sports among Public Health Nurses at Elite Sport Schools. SAGE Open Nurs. 2026;12:23779608251407784. doi: 10.1177/23779608251407784.

Appendix A. PubMed/MEDLINE search strategy

Database: PubMed/MEDLINE
Primary search date: 13 May 2026
Language: English-oriented screening
Publication-date restriction: none

Search string

("Relative Energy Deficiency in Sport"[Mesh] OR "Relative Energy Deficiency in Sport"[tiab] OR RED-S[tiab] OR REDs[tiab] OR "low energy availability"[tiab] OR "energy availability"[tiab] OR LEA[tiab] OR "Female Athlete Triad Syndrome"[Mesh] OR "female athlete triad"[tiab] OR "athlete triad"[tiab])
AND
("Athletes"[Mesh] OR "Sports"[Mesh] OR athlete*[tiab] OR sport*[tiab] OR "elite athlete*"[tiab] OR "recreational athlete*"[tiab] OR "endurance athlete*"[tiab])
AND
("Cognition"[Mesh] OR cognit*[tiab] OR neurocognitive[tiab] OR "Executive Function"[Mesh] OR "executive function"[tiab] OR "Decision Making"[Mesh] OR "decision-making"[tiab] OR "decision making"[tiab] OR "Attention"[Mesh] OR attention[tiab] OR "Memory"[Mesh] OR memory[tiab] OR "working memory"[tiab] OR "processing speed"[tiab] OR "reaction time"[tiab] OR "Mental Health"[Mesh] OR "mental health"[tiab] OR psychological[tiab] OR "psychological distress"[tiab] OR distress[tiab] OR "Depression"[Mesh] OR depression[tiab] OR depressive[tiab] OR "Anxiety"[Mesh] OR anxiety[tiab] OR mood[tiab] OR "Mood Disorders"[Mesh] OR wellbeing[tiab] OR "well-being"[tiab] OR "Sleep"[Mesh] OR sleep[tiab] OR "sleep quality"[tiab] OR "sleep duration"[tiab] OR "sleep efficiency"[tiab] OR "Sleep Initiation and Maintenance Disorders"[Mesh] OR insomnia[tiab] OR "insomnia disorder"[tiab] OR "Circadian Rhythm"[Mesh] OR circadian[tiab] OR "circadian rhythm"[tiab] OR actigraphy[tiab] OR polysomnography[tiab] OR PSG[tiab] OR "Hydrocortisone"[Mesh] OR cortisol[tiab] OR "HPA axis"[tiab] OR "hypothalamic-pituitary-adrenal"[tiab] OR "Insulin-Like Growth Factor I"[Mesh] OR IGF-1[tiab] OR "growth hormone"[tiab] OR "Leptin"[Mesh] OR leptin[tiab] OR "Ghrelin"[Mesh] OR ghrelin[tiab] OR "Triiodothyronine"[Mesh] OR T3[tiab] OR "thyroid hormone*"[tiab] OR "Serotonin"[Mesh] OR serotonin[tiab] OR "Dopamine"[Mesh] OR dopamine[tiab] OR neuroendocrine[tiab] OR neuroendocrinology[tiab])