2023 International Olympic Committee's (IOC) consensus statement on Relative Energy Deficiency in Sport (REDs)

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ABSTRACT

Relative Energy Deficiency in Sport (REDs) was first introduced in 2014 by the International Olympic Committee's expert writing panel, identifying a syndrome of deleterious health and performance outcomes experienced by female and male athletes exposed to low energy availability (LEA; inadequate energy intake in relation to exercise energy expenditure). Since the 2018 REDs consensus, there have been >170 original research publications advancing the field of REDs science, including emerging data demonstrating the growing role of low carbohydrate availability, further evidence of the interplay between mental health and REDs and more data elucidating the impact of LEA in males. Our knowledge of REDs signs and symptoms has resulted in updated Health and Performance Conceptual Models and the development of a novel Physiological Model. This Physiological Model is designed to demonstrate the complexity of either problematic or adaptable LEA exposure, coupled with individual moderating factors, leading to changes in health and performance outcomes. Guidelines for safe and effective body composition assessment to help prevent REDs are also outlined. A new REDs Clinical Assessment Tool-Version 2 is introduced to facilitate the detection and clinical diagnosis of REDs based on accumulated severity and risk stratification, with associated training and competition recommendations. Prevention and treatment principles of REDs are presented to encourage best practices for sports organisations and clinicians. Finally, methodological best practices for REDs research are outlined to stimulate future high-quality research to address important knowledge gaps.

INTRODUCTION

> My body was just deteriorating because it was working harder, but with less food. It's a sign that everything was basically just shutting down. I'd completely lost control of it [body], yet still thought it was just something I had to go through, because the ultimate aim is a certain weight or look.[1]

Athletes are driven by strong internal and external pressure to achieve optimal performance. Many forms of performance pressure contribute to scenarios that either, intentionally or unintentionally, alter energy intake (EI) and exercise energy expenditure (EEE), resulting in low energy availability (LEA). The mathematical formula for energy availability (EA) that identifies the amount of energy that the body can contribute to functions associated with health, well-being and performance is well-established in sports science/medicine[2–4]:

$$\text{EA [Energy Availability]} = \frac{\{\text{EI Energy Intake (kcal)} - \text{EEE [Exercise Energy Expenditure (kcal)]}\}}{\text{FFM [Fat-Free Mass (kg) / day]}}$$

Scenarios commonly encountered in sport include extreme volumes of EEE, attempts to improve power-to-weight ratios, desire for excessive leanness and sport-specific physique alterations. All of these scenarios can lead to problematic LEA (see Definitions box 1), which can result in negative health and performance implications known as 'Relative Energy Deficiency in Sport' (REDs). REDs (altered from the original acronym 'RED-S' for improved comprehension and dissemination), was first introduced by the International Olympic Committee (IOC) in a consensus statement in 2014,[5] and was updated in 2018.[6] Since 2018, there have been considerable scientific advancements in the REDs research field including ~178 REDs and/or LEA original research publications featuring ~23 822 participants; (80% female), with ~62% of these studies implementing a cross-sectional design, ~14% as longitudinal observational and ~12% longitudinal intervention (see literature summary in online supplemental file 1). These scientific advances have improved our understanding of the underpinning physiology and psychology of REDs and the different clinical presentations between the sexes. There is a wide range in the reported estimated prevalence of LEA/REDs indicators in female (23%–79.5%[7–16]) and male (15%–70%[12–20]) athletes across a variety of sports due to the lack of a singular definitive diagnosis, mistaken use of LEA and REDs as interchangeable terms, lack of standardisation and accuracy of research methodologies (eg, inaccurate EA measurements), variation in physiological demands among the study populations and participant study volunteering biases.[21]

Compared with previous REDs consensus statements, this updated IOC REDs consensus is more

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Box 1 Definitions: Low Energy Availability

Energy availability
Energy availability is the dietary energy left over and available for optimum function of body systems after accounting for the energy expended from exercise. Energy availability is expressed as kcal/kg FFM/day, and is defined in the scientific literature in the form of a mathematical formula[2–4]:

***EA** [Energy Availability] ={**EI** [Dietary energy Intake (kcal)]-**EEE** [Exercise Energy Expenditure (kcal)]} / **FFM** [Fat-Free Mass (kg) / day]*

Low energy availability (LEA)
LEA is any mismatch between dietary energy intake and energy expended in exercise that leaves the body's total energy needs unmet, that is, there is inadequate energy to support the functions required by the body to maintain optimal health and performance.[6] LEA occurs as a continuum between scenarios in which effects are benign (*adaptable LEA*) and others in which there are substantial and potentially long-term impairments of health and performance (*problematic LEA*).

Adaptable LEA
Adaptable LEA is exposure to a reduction in energy availability that is associated with benign effects, including mild and quickly reversible changes in biomarkers of various body systems that signal an adaptive partitioning of energy and the plasticity of human physiology. In some cases, the scenario that underpins the reduction in energy availability (eg, monitored and mindful manipulation of body composition or scheduled period of intensified training or competition) might be associated with acute health or performance benefits (eg, increased relative VO_{2max}). Adaptable LEA is typically a short-term experience with minimal (or no) impact on long-term health, well-being or performance. Moderating factors may also alter the expression of outcomes.

Problematic LEA
Problematic LEA is exposure to LEA that is associated with greater and potentially persistent disruption of various body systems, often presenting with signs and/or symptoms, and represents a maladaptive response. The characteristics of problematic LEA exposure (eg, duration, magnitude, frequency) may vary according to the body system and the individual. They may be further affected by interaction with moderating factors that can amplify the disruption to health, well-being and performance.

Moderating factors
Characteristics of individual athletes, their environment or behaviour/activities that may amplify or attenuate the effect of LEA exposure on various body systems. Relevant moderating factors (eg, gender, age, genetics) vary according to the body system. They may offer protection or additional risk in the progression from LEA exposure to the expression of disturbances to health, well-being or performance.

Eating disorders
Mental illnesses clinically diagnosed by meeting defined criteria characterised by abnormal eating behaviours [eg, self-induced restricting food intake, preoccupation with body shape or weight, bingeing and purging (self-induced emesis, laxative use, excessive exercise, diuretic use)].[172]

Continued

Box 1 Continued

Disordered eating behaviours
Abnormal eating behaviours including restrictive eating, compulsive eating or irregular or inflexible eating patterns, excessive exercise beyond assigned training to compensate for dietary intake, and use of purgatives. The behaviours do not meet the clinical criteria for an eating disorder.

Relative Energy Deficiency in Sport (REDs)
A syndrome of impaired physiological and/or psychological functioning experienced by female and male athletes that is caused by exposure to problematic (prolonged and/or severe) LEA. The detrimental outcomes include, but are not limited to, decreases in energy metabolism, reproductive function, musculoskeletal health, immunity, glycogen synthesis and cardiovascular and haematological health, which can all individually and synergistically lead to impaired well-being, increased injury risk and decreased sports performance.[5]

robust in its methodology including (1) outlining criteria for consensus panel inclusion, thresholds for reaching consensus via voting statements, and the provision for dissent[22 23]; (2) being supported by a dedicated edition of related reviews and editorials providing detailed context to facilitate further understanding[21 24–30]; and (3) featuring a blend of science and knowledge translation (implementing an athlete-centric and coach-centric approach).

The primary target audience for this consensus statement includes clinicians and REDs research scientists, with secondary educational materials being developed for coaches and athletes to support the primary prevention of REDs. We have intentionally developed real-world content for clinicians in the athlete health and performance team involved in the prevention, diagnosis and treatment of REDs.[25 27 29] For REDs scientists, in addition to a summary of the underpinning science in the field, we have also provided suggestions for future research implementing recommended methodologies.[21] The outcomes of this consensus are focused on the developing to world-class level athlete (Tiers 2–5).[31]

The goals of this consensus statement are to (1) summarise the recent scientific advances in the field of REDs; (2) introduce a novel REDs Physiological Model template and validated REDs Clinical Assessment Tool-Version 2 (IOC REDs CAT2); and (3) provide practical, REDs-related clinical and methodological research guidelines to promote athlete health and well-being, along with safe optimisation of sport performance. This consensus is organised into five sections: (1) What is REDs?, (2) methodology and consensus results, (3) key scientific advances since the 2018 REDs consensus statement, (4) clinical applications and (5) research methodology guidelines.

What is REDs?

Life History Theory proposes that various biological processes related to growth, health, activity and reproduction compete for finite energy resources, with different priorities depending on the phase in the life cycle and other circumstances.[32–34] In sports science literature, EA to meet various biological functions is the amount of energy remaining of the EI after the energy demands of exercise are accounted for. Inadequate EI or an increased energy commitment to one biological process favours trade-offs that allocate energy away from other processes, especially growth, reproduction or maintenance.[32] In particular,

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such evolutionary selective pressures have favoured adaptations that allocate limited energy supplies during periods of LEA (eg, famines) to biological processes that support immediate survival, as well as long-term reproductive success.[32] Therefore, humans, like other animals, are adapted to cope with periods of LEA by downregulating biological processes that are temporarily unnecessary or reducible.[32] Some of these perturbations to body systems might be considered mild and/or transient, representing physiological plasticity[35] and could be termed *adaptable* LEA (see Definitions box 1).

However, although humans evolved to be physically active, they did not evolve to tolerate some modern elite training programs[36] or sports-related practices. This is especially the case in endurance sports (often >30 hours of training/week),[37] which can sometimes result in extreme EEE that exceeds the capacity of the human alimentary tract for sustained energy absorption.[38] Indeed, the spectrum of exposure to LEA can include scenarios (eg, significant duration, magnitude, frequency—see Definitions box 1), that in conjunction with moderating factors (eg, sex, age, health status), are associated with negative effects on various body systems. Such scenarios, termed *problematic* LEA manifest as impairments of health and well-being, as well as interruption to training (adaptation and enhancement of body systems via exposure to physiological stress) or competition (demonstration of optimal mental and physiological prowess).[39] In the real world, athletes experience exposure to LEA (purposefully or inadvertently) in various manners along the continuum from adaptable to problematic.[3 40] Indeed, under certain circumstances, some practices associated with LEA, such as body composition manipulation, periods of intensified training or competition workloads involving prodigious EEE, can be safely and effectively periodised into an athlete's annual plan (eg, the implementation is guided by experts, the athlete has the physical and psychological readiness, adequate recovery is included, and health is maintained).[41 42]

REDs is a clinically diagnosed, multifactorial syndrome characterised by the accumulation of the deleterious health and performance outcomes resulting from exposure to problematic LEA. Thus, given the significant scientific advances in the field, the updated 2023 definition of REDs is:

> a syndrome of impaired physiological and/or psychological functioning experienced by female and male athletes that is caused by exposure to problematic (prolonged and/or severe) low energy availability. The detrimental outcomes include, but are not limited to, decreases in energy metabolism, reproductive function, musculoskeletal health, immunity, glycogen synthesis and cardiovascular and haematological health, which can all individually and synergistically lead to impaired well-being, increased injury risk and decreased sports performance.

Methodology and consensus results

In addition to facilitating the synthesis of compiled information, consensus methodology also harnesses experts' insights to enable more validated recommendations to be made when the published evidence ranges from insufficient to adequate. The goal of consensus methods is to determine how much independent and diverse experts agree on nuanced and complex issues within a defined topic area while seeking to overcome some of the drawbacks associated with decision-making in groups or committees, which can be frequently dominated by one individual or coalitions representing vested interests.

This REDs consensus statement used the RAND-UCLA Appropriateness Method (RAM).[43] A diverse (ie, gender, geographic location, expertise) expert panel of authors was invited, consisting of sports medicine physicians, a sports endocrinologist, registered sports dietitians, sports physiologists, sports scientists, an athlete, a coach and a mental performance consultant. Authors were invited based on their expertise, as demonstrated by previous research, clinical and/or coaching experiences with REDs. From the entire group of authors, smaller working groups of content experts were tasked with preparing specific subtopics prior to the in-person consensus in the form of (1) a referenced summary of the existing scientific literature and (2) voting statements based on key novel and potentially controversial aspects identified in the literature review. These literature summaries and voting statements were compiled, then circulated for online confidential voting (Delphi method[44]). Answer categories were from strongly disagree, undecided, to strongly agree. We defined three levels of agreement based on which subsequent discussions were held:

1. Agreement: ≥80% of authors agreeing on the voting statement, without any author disagreeing.
2. Agreement with minority disagreement: ≥80% of authors agreeing on the voting statement, but with one or more authors disagreeing.
3. Disagreement: <80% of authors agreeing on the voting statement.

Statements without agreement were discussed at the subsequent meeting held at the Olympic House in Lausanne, Switzerland (September 2022). Authors were allowed to write a minority opinion in the event of disagreement with a statement when the consensus threshold was reached. The voting statements were revised after discussions and then subjected to a second round of confidential electronic voting at the end of the meeting (full details of voting statements, outcomes and actions are available via supplementary materials (online supplemental files 2–4)).

Consensus results

In the first round of online voting, we presented 135 evidence statements to the panel. Full agreement was reached for 76 of the statements. We have outlined our actions taken after in-person discussions in table 1. In the second round of confidential voting, 44 statements were presented to the authors. Of these, 24 were previous statements with disagreement that required a revote, and 20 were new statements. All voting statements reached an agreement or minority disagreement after two rounds of voting, providing a total of 144 statements of which 27 remained with a minority disagreement (ie, 80% agreement was reached, but one or more individuals disagreed with the statement).

Equity, diversity and inclusion statement

A diverse expert panel of authors consisted of sports medicine physicians, registered sports dietitians, athletes, coaches, sports physiologists, sports scientists and mental performance consultants. Authors were invited based on their expertise, as demonstrated by previous research, clinical and/or coaching experiences with REDs. In total, 10 females and 7 males from four continents participated.

Key scientific advances since the 2018 REDs consensus statement

There has been significant growth in the number of studies clearly showing that problematic LEA is the underlying aetiology of REDs. The new evidence on this topic provides a deeper fundamental understanding of how problematic versus adaptable LEA, along with its moderating factors, influences the health and performance of athletes (see Definitions box 1). The key

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Table 1 Results of the online Delphi survey and subsequent actions taken

	Total	Agreement*	Minority disagreement†	Disagreement‡
Round 1 voting	**135**	**76**	**29**	**30**
Action taken				
Removed	11	–	2	9
Adjusted wording: revote	23	–	3	20
Adjusted wording: no revote	23	23	–	–
Revote	1	–	–	1
Added statements	20	–	–	–
Round 2 voting	**44**	**41**	**3**	
Overall outcome	**144**	**117**	**27**	

*Agreement: ≥ 80% agree without disagreement but potentially includes 'undecided' votes.
†Minority disagreement: ≥ 80% agree but with one or more disagreeing opinions.
‡Disagreement: <80% agreement.

emerging themes are (1) the additive impact of low carbohydrate availability (LCA) with LEA in the development of REDs; (2) the overlap of REDs and overtraining syndrome (OTS) symptomology; (3) the time-course of biomarker responses to problematic LEA in the development of REDs; (4) improved understanding of mental health associations of REDs; (5) advances in knowledge pertaining to REDs in male athletes and (6) para athletes.

The magnifying impact of LCA in the context of REDs

Most LEA intervention studies are also accompanied by a substantial reduction (25%–60%, depending on magnitude of LEA) in carbohydrate (CHO) ingestion, resulting in concurrent LCA.[45–48] In the real world, the magnitude of LCA is likely to be even greater considering the emphasis on protein intake during periods of calorie restriction.[49–51] Recently, several investigations have elucidated CHO's energy-independent or magnifying role in REDs-related health outcomes. There have been several short-term (≤6 days) investigations in male endurance athletes comparing the effects of high energy and high CHO availability, high energy with low CHO (<3 g CHO/kg BM/day) but high fat (LCHF), or low energy with low to moderate CHO availability diets on bone, immunity and iron biomarkers. These studies have reported increases in bone resorption biomarkers[52 53] with a concomitant impairment in biomarkers of bone formation,[53] as well as increased postexercise concentrations of interleukin-6 (IL-6) and hepcidin after LCA.[54] These findings suggest deleterious effects on bone, immunity and iron biomarkers as a result of LCA, sometimes in the absence of LEA. More recently, a 3-day intervention in young females also showed a 264% increase in hepcidin with a low energy, low CHO diet compared with only a 69% increase in hepcidin with isocaloric low energy but higher CHO diet.[55] Additionally, ~3.5 weeks of LCHF diet in elite endurance athletes resulted in impaired markers of bone remodelling both at rest as well as around exercise (up to 3 hours postexercise),[56] and elevated postexercise IL-6 concentrations compared with an isocaloric high CHO treatment.[57] Six studies since 2019 have shown an energy-independent and/or magnifying impact of LCA in the accelerated development of REDs outcomes.[52–57] Accordingly, LEA intervention studies need to also control and account for CHO intake and need to be of longer duration to determine long-term adaptation.

Symptomology overlap between REDs and OTS

REDs and OTS are syndromes involving the hypothalamic–pituitary–adrenal axis and have no single validated diagnostic biomarker; they feature a complex overlap of symptoms that hinge on a diagnosis utilising exclusion criteria.[37 58] Accordingly, a recent narrative review found that 18 of 21 identified OTS-based studies showed indications of LEA and LCA due to large increases in training while failing to compensate with increased EI, and thus may have demonstrated REDs outcomes rather than OTS.[37] It is important to note that LEA and/or LCA, although challenging to assess, should be *excluded* from an OTS diagnosis as LEA is the underlying aetiology for a REDs diagnosis.[37 59]

Time-course of LEA resulting in REDs

Although acute mild periods of LEA do not always lead to adverse outcomes, problematic LEA exposure leads to REDs. Our scientific understanding of the time-course of LEA leading to validated physiological and psychological signs/symptoms are still emerging, largely due to difficulties in accurately assessing and controlling for EA in prospective research.[21] Emerging definitions highlight short-term LEA as a few days to weeks, medium-term as weeks to months and long-term as months to years.[37 40] However, time-course cut-offs require further scientific validation, may differ between males and females and change with the severity and duration of LEA dose. Still, some signs/symptoms and REDs outcomes that appear to present temporally to various exposure periods of LEA have emerged. Importantly, some short-term signs or symptoms during the acute assessment may only represent a snapshot of a current LEA state and require the exclusion of other potential aetiologies (differential diagnoses). Such signs or symptoms do not always reflect a problematic LEA exposure leading to REDs.

Mental health outcomes of REDs

The sports community has prioritised the mental health of elite-level athletes as evidenced by a sharp rise in consensus statements[60–62] and prevalence studies[63–65] on this theme. A parallel focus has been the increased awareness of the risk factors for and the consequences of REDs, where psychological factors contributing to LEA and mental health consequences have been highlighted,[5] although less well understood.[6 35 66] Recent qualitative studies[1 67] involving mainly subelite endurance athletes provide support for this premise, reporting that LEA from intentional (eg, weight regulation) or unintentional (eg, failing to consciously increase EI with increased EEE) origins can be associated with short-term positive results such as performance improvements or social approval from the coach and the sports culture.[1] These short-term 'positive' outcomes make it more challenging for athletes to recognise the longer-term potential health and performance implications of exposure to problematic LEA.

Disordered eating (DE) behaviours, eating disorders (EDs) and/or REDs are common among certain athlete cohorts.[68] LEA

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and DE behaviours, which exist along the spectrum between optimised nutrition and clinical EDs, may occur in isolation or together.[68] A prior history of DE behaviours or an ED might perpetuate a continued under-fuelling of energy[1] and must therefore be considered an important risk factor for developing REDs. DE behaviours and EDs may be exacerbated by social media influence, societal pressures, the athlete's training/coaching entourage, a belief that a specific physique/weight/appearance will improve performance and/or overall body dissatisfaction.[69] Given the potentially serious outcomes of DE behaviours and EDs, prevention, early identification, and timely interventions should be prioritised.[60 70]

Psychological indicators associated with problematic LEA and REDs are mood disturbances/fluctuations,[8 71 72] cognitive dietary restraint,[73] drive for thinness,[74 75] reduced sleep quality[50 76] and perfectionistic tendencies.[77] Depressive symptoms and affective disorders,[8 78 79] subjectively reported reduced well-being,[73] primary or secondary exercise dependence/addiction,[80 81] anxiety related to injury and/or recovery, sport-specific issues such as difficulty coping with weight requirements[67 76] and the development of EDs[1 82] are additional adverse mental health outcomes associated with problematic LEA and REDs. However, we must recognise that the picture is still unclear regarding the dynamics of mental health and DE behaviours according to sex and level of competition,[83] as well as in athletes with physical disabilities.[84] Furthermore, studies are required to (1) ascertain why many athletes experience few or no negative mental health consequences in the early stages of problematic LEA exposure[20 72 85] and (2) to better understand the reciprocal function of the different psychological variables.[86 87] As perceived stress appears to be common for many mental health concerns related to LEA and REDs, a heightened focus should be placed on developing psychologically safe environments surrounding athletes. Details on creating safe sport environments are outlined in the IOC consensus statement on mental health in elite athletes.[60]

REDs in male athletes

Although the 2014 IOC REDs consensus statement[5] and the 2018 update paper[6] alluded to the impact of LEA and REDs in male athletes, the available research on males at the time was scant. Since then, although the research community has emphasised the need for studies in men, currently only 20% of original studies from 2018 to 2022 include male athletes as subjects (see literature search summary in online supplemental file 1).

While a universal cut-off of 30 kcal/kg FFM/day as a threshold of LEA leading to some REDs outcomes in females is debated,[88] such a cut-off or range at which males experience REDs-related symptoms is even less understood,[89] but appears to be lower (eg, ~9 to 25 kcal/kg FFM/day).[17 46 72 90 91] Indeed, there is evidence that most males can sustain a lower EA before physiological and psychological disturbances manifest. Nevertheless, problematic LEA can occur in male athletes and is associated with negative effects on the hypothalamic–pituitary–gonadal (HPG) axis and associated hormones[72 92–100]; changes in metabolic hormones[46 101–103]; impairments to immune function[104]; detriments to bone health[105]; as well as negative performance outcomes[18 90 104 106] and decreased lean body mass accrual.[107] Although changes are comparable to those REDs outcomes found in female athletes, the magnitude of the effects on some physiological parameters and the threshold at which these effects manifest appear to be variable between the sexes. Two emerging potential indicators of REDs in males are the presence of low libido and decreased morning erections, which have been identified as physiological consequences of LEA.[108–111]

REDs in para athletes

The estimated prevalence of REDs in para athletes is unknown; however, there are concerns that para athletes may be at even higher risk of problematic LEA than able-bodied athletes.[112] Among US para athletes preparing for Paralympic Games, 62% attempted to alter weight or body composition to enhance performance, 32% had elevated scores on the Eating Disorder Examination Questionnaire (EDE-Q) pathological behaviour subscale scores and 44% of the female athletes reported menstrual dysfunction.[113] Another study of EA estimates in wheelchair athletes reported that nearly the entire cohort fulfilled criteria of LEA across at least one 24-hour period during the week-long study.[114] Whether negative body image, risk of LEA and/or DE behaviours and EDs are related to their disability, athletic status, competitive pressure, training environment or a combination of factors remains to be elucidated.

Problematic LEA can lead to impaired bone health and bone-related injury secondary to factors such as altered skeletal loading experienced by para athletes (ie, the lack of loading stimulus experienced by wheelchair athletes and/or low-impact sports). Furthermore, in unilateral amputees, the affected limb may exhibit reduced bone mineral density (BMD).[115] Additionally, the presence of central neurological injury may result in alterations of the HPG axis and baseline menstrual function, regardless of energy status.[116 117] The risk of bone stress injury (BSI) is of particular concern in athletes with spinal cord injury who experience a substantial loss of BMD immediately post-injury and hence have a high incidence of low BMD for age and/or osteoporosis.[118] Dual-energy X-ray absorptiometry (DXA) is the most well-accepted tool for the measurement of BMD, but there are limitations in using standard population comparison reporting (eg, Z-scores); normative, reference datasets are determined from measurements in able-bodied populations and stratified by age-matched, sex-matched and limited race/ethnicity-matched categories to determine diagnostic cut-offs for 'low BMD for age' and 'osteoporosis'.[119 120] Therefore, there is a need for research in a wide variety of para athletes to develop BMD assessment techniques and reference ranges appropriate for the para athlete population.[112]

REDs Conceptual Models

The REDs Conceptual Models were developed to raise awareness of the athletic, coach, sports science and sports medicine communities to this syndrome. Figure 1 (REDs Health Model) and figure 2 (REDs Performance Model) are conceptual models that demonstrate the range of body systems for which there is theoretical, empirical, and/or clinical evidence of impairments that manifest in different ways. Undoubtedly, these outcomes occur over different timeframes and with different severity and significance to the individual athlete due to various moderating factors.[24]

Unlike earlier REDs models,[5 6] LEA is placed at the centre of the hub to note its role as an exposure variable. Graded arrows illustrate a continuum from adaptable LEA to problematic LEA exposure, with the former representing benign physiological adaptations to energy fluctuations (ie, physiological plasticity),[44] while the outer region of the hub notes the range of health and performance concerns which can be associated with the latter. A spectrum of energy mismatches, with differing severity of consequences, was part of the original concept of EA.[2 4] However, the updated model uses qualitative terms (adaptable, problematic) as an alternative to the previous focus on quantitative assessments with universally applied thresholds of concern. The most well-documented sequelae of problematic LEA are impairments

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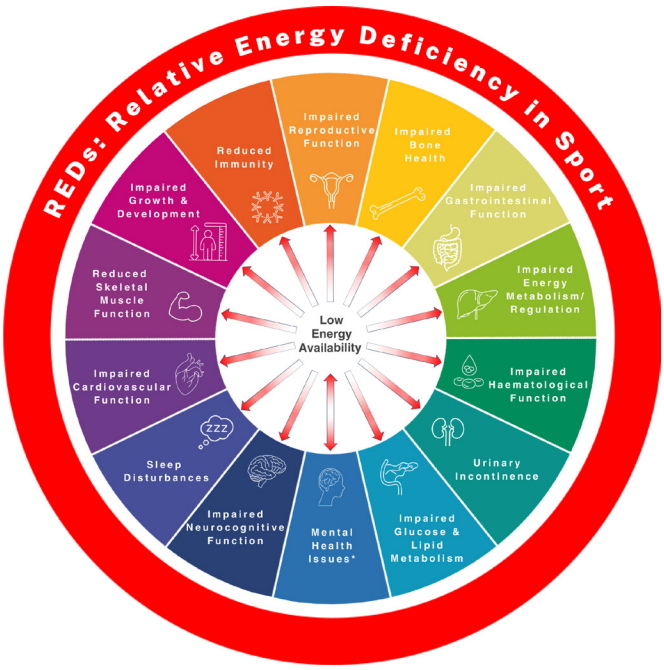

Figure 1 REDs Health Conceptual Model. The effects of LEA exist on a continuum. While some exposure to LEA is mild and transient termed adaptable LEA (arrow depicted in white), problematic LEA is associated with a variety of adverse REDs outcomes (arrow depicted in red). *Mental Health Issues can either precede REDs or be the result of REDs. LEA, low energy availability; REDs, Relative Energy Deficiency in Sport.

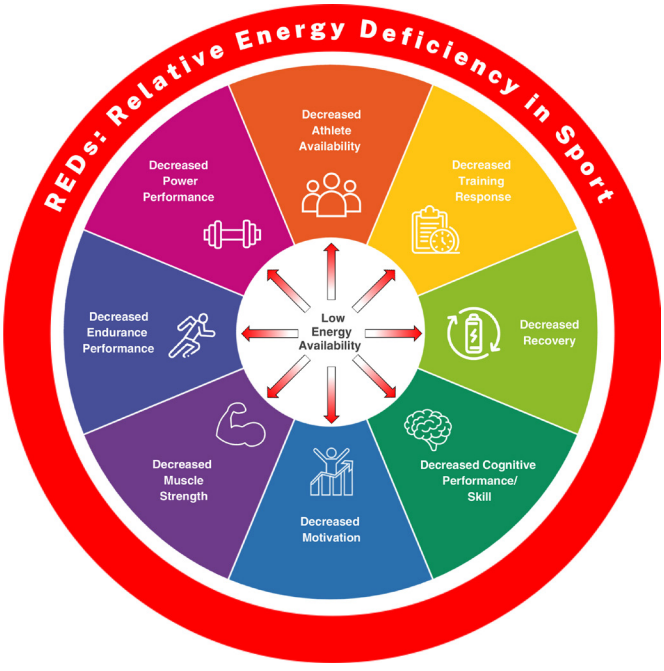

Figure 2 REDs Performance Conceptual Model. The effects of LEA exist on a continuum. While some exposure to LEA is mild and transient, termed adaptable LEA (arrow depicted in white), problematic LEA is associated with a variety of adverse REDs performance outcomes (arrow depicted in red). LEA, low energy availability; REDs, Relative Energy Deficiency in Sport.

of reproductive function and bone health in female and male athletes.[121–123] Tables 2 and 3 summarise these and many other conditions associated with LEA in athletes and other populations. Future updates will likely revise the range of recognised sequelae associated with REDs as we learn more about the effects of energy allocation and potential prioritisation of various body systems.

It is important to note that the REDs Health and Performance Conceptual Models are not separate entities; they involve considerable overlap. Indeed, presenting this information in two wheels simply offers different audiences an appreciation of the issues of greatest relevance to them. Each sign or symptom within the REDs Conceptual Models can occur due to aetiologies other than problematic LEA (tables 2 and 3). Therefore, the exclusion of primary aetiologies (differential diagnoses) should occur when diagnosing REDs (see Clinical Assessment Tool section below).

REDs Physiological Model

Experts in the field have long realised that applying LEA exposure (ie, severity, duration, frequency) on subsequent REDs short, medium and long-term outcomes is complex and dependent on many moderating factors. Accordingly, and novel to this 2023 consensus update, a more researched and clinically based unifying physiological model has been developed. To progress the REDs scientific field forward, we need integrated dynamic physiological models that can help explain the biological complexity and interaction within and between various body systems, as well as the inconsistencies in the manifestation of REDs signs and symptoms resulting from problematic LEA. Ideally, unique physiological models can be developed for each body system within the Health Conceptual Models (see figure 1) before being integrated to acknowledge substantial physiological 'cross-talk' among systems.

Step 1 of the REDs Physiological Model for each body system (figure 3) is to identify the range of specific health and performance impairments that might occur from LEA exposure, along with details of the criterion tests and metrics that best assess the presence of such disturbances. ***Step 2*** is to focus on characteristics of an athlete's LEA exposure (see figure 3 for examples) that might create a higher risk of it being problematic; for example, the duration, magnitude or origin of the LEA mismatch (see figure 3 for examples). ***Step 3*** is to consider moderating factors in an individual athlete's makeup, behaviours or environment that may either exacerbate or protect against various LEA-associated health and/or performance dysfunctions as they related to the specific body system. A systematic identification of such moderating factors is proposed (figure 3).

The development of a physiological model for each body system, underpinned by a 'systems biology mindset',[124] will enable a more nuanced assessment of the individual athlete and whether their specific combination of LEA exposure and secondary moderators is likely to lead to positive, neutral or negative health and/or performance outcomes.

Clinical applications

Assessment of EA

Seminal research[45 125] around EA in habitually sedentary females identified a continuum of zones ranging from low to high risk of harm (eg, high EA for mass gain and growth ≥45 kcal/kg FFM/day; adequate EA for weight maintenance and support of body function = ~45 kcal/kg FFM/day; reduced EA for body

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Table 2 Potential REDs health outcomes resulting from problematic LEA

Spoke	Examples of impairment	Populations with LEA (assessed directly or via surrogates) providing evidence of impairment	Examples of differential diagnoses (issues to be excluded)
Impaired reproductive function	***Females***	SF,[45 127 173–175] FA[176–179]	***Females***
	Alteration in LH concentrations or pulsatility	SF,[88] FA[168 180–184]	*Primary amenorrhoea*: constitutionally delayed puberty, various genetic syndromes, anatomic abnormalities
	Reduced oestrogen and progesterone	FA[178]	*Secondary amenorrhoea:* pregnancy, PCOS, pituitary mass (eg, prolactinoma), thyroid abnormalities
	Reduced testosterone	FA[185 186]	*Other menstrual dysfunction:* use of hormonal birth control methods, physiologic stress
	Primary amenorrhoea	SF,[88] FA[183 187 188]	***Males***
	Oligomenorrhoea/menstrual irregularities	FA[181–183 187 189 190]	Primary hypogonadism (gonadal disease), Hypogonadism (eg, hypothalamic/pituitary disease), toxic exposures, infection, psychosomatic neurological dysfunction
	Secondary amenorrhoea (FHA)	SF,[88 174] FA[187]	
	Luteal phase defects/deficiency	SF,[88] FA[187]	
	Anovulatory cycles		
	Males		
	Reduced testosterone	MA[18 90 98 102 191–193]	
	Sperm abnormalities	MA[194]	
	Erectile dysfunction	MA[81 108 111]	
	Females and males		
	Decreased libido	MA[108 111 194]	
Impaired bone health	Longitudinal loss of BMD/lack of expected bone accrual or maintenance (younger populations)	ANF,[195] FA,[196] MA[197]	*Low BMD:* genetic bone disorders (eg, osteogenesis imperfecta), hyperparathyroidism, poor micronutrient intake (eg, calcium and vitamin D), malabsorption disorders (eg, coeliac disease), malignancies (eg, leukaemia, lymphoma, metastasis), renal diseases, medications (eg, anabolic steroids)
	Lower BMD/low Z-score	FA[49 198–200] MA[18 49 200 201]	
	Impaired bone strength or microarchitecture	FA[202–204] MA[205]	
	Bone stress injuries		*Bone stress injury*: External reasons (eg, training errors, surface, shoes) or internal issues (eg, body build, medical predispositions as above)
	Change/differences in bone remodelling biomarkers	FA,[49 81 206–209] MA[49 81 210]	
		SF,[125] FA,[47 170 179 211] MA[53 212 213]	
Impaired GI function	Abdominal pain/cramps/bloating/alteration in bowel movements	FA,[8 81 189 214] MA[81]	GI diseases (eg, Coeliac disease, inflammatory bowel disease, *Helicobacter pylori*, gastro-oesophageal reflux, functional dyspepsia/constipation), medications (eg, antidepressants, iron pills, narcotics, laxative/cathartic use in EDs)
Impaired energy metabolism/ regulation	Subclinically or clinically low T3	SF,[127 165 215 216] FA,[49 168 170 184 188 190 217 218] MA[49 192]	Primary or central (secondary and tertiary) hypothyroidism, medications/supplements
	Low RMR/RMR ratio		
	Reduced leptin	FA[182 189 190 217–222] MA[103 191 223]	
		SF,[45 160] FA,[47 170 179 188 217] MA[46 224]	
		SF,[127 175] FA,[178 179 184 222 225] MA[80 102]	
	Increased cortisol		*Increased cortisol:* physiologic stress, Cushing disease, steroid use
Impaired haematological status	Low iron status	FA[226]	Acute or chronic blood loss (eg, menstrual cycle, GI bleeding), RBC destruction (eg, haemolysis, haemoglobinopathy, splenomegaly), poor micronutrient intake (eg, iron, vitamin B_{12}, folate), bone marrow diseases
	Increased hepcidin concentrations/response	SF,[55] MA[171 227]	
	Reduced iron absorption	MA[227]	
	Lower haemoglobin concentration/mass	FA,[228] MA[73]	
	Reduced response to altitude training	MA[229]	
Urinary incontinence	Urinary incontinence	FA[230–232]	Persistent urinary incontinence: trauma (eg, childbirth, surgery, radiation), anatomical abnormalities, neurological diseases Temporary urinary incontinence: pregnancy, urinary tract infection, constipation, certain foods and drugs
Impaired glucose and lipid metabolism	Reduced fasting/24-hour glucose	SF,[127] FA,[184 214 219] MA[233]	Impaired glucose metabolism: insulinoma, critical illness, medications, adrenal insufficiency Impaired lipid metabolism: familial hyperlipidaemia
	Reduced fasting/24-hour insulin	SF,[127] FA,[47] MA[46 102 233 234]	
	Elevated total cholesterol/LDL cholesterol	FA,[181 235 236] MA[72 192 193]	
Mental health issues	Depression	FA,[8 78 79] MA[79]	Primary psychologic/mood disorders
	Exercise dependence/addiction	FA,[81 237] MA[80 81]	
	DE behaviours/EDs	FA,[81 182 219] MA[80 81]	
Impaired neurocognitive function	Reduced/impaired memory	FA,[238] ANF[239]	Dementia (eg, Alzheimer's disease), vitamin deficiencies, infections, malignancies, ADHD, substance use disorder, primary psychologic/mood disorders, traumatic brain injury
	Reduced/impaired decision-making	ANF[240]	
	Reduced/impaired spatial awareness	FA[241]	
	Poor planning/cognitive flexibility	ANF[242]	
	Reduced executive function	FA[238]	

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Table 2 Continued

Spoke	Examples of impairment	Populations with LEA (assessed directly or via surrogates) providing evidence of impairment	Examples of differential diagnoses (issues to be excluded)
Sleep disturbances	Sleep disturbances (self-reported)	FA,[76] MA[50]	Primary psychologic/mood disorders, shift-work, obstructive sleep apnoea, chronic pain/injury, nocturia, medications/substance use, restless legs syndrome
Impaired cardiovascular function	ECG abnormalities (eg, sinus bradycardia, QT prolongation and QT dispersion)	FA,[189 243] MA,[72 244] ANM,[245] ANF[246 247]	*Bradycardia*: Genetic, ultra-endurance training, hypothyroidism, medications (eg, beta-blockers), toxic exposures, electroconductive disorders, electrolyte abnormalities
	Haemodynamic abnormalities (eg, hypotension and orthostatic hypotension, syncope)	FA,[243 248] ANF,[249] MA[244]	
	Impaired endothelial function/reduced blood flow	FA,[221 235 243 250–254] MA[255]	
	Cardiac abnormalities (eg, MVP, decreased left ventricular mass, decreased left ventricular systolic function, myocardial fibrosis)	ANF,[256] ANM[245 256]	*Hypotension:* illness, medications, dehydration
Reduced skeletal muscle function	Reduced rate of muscle protein synthesis	FA,[257–259] SM,[260] MA[257 258]	Inadequate protein intake
	Reduced rates of muscle glycogen restoration	FA,[261] MA[48 262]	Inadequate CHO intake
Impaired growth and development	Reduced IGF-1	SF,[127 215] FA,[168 170] MA[192 234 263 264]	Constitutional delayed puberty, chronic diseases, GH deficiency, congenital or acquired hypogonadotropic hypogonadism, genetic defects, hyperprolactinaemia, long-term drug use (eg, anabolic steroids, opioids, glucocorticosteroids)
	Increased GH/GH resistance	SF,[127] FA,[178] MA[102 264]	
	Deviation from the expected growth curve	FA,[186] ANF,[265 266] ANM[267 268]	
Reduced immunity	Increased infection/illness susceptibility	FA,[10 269–271] MA[10 269 271]	Primary or acquired immune deficiency (eg, chemotherapy, viral infections)
	Change in immune biomarkers	FA,[272] MA[273]	Intensive exercise without LEA

Each of these outcomes can occur in the absence of LEA, therefore the differential diagnosis should be considered in the assessment and diagnosis of REDs severity and/or risk.
Populations providing evidence types: SF: sedentary females; FA: female athletes; ANF: females with anorexia nervosa; MA: male athletes; SM: sedentary males; ANM: males with anorexia nervosa.
ADHD, attention-deficit/hyperactivity disorder; CHO, carbohydrate; ECG, electrocardiogram; EDs, eating disorders; FHA, functional hypothalamic amenorrhoea; GH, growth hormone; GI, gastrointestinal; IGF-1, insulin-like growth factor-1; LDL, low density lipoprotein; LEA, low energy availability; LH, luteinising hormone; MVP, mitral valve prolapse; OCD, obsessive compulsive disorder; PCOS, polycystic ovary syndrome; RMR, resting metabolic rate; T3, triiodothyronine.

mass/fat loss=30–45 kcal/kg FFM/day; and LEA causing health implications ≤30 kcal/kg FFM/day).[126] The concept of the LEA threshold (30 kcal/kg FFM/day), below which health problems occurred, was based on elegant but short-term laboratory studies that investigated stepwise changes in EA, perturbations of sex hormones[45 127 128] and changes in markers of bone turnover[125] in a small sample of sedentary females. Although this concept was intended as a guide, rather than a diagnostic end-point, more recent information gleaned from real-life clinical observations, as well as short-term studies,[88] theoretical constructs and methodological challenges in assessment, around the frailty of a single, universal threshold,[129] have identified large differences in the EA level associated with health and performance concerns between individuals, the sexes, and among different body systems. Therefore, although EA calculations may inform research interventions or observations, there are risks in setting a definitive clinical threshold of EA due to many moderating factors.

Unfortunately, the measurement of EA in free-living athletes is challenged by a high level of burden (eg, time, effort) to the participant and assessor. Also, protocols to undertake EA assessments or EA-based diet prescription will continue to be challenged by the errors associated with accurately measuring EI, EEE and other contributing components (eg, FFM, resting metabolic rate (RMR)),[40 49 129] but these can be better managed in the future by implementing a standardised approach. Protocols that achieve a harmonised time-course for assessment and the individual components of EA may assist in future LEA and REDs activities by standardising the errors and limitations of the assessment, and balancing the issues of time and resource burden, feasibility and measurement precision. Future use of standardised methodologies should assist in better assessment of EA, more nuanced interpretation of past and future data, and better replication or comparison of work in this area.

Body composition assessment and management

Body composition assessment and management are important for optimising health and athletic performance, particularly in weight-sensitive and leanness-demanding sports.[130] Athletes may experience internal and/or external pressure to attain an 'athletic look' (aesthetic), potentially leading to body dissatisfaction and LEA, and then to symptoms of REDs, DE behaviours or EDs.[76 131] This is of concern, especially for young athletes, due to potentially long-lasting negative physical and psychological outcomes. Thus, body composition assessment is recommended only for medical purposes under 18 years of age[26 132 133] (see figure 4). Exceptional circumstances may exist where body composition assessment may be justified for athletes <18 years. Still, such a decision warrants careful consideration and consensus among the athletes' health and performance team and requires guardian consent.

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Many sports have engrained cultures where coaches and members of the athlete health and performance team exert subtle to extreme pressure on athletes to regulate body weight and composition.[131 134] Unfortunately, many members of the athlete entourage appear to (1) lack the knowledge of safe regulation of body weight and composition and how it can be utilised to improve performance while maintaining health; (2) have ignorance of the suitability of various body composition methods and the possible negative health effects consequent to inappropriate assessment and (3) have inadequate communication skills, with lack of optimised protocols on how to manage and safely implement the data to promote health and performance without the added risk of developing REDs, DE behaviours or EDs. In some instances, erroneous and intensive body composition measurement could lead to allegations of harassment and abuse by athletes.[132 135] It is important, therefore, to identify valid and

Table 3 Potential REDs performance outcomes that can result from problematic LEA

Spoke	Examples of direct or indirect impairment	Athletic populations with LEA (assessed directly or via surrogates) providing evidence of impairment
Decreased athlete availability (illness and injury)	Increase in training days lost or modified due to illness or injury (eg, impaired preparation)	Tier 4* FA (n=85) and MA (n=47) Olympic athletes from 11 different sports[10] Tier 4 FA (n=55) and MA (n=26) Olympic athletes from 11 different sports[269] Tier 4 FA endurance athletes (n=45)[214] Tier 4 FA endurance athletes (n=13)[274] Tier 3 FA college athletes (n=116) from endurance, power and team sports[275] Unspecified Tier FA high-school athletes (n=163) from endurance, power and team sports[276] Unspecified Tier FA high-school athletes (n=249) from aesthetic, endurance and team sports[277] A mix of Tier 1–4 FA (n=833)[270] Tier 2 FA figure skaters (n=137)[278]
	Inability to compete at key competitions due to illness or injury	Tier 4 FA endurance athletes (n=13)[274]Unspecified Tier FA high school athletes (n=163) from endurance, power and team sports[276]
Decreased training response	Decreased rather than increased performance of treadmill protocol following 4 weeks intensified training plus 2 weeks recovery	Tier 2 club level FA endurance runners (n=16)[71]
	Reduced performance of 5 km on-water rowing following a period of intensified training	Tier 4 national level MA (n=5) and FA rowers (n=5)[279]
	Reduced swimming velocity in 400 m time trial after 12 weeks of training	Tier 3 junior national level FA swimmers (n=10)[168]
	Self-reported reduction in training response	Unspecified mixed tier FA (n=1000)[8]
	Decreased aerobic (4000 m time trial) and anaerobic (15 s) performance after 2 weeks intensified training including inadequate energy intake	Tier 3 MA road cyclists (n=13)[106]
Decreased recovery	Direct: self-reported failure to recover between training sessions	Tier 4 FA (n=8) and MA (n=4) lightweight rowers[76]
	Indirect: reduced glycogen synthesis	Tier 3 MA endurance runners (n=7)[48] Tier 1 MA (n=6) and FA (n=7) endurance athletes[261]
	Indirect: reduced muscle protein synthesis	Unspecified tier resistance-trained FA (n=7) and MA (n=8)[257]
	Indirect: reduced PCr recovery	Tier 2 FA (n=19) endurance athletes[280]
Decreased cognitive performance/skill	Reduced reaction time Self-reported impaired judgement and decreased coordination and concentration	Tier 4 FA endurance athletes (n=30)[184] Unspecified tier FA (n=1000)[8]
Decreased motivation	Decreased well-being Increase in total mood disturbance (eg, fatigue, vigour) Self-reported increase in irritability and depression Emotional lability Increased irritability Increase in total mood disturbance and general stress Self-reported decrease in mood, emotional self-regulation, concentration, social interaction, food anxiety	Tier 3 MA endurance athletes (n=18)[90] Tier 4 national level MA (n=5) and FA rowers (n=5)[279] Unspecified tier FA (n=1000)[8] Tier 2–4 Mix of sports FA (n=8)[67] Tier 3 Endurance FA (n=10) and MA (n=2)[67] Tier 3 MA Road cyclists (n=13)[106] Tier 4 FA (n=8) and MA (n=4) lightweight rowers[76]
Decreased muscle strength	Decreased neuromuscular strength Decreased explosive power (countermovement jump) Decreased explosive power (countermovement jump, reactive jump) Decreased concentric hamstring peak torque Decreased isometric bench press Decreased one rep max squat, bench press, deadlift Decreased concentric and eccentric peak force	er 4 FA endurance athletes (n=30)[184] Tier 3 MA endurance athletes (n=18)[90] Tier 2–3 MA bodybuilder (n=1)[281] Tier 2 junior elite FA cross country skiers (n=19)[282] Tier 2–3 MA bodybuilder (n=1)[85] Tier 2–3 FA fitness competitors (n=27)[188] Tier 2–3 MA bodybuilder (n=1)[244] Tier 2–3 FA physique athlete (n=1)[283]

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Table 3 Continued

Spoke	Examples of direct or indirect impairment	Athletic populations with LEA (assessed directly or via surrogates) providing evidence of impairment
Decreased endurance performance	Decreased performance of treadmill run protocol Reduced 5 km on-water rowing performance Decreased neuromuscular endurance Self-reported reduction in endurance performance	Tier 2 club level FA endurance runners (n=16)[71] Tier 4 national level MA (n=5) and FA rowers (n=5)[279] Tier 4 FA endurance athletes (n=30)[184] Unspecified Tier FA athletes (n=1000)[54]
	Decreased $VO_{2\ max}$	Tier 3–4 FA endurance athletes (n=33)[284] Tier 3 MA road cyclists (n=50)[18] Tier 3 MA road cyclists (n=13)[106]
	Apparent underperformance in 60 min functional power threshold vs training load Decreased performance of 4000 m time trial Self-reported decrease in rowing performance	Tier 4 FA (n=8) and M (n=4) lightweight rowers[76]
Decreased power performance	Reduced velocity during 400 m swim time trial Decreased anaerobic (Wingate) performance Decreased number of throws in a Judo Specific Fitness Test Decreased performance of 15 s cycling sprint	Tier 3 junior national level FA swimmers (n=10)[168] Tier 2–3 MA bodybuilder (n=1)[50] Tier 2 MA second and third Dan black belt Judo athletes (n=11)[104] Tier 3 MA road cyclists (n=13)[106]

Each outcome can occur in the absence of LEA; therefore a differential diagnosis should always be considered in the assessment of REDs severity and/or risk.
*Tiering system according to McKay *et al*.[31]
FA, female athlete; MA, male athlete; PCr, phosphorylated creatine; VO_2 max, maximal oxygen consumption.

reliable body composition assessment methods and develop clear guidelines on how to interpret, manage, and communicate safely to athletic populations.[132]

Choosing an appropriate body composition assessment method involves consideration of its accuracy, repeatability, utility and cost. Some easy-to-use methods are 'doubly indirect', relying on regression equations to derive a body fat per cent; they do not provide valid data, use spurious assumptions and/or are influenced greatly by athlete presentation (eg, hydration levels).[136] Conversely, with operator training and sampling several sites, reliable assessments of subcutaneous adipose tissue thicknesses can be obtained via skinfolds (compressed and skin included) and brightness-mode (B-mode) ultrasound (uncompressed) method demonstrating good accuracy and sensitivity, especially for lean individuals.[137] Though costlier, DXA is a reliable method for assessing BMD and estimating fat and lean masses, provided standard test protocols are used.[138–140] In summary, using skinfolds, DXA, and B-mode ultrasound are the proposed body composition assessment methods available at the time of publication. For para athletes, adjustments of the assessment protocol and analysis of results may be needed. If that is impossible, the assessment should not proceed.

To minimise the risk of problematic LEA and DE behaviours, assessment of body mass and body composition is best conducted

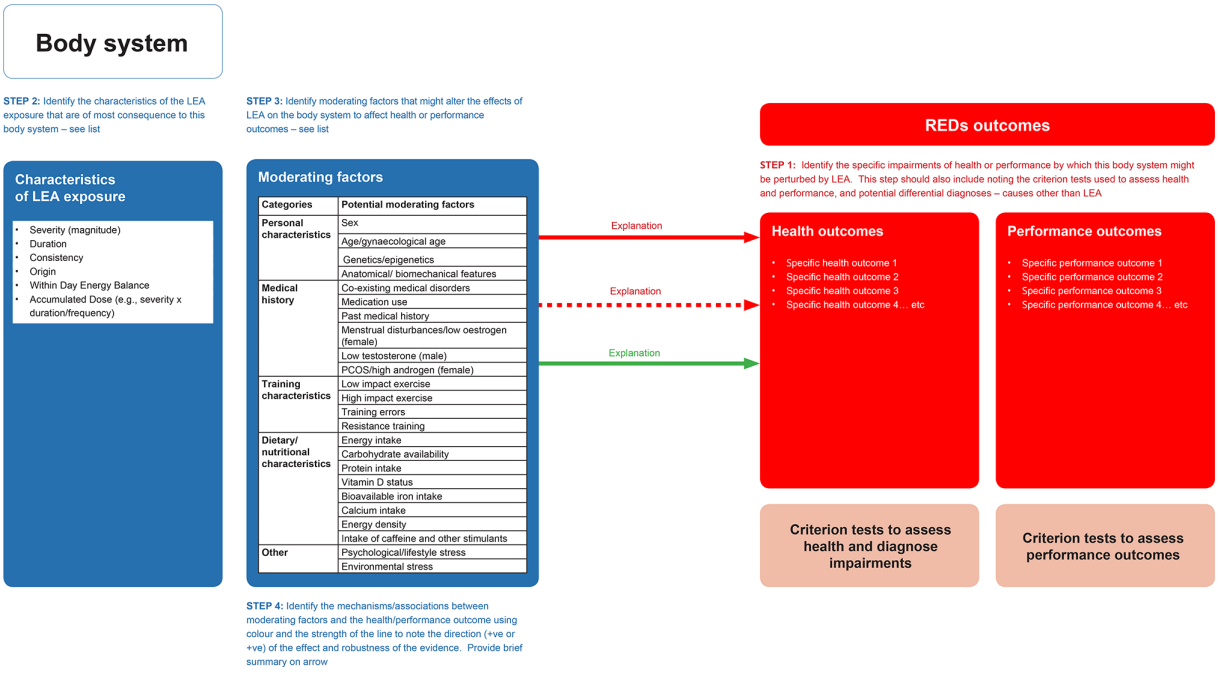

Figure 3 Integrated template of a clinical Physiological Model to show how problematic LEA 'exposure', with various associated moderating factors, can lead to various REDs 'outcomes', as represented by body system/health dysfunction(s) and potential performance impairment(s). This template outlines four steps to adapt and update the model as the future science of LEA/REDs evolves. Examples of moderating factors are also provided (step 3). LEA, low energy availability; REDs, Relative Energy Deficiency in Sport.

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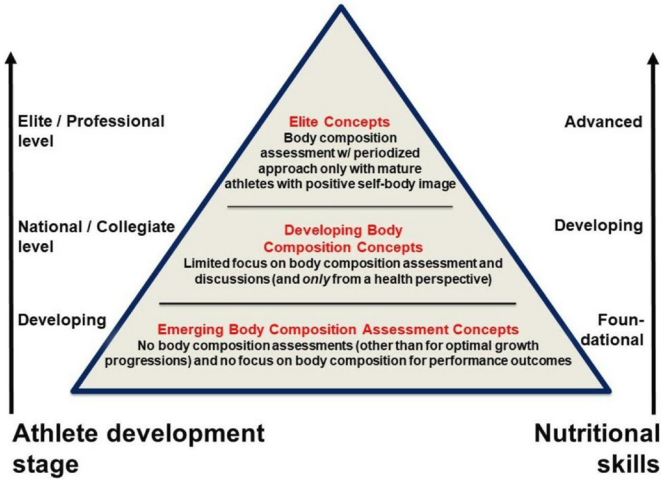

Figure 4 A conceptual framework on the implementation of body composition assessments (eg, height, weight, anthropometrics, skinfolds) within the context of athlete stage of development and their nutritional preparation skills[132] (reprinted with permission from BJSM).

by the athlete health and performance team who are trained in the specific methods and are competent to support the athlete and coach in making informed 'health first–performance second' decisions relating to body composition manipulation.[26 132] This should include prescreening to assess body image concerns and problematic eating behaviours, as well as implementing appropriate dietary interventions and subsequent athlete monitoring. Finally, body composition data are considered health data and must be kept confidential with appropriate levels of data protection. Accordingly, each body composition assessment and outcome report requires athlete informed consent and should only be shared with those the athlete authorises to be privy to the results.[68]

IOC REDs Clinical Assessment Tool-Version 2 (IOC REDs CAT2)

Significant scientific progress in REDs severity and risk assessment has been made since the original IOC REDs Clinical Assessment Tool (CAT) was published in 2015.[141] Because problematic LEA is the underlying aetiology for the health and performance outcomes of REDs, various LEA indicators (signs and symptoms) have emerged as the current best practice for clinical assessment and research purposes. These indicators underpin the new IOC REDs CAT2[25] (figures 5 and 6, and tables 4 and 5), which has undergone internal expert voting statement validation (see online supplemental files 2–4) and external REDs expert clinical cross-agreement validation.[25]

The IOC REDs CAT2 consists of a three-step process (figure 5): *Step 1*: implementation of population-specific validated REDs Screening Questionnaire(s) and/or clinical interviews, which are less sensitive and objective but inexpensive and easy to implement for the initial identification of athletes at risk; *Step 2*: implementation of the IOC REDs CAT2 Severity/Risk Assessment (tables 4 and 5) and Stratification with Sport Participation Guidelines (figure 6). These tools are based on accumulating various primary and secondary risk indicators (eg, biomarkers, BMD, injury history (tables 4 and 5), resulting in the stratification of an athlete's severity and risk as either green, yellow, orange or red light; and *Step 3*: an expert physician diagnosis including a treatment plan ideally integrating a collaborative multidisciplinary team (see Definitions box 2).

The IOC REDs CAT2[25] introduces a four-colour traffic-light severity/risk categorisation, in contrast to the three-colour stratification in the 2015 RED-S CAT,[141] due to the appreciation that the 2015 yellow zone had an extensive clinical severity/risk range of very low (a few minor symptoms) to very high (a few indicators away from removal from sport). Furthermore, each REDs traffic-light outcome is associated with varying severity/risk and sport participation recommendations (figure 6), ranging from full participation in training and competition (green) to continued monitoring (yellow) to intensive medical interventions and monitoring (orange) all the way to full medical support coupled with consideration for removal from competition and training (red). The IOC REDs CAT2 also provides a more concrete scientific framework and, where scientifically supported, a scoring system identified for each indicator. It is important to note that despite diagnostic progress, there is no singular validated diagnostic method for REDs, as the syndrome has a complex mosaic of signs and symptoms, necessitating the exclusion of other potential aetiologies in the differential diagnosis for each REDs indicator. Over time, the IOC REDs CAT2 will be modified to reflect advances in scientific knowledge and feedback from widespread utilisation.

Prevention and treatment of REDs

Primary and secondary prevention of REDs

Primary prevention includes tackling inadequate awareness and knowledge of the health and performance sequelae of REDs

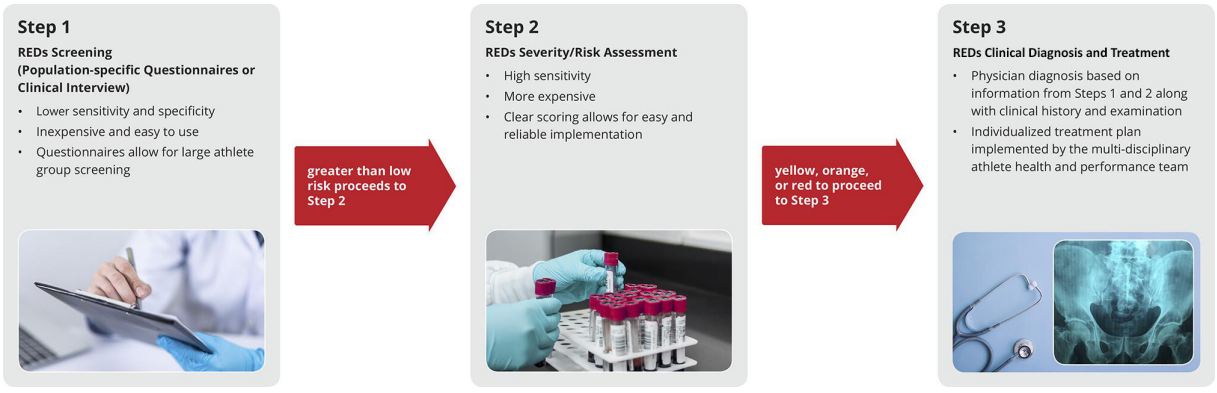

Figure 5 The IOC REDs CAT2 three-step protocol including: Step (1) screening; Step (2) severity and risk assessment and stratification; and Step (3) clinical diagnosis and treatment. CAT, Clinical Assessment Tool; IOC, International Olympic Committee; REDs: Relative Energy Deficiency in Sport.

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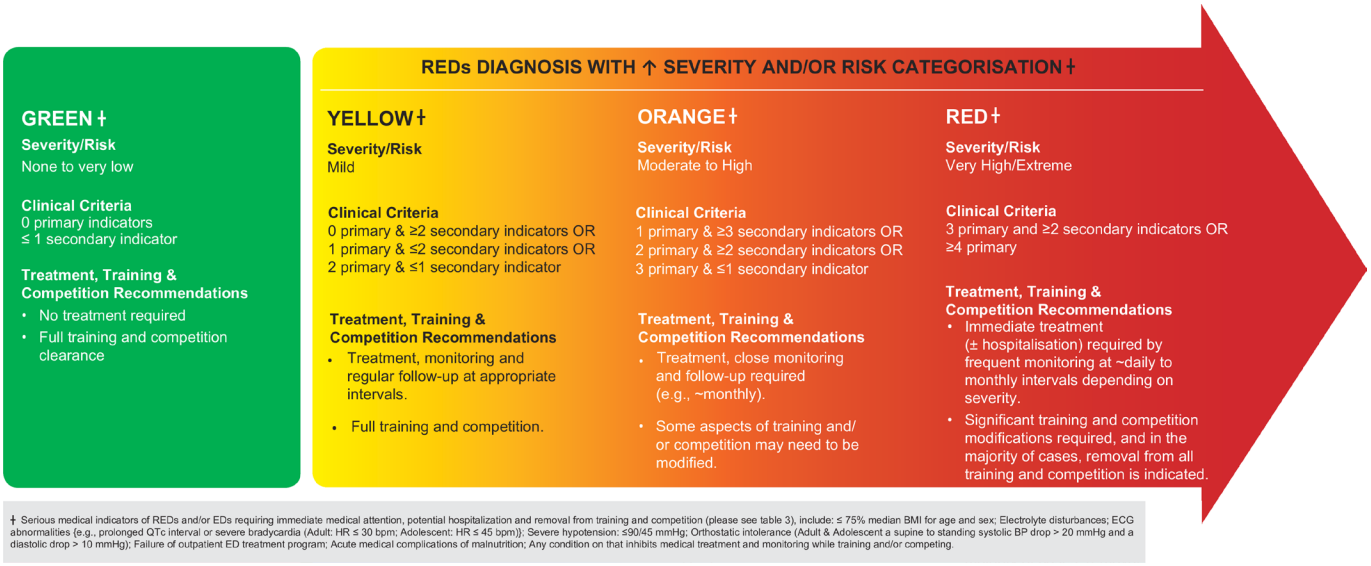

Figure 6 IOC REDs CAT2 Severity/Risk stratification with sport participation guidelines implementing the associated IOC REDs Severity/Risk Assessment tool (see table 4), with varying clinical management recommendations. Please see online supplemental file 5 for the IOC REDs CAT2 scoring tool. *Disclaimer*: these guidelines are not to be used in isolation and are not to be solely used for diagnosis. Furthermore, these guidelines are less reliable when it is impossible to assess all indicators in table 4. These guidelines are not a substitute for professional clinical diagnosis, advice and/or treatment from a team of REDs health and performance experts led by a physician. along with the evaluation of health status presented here, Severity/Risk stratification and sport participation decisions need to be made in the context of various decision modifiers, such as performance level of the athlete, sport type, participation risk, conflict of interest, athlete/coach pressures, timing and season.[285]
bpm, beats per minute; BMI, body mass index; bp, blood pressure; ECG, electrocardiogram; EDs, eating disorders; HR, heart rate; REDs, Relative Energy Deficiency in Sport.

and sports nutrition among athletes[113 142–144] and their entourage (eg, coaches,[145–147] parents, athlete health and performance team).[142 148 149] For example, less than half of coaches and physicians surveyed were able to identify the three components of the female athlete triad[147 148 150 151]; other studies reported similar knowledge gaps among physiotherapists and athletic trainers.[142 145] Short-term education programmes, using various delivery methods and focusing on factors associated with EDs, DE behaviours, and REDs have been shown to improve nutritional knowledge and reduce signs of dieting and body image concerns in female and male athletes.[70 152–158] Furthermore, early identification of symptoms using screening instruments, individual health interviews and objective assessment of REDs biomarkers may be useful as secondary prevention.[25] However, the REDs education and behaviour modification research field is underdeveloped, and specific REDs education programmes targeting athletes and other key personnel require further exploration and validation.[27]

Treatment (tertiary prevention) principles of REDs

Clinical treatment of diagnosed REDs cases (risk stratified in the yellow, orange and red light) should prevent further long-term health and performance sequelae,[27] sometimes requiring adjuvant treatment of body system dysfunction(s) (eg, low BMD, GI dysfunction, depression (see figures 1 and 2)) while reversing problematic LEA and its various underpinning causes.[69] The primary approach to treating REDs should be a restoration of optimal EA via non-pharmacological approaches, including changes to diet and exercise to achieve sustained optimal EA with appropriate contributions of macronutrients and micronutrients.[159]

Studies of LEA exposure have identified a somewhat more prominent effect of poor EI, rather than excessive EEE, in causing most of the physiological perturbations.[127 160] Long-term, well-controlled dietary and/or exercise intervention studies of REDs are needed, but numerous practical and methodological challenges exist. Indeed, in the one intention-to-treat 12-month, randomised controlled clinical trial that implemented dietary changes to increase EI in exercising females with REDs-related biomarkers, there was a high drop-out rate (57%), and improvement in some (eg, menstrual function resumption in select participants),[161] but not all symptoms (eg, inability to retard bone loss).[162] Such findings may indicate that optimal dietary interventions are not yet identified, dietary changes are difficult to accept or implement, various REDs sequelae improve at different rates, the dose of LEA may influence time to recovery, or a combination of these and other factors.

There are some useful pharmacological and psychological approaches emerging to treat clinical issues associated with REDs.[27] One example is 17β-oestradiol transdermal patch continuously with cyclic oral micronised progesterone administration, which demonstrated increased BMD Z-scores at the spine (2.75%), femoral neck (5.25%) and total hip (1.85%) at the end of a 12-month intervention in oligo-amenorrhoeic endurance athletes; those randomised to combined oral contraceptive pills (ethinyl oestradiol and desogestrel) or no treatment had inferior BMD results.[163]

A comprehensive team approach of the athlete health and performance team, including sports medicine, nutrition, psychology and sports science personnel, together with coach and family engagement is recommended. The team approach is especially important in athletes with severe REDs stemming from DE behaviours or EDs.[27 68 164] Treatment goals should ensure safe sport participation while undergoing long-term treatment and monitoring, including risk stratification to assess the safety of continued sports participation.

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Table 4 IOC REDs CAT2 Severity/Risk Assessment tool that implements primary, secondary and potential indicators into a traffic-light criterion outlined in figure 6

REDs indicator	References
Severe primary indicators (count as 2 primary indicators)	
Primary amenorrhoea (*females*: primary amenorrhoea is indicated when there has been a failure to menstruate by age 15 in the presence of normal secondary sexual development (two SD above the mean of 13 years), or within 5 years after breast development if that occurs before age 10); or prolonged secondary amenorrhoea (absence of 12 or more consecutive menstrual cycles) due to FHA	6 141 286–288
Clinically low free or total testosterone (*males:* below the reference range)	49 92 121 289–291
Primary indicators	
Secondary amenorrhoea (*females*: absence of 3–11 consecutive menstrual cycles) caused by FHA	6 141 286 287
Subclinically low total or free testosterone (*males*: within the lowest 25% (quartile) of the reference range)	49 92 95 121 289–291
Subclinically or clinically low total or free T3 (within or below the lowest 25% (quartile) of the reference range)	49 219 290
History of ≥1 high-risk (femoral neck, sacrum, pelvis) or ≥2 low-risk BSI (all other BSI locations) within the previous 2 years or absence of ≥6 months from training due to BSI in the previous 2 years	206 286 292
Pre-menopausal females and males <50 years old: BMD Z-score* <−1 at the lumbar spine, total hip or femoral neck or decrease in BMD Z-score from prior testing *Children/adolescents*: BMD Z-score* <−1 at the lumbar spine or TBLH or decrease in BMD Z-score from prior testing (can occur from bone loss or inadequate bone accrual)	119 120 123 293
A negative deviation of a paediatric or adolescent athlete's previous growth trajectory (height and/or weight)	294 295
An elevated score for the EDE-Q global (>2.30 in females; >1.68 in males) and/or clinically diagnosed DSM-5-TR-defined Eating Disorder (*only one primary indicator for either or both outcomes*)	68 80 276 296–298
Secondary indicators	
Oligomenorrhoea caused by FHA (>35 days between periods for a maximum of 8 periods/year)	6 141 286 287
History of 1 low-risk BSI (see high vs low-risk definition above) within the previous 2 years *and* absence of <6 months from training due to BSI in the previous 2 years	206 286 292
Elevated total or LDL cholesterol (above reference range)	191 235 299
Clinically diagnosed depression and/or anxiety (*only one secondary indicator for either or both outcomes*)	296 300 301
Potential indicators (not scored, emerging)††	
Subclinically or clinically low IGF-1 (within or below the lowest 25% (quartile) of the reference range)	11 168 290
Clinically low blood glucose (below the reference range)	11 80
Clinically low blood insulin (below the reference range)	45 127 290
Chronically poor or sudden decline in iron studies (eg, ferritin, iron, transferrin) and/or haemoglobin	169 302–304
Lack of ovulation (via urinary ovulation detection)	287 305–307
Elevated resting AM or 24-hour urine cortisol (above the reference range or significant change for an individual)	45 127 179 290
Urinary incontinence (*females*)	230 308 309
GI or liver dysfunction/adverse GI symptoms at rest and during exercise	8 214 310
Reduced or low RMR <30 kcal/kg FFM/day or RMR ratio <0.90	9 219 311 312
Reduced or low libido/sex drive (especially in males) and decreased morning erections	108–111
Symptomatic orthostatic hypotension	294 313 314
Bradycardia (HR <40 in adult athletes; HR <50 in adolescent athletes)	294 295 313
Low systolic or diastolic BP (<90/60 mm Hg)	315 316
Sleep disturbances	50 76 317
Psychological symptoms (eg, increased stress, anxiety, mood changes, body dissatisfaction and/or body dysmorphia)	8 68 296 300 301 318
Exercise dependence/addiction	68 80 319 320
Low BMI	286 294 295

Continued

Table 4 Continued

REDs indicator	References
Every indicator above requires consideration of a non-LEA-mediated differential diagnosis. All indicators apply to females and males unless indicated. Menstrual cycle status and endogenous sex hormone levels cannot be accurately assessed in athletes who are taking sex hormone-altering medications (eg, hormone-based contraceptives), and thyroid hormone status indicators cannot be accurately assessed in athletes who are taking thyroid medications. All laboratory values should be interpreted in the context of age-appropriate and sex-appropriate and laboratory-specific reference ranges. Most REDs data and associated thresholds have been established in premenopausal/andropausal adults unless indicated. *Disclaimer*: this tool should not be used in isolation nor solely for diagnosis, as every indicator requires clinical consideration of a non-LEA-mediated differential diagnosis. Furthermore, the tool is less reliable in situations where it is impossible to assess all indicators (eg, menstrual cycle status in females who are using hormonal contraception). This tool is not a substitute for professional clinical diagnosis, advice and/or treatment from a physician-led team of REDs health and performance experts Adolescent refers to <18 years of age. *BMD assessed via DXA within ≤6 months. In some situations, using a Z-score from another skeletal site may be warranted (eg, distal 1/3 radius when other sites cannot be measured or including proximal femoral measurements in some older (>15 years) adolescents for whom longitudinal BMD monitoring into adulthood is indicated).[119 321] A true BMD decrease (from prior testing) is ideally assessed in comparison to the individual facilities DXA's LSC based on the facilities calculated coefficient of variation (%CV). As established by ISCD, at the very least, LSC should be 5.3%, 5.0% and 6.9% for the spine, hip and femoral neck to detect a clinical change.[120 321] †Potential indicators are purposefully vague in quantification, pending further research to quantify parameters and cut-offs more accurately. BMD, bone mineral density; BMI, body mass index; BP, blood pressure; BSI, bone stress injuries; DSM-5-TR, Diagnostic and Statistical Manual of Mental Disorders, fifth edition, text revision; DXA, dual-energy X-ray absorptiometry; EDE-Q, Eating Disorder Examination Questionnaire; FFM, fat-free mass; FHA, functional hypothalamic amenorrhoea; GI, gastrointestinal; HR, heart rate; IGF-1, insulin-like growth factor 1; ISCD, International Society for Clinical Densitometry; LDL, low-density lipoprotein; LSC, least significant change; RMR, resting metabolic rate; T3, triiodothyronine; T, testosterone; TBLH, total body less head.	

REDs research methodology guidelines

Although the seminal REDs research implemented randomised clinical trials with strict laboratory-controlled EA interventions in habitually sedentary females,[45 125 127 160 165] most of the research since has involved cross-sectional study designs investigating the prevalence of various LEA indicators (indirectly via questionnaires or directly via indicators).[8 11 21 49 78 166 167] While results have confirmed the aetiology of REDs is problematic LEA, findings also show significant individualised responses concerning the type, prevalence and severity of the impairments of various body systems associated with this exposure,[8 11 49 78 166 167] as well

Table 5 Serious medical indicators of REDs and/or EDS requiring immediate medical attention, potential hospitalisation and removal from training and competition (adapted from ED clinical management recommendations, paediatric and adult ED papers and athlete cardiovascular health consensus papers.[294 295 313 315 316 322 323]
Disclaimer: this list should not be used in isolation and should be based on a thorough clinical assessment that considers the severity of the athlete's physical and mental health.

Serious medical indicators
- ≤75% median BMI for age and sex
- Electrolyte disturbances (eg, hypokalaemia, hyponatraemia, hypophosphataemia)
- ECG abnormalities (eg, prolonged QTc interval or severe bradycardia (adult: HR≤30 bpm; adolescent: HR≤45 bpm))
- Severe hypotension: ≤90/45 mm Hg
- Orthostatic intolerance (adult and adolescent: a supine to standing systolic BP drop>20 mm Hg and a diastolic drop>10 mm Hg)
- Failure of outpatient ED treatment programme
- Acute medical complications of malnutrition (eg, syncope, seizures, cardiac failure, pancreatitis)
- Any condition that inhibits medical treatment and monitoring while training and/or competing

BMI, body mass index; BPM, beats per minute; ECG, electrocardiogram; ED, eating disorder; HR, heart rate; QTc, corrected QT.

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Box 2 Definitions - IOC REDs-Clinical Assessment Tool-2 (IOC REDs CAT2)

REDs CAT primary indicators
Outcome parameters most consistently resulting from problematic LEA leading to REDs signs and/or symptoms identified in the scientific literature and/or with the greatest measurement validity (ie, sensitivity, specificity) and/or indicative of increased severity and risk of REDs. Accordingly, these indicators hold the most evidence and impact in the overall IOC REDs CAT2 Severity/Risk Assessment and Stratification Tool.

REDs CAT secondary indicators
Outcome parameters with some scientific evidence, resulting from problematic LEA leading to REDs signs and/or symptoms identified in the scientific literature and/or with lower measurement validity (ie, sensitivity, specificity) and/or have shown less severity and risk of REDs. Accordingly, these indicators hold a secondary level of evidence and impact in the overall IOC REDs CAT2 Severity/Risk Assessment and Stratification Tool.

REDs CAT potential indicators
Emerging outcome parameters lacking robust scientific evidence but may possibly be linked to problematic LEA leading to REDs signs and/or symptoms. These parameters generally demonstrate many of the following:
⇒ poor and/or inconsistent evidence
⇒ lack of existing validated screening tool, including a lack of validated cut-offs or thresholds in athletes
⇒ poor measurement validity (ie, sensitivity, specificity or high variability)
⇒ high cost and/or poor global availability
Accordingly, these indicators are listed as supportive in the Severity/Risk Assessment of REDs but are not directly involved in the IOC REDs CAT2 Severity/Risk Assessment and Stratification Tool. Potential indicators may move up to secondary or primary designation or off any list, pending more research validity and/or improved availability and/or cost.

REDs symptoms
Any REDs primary, secondary or potential indicator parameter(s) that an athlete directly reports or experiences (eg, pain from a BSI, amenorrhoea, depression, hunger, low libido, performance and training plateaus or declines) in the IOC REDs CAT2 Severity/Risk Assessment and Stratification Tool.

REDs signs
Any REDs primary, secondary or potential indicator parameter(s) that a clinician identifies on the IOC REDs CAT2 Severity/Risk Assessment Tool. A REDs sign may also be a significant individual change in a primary, secondary or potential indicator from the athlete's baseline within the context of REDs, with or without athlete symptoms (eg, a significant change in sex hormones, resting metabolic rate, cholesterol). *Note*: some indicators can be both signs and symptoms (eg, amenorrhoea).

IOC REDs CAT2 Severity/Risk Assessment and Stratification with Sport Participation Guidelines
A clinical tool to assist with identifying the current severity and/or the future risk of REDs that is comprised of an accumulation of primary and secondary indicators of REDs. The IOC REDs CAT2 Severity/Risk Stratification with Sport Participation Guidelines identifies the severity and/or risk of REDs for a given athlete along a spectrum characterised by a traffic light continuum from healthy (green) to mild (yellow), to moderate (orange), to severe (red), and provides sport participation guidelines for each level.

REDs diagnosis
A diagnosis of REDs results from the clinical assessment by a physician with expertise in REDs, using information collected from a multidisciplinary team (eg, sports medicine physician, sports dietitian, sports physiologist, sports psychologist/psychiatrist), which ideally includes: (1) appropriately validated questionnaires and/or clinical interview; (2) physical assessment; and (3) laboratory and imaging data as indicated in the IOC REDs Severity/Risk Assessment and Stratification Tool. A REDs diagnosis is predicated on excluding other aetiologies in the differential diagnosis for each REDs indicator and ranges from yellow to orange to red severity/risk.

as a lack of a universal EA threshold below which problems are observed.[88] Cross-sectional studies are useful for clinical REDs assessment and prevalence, but an analysis of this literature reveals multiple limitations (eg, lack of a classification of subject calibre/training status; lack of a standardisation of recruitment and assessment protocols; poor characterisation of menstrual status and hormonal contraceptive use; varied use of indicators of physiological, hormonal and performance status; and poor or non-existent assessment of EA). It is noted that there are few prospective or cohort studies in which groups of athletes with and without signs of LEA have been monitored longitudinally to note changes in health and performance.[168 169] Finally, there is also a need for controlled intervention studies in which EA manipulations are implemented with rigorous designs and careful assessment of the dose–response, time-course and variability in the development of perturbations to body systems and functional impairments.[46–48 53 54 170 171] By the triangulation of data from these various approaches (cross-sectional/longitudinal/interventional studies), the complexity of the relationship between LEA and REDs can be realised. It is recommended that future REDs research be conducted using standardised methodology to provide more accurate insights and to facilitate cross-study comparisons.[21]

Table 6 summarises methods that are considered to be preferred techniques for assessing health and performance outcomes associated with REDs, as well as others that do not reach that criterion but are commonly used *and* considered acceptable in terms of validity (ie, variability and precision) and feasibility (eg, availability, cost). Some tests have standards and diagnostic criteria for what is considered 'normal' versus 'impaired'. Meanwhile, the assessment of other features provides quantitative data that can be compared over time or between individuals and interpreted with consideration of the known precision/errors of measurement.

CONCLUSION

As evidenced by this consensus statement, there have been numerous scientific advances in the field of REDs since the publication of the 2018 IOC consensus update statement[6]: from new scientific concepts around our understanding of the evolution of various REDs signs and symptoms to the development of a Physiological Model depicting the nuanced complexity of how

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Table 6 Methods (preferred, used and recommended, and potential) for studying various health and performance outcomes of REDs

Health outcome	Methods and notes
Impaired reproductive function	*Preferred* ► Overnight sampling of LH and FSH[324] ► Menstruating females: phase-based hormonal approach using urinary ovulation kits (testing mid-cycle LH surge) and blood sampling[287] ► Postpubertal males: morning total and free testosterone level[325 326] *Used and recommended* ► Females: self-reported menstrual history, urinary ovulation testing,[287 327] LEAF-Q[171] ► Males: self-reported libido/morning erection (eg, LEAM-Q[328] or ADAM-Q[111 329]) *Potential*
Impaired bone health	*Preferred* ► DXA[123 330] – Using age-appropriate and medically appropriate body-site scanning[330] – Using age-appropriate, sex-appropriate and activity-appropriate interpretation (eg, Z-score vs T-score) *Used and recommended* ► Bone stress injury and fracture history *Potential* ► HRpQCT
Impaired gastrointestinal function	*Preferred* ► Oesophageal motility: oesophageal manometry, barium swallow ► GERD: upper endoscopy ► Gastric motility: electrogastrography[331 332] ► Gastroparesis: gastric emptying study ► Pancreatitis: ≥2 of: (a) lipase >3× upper limit of normal; (b) imaging findings consistent with pancreatitis; (c) characteristic epigastric pain ► Intestinal transit: radiopaque marker study,[333] orocaecal transit time test[334 335] ► SMA syndrome: upper GI oral contrasted study, MRI or CT[336–338] *Used and recommended* ► GERD: many questionnaires,[339] including GerdQ[340] ► Constipation: Wexner Constipation Score,[341] Bristol Stool Scale[342] ► Diarrhoea: Bristol Stool Scale[342] ► Irritable bowel syndrome: Rome IV Criteria[343] Elevated transaminases[344 345] ► Defecatory disorders, faecal incontinence[346]: Faecal Incontinence Questionnaire,[8 347] Faecal Incontinence Severity Index (FISI),[348] Altomare's Obstructed Defecation Scale (ODS) score[349] ► Multiple GI symptoms: Rome II questionnaire[350] ► GI symptoms during exercise[351 352] ► LEAF-Q GI subsection score ≥2 indicative of LEA[214 353] ► Athlete-specific GI symptom inventory[354] ► Feeding challenge during exercise[334 355] *Potential* ► Intestinal transit: wireless motility capsule ► Gut bacterial profile ► Faecal or plasma short-chain fatty acid concentration
Impaired energy metabolism/regulation	*Preferred* ► Thyroid function tests: TSH, free T4, total and free T3[165] ► Leptin: overnight sampling[356] ► Cortisol: overnight sampling,[179] 24-hour urinary free cortisol[357] ► Laboratory/expert-controlled measurements/estimates of all compartmentalised energetic intakes and total daily expenditures (exercise, non-exercise activity, basal metabolic rate, thermic effect of food)[358] *Used and recommended* ► Cortisol: morning serum cortisol, late-night salivary cortisol[357] ► RMR: indirect calorimetry,[359] room calorimetry[311]
Impaired haematological status	*Preferred* ► CBC with differential ► Iron studies (iron, ferritin, transferrin, total iron binding capacity) with age-appropriate, sex-appropriate and laboratory-appropriate cut-offs ► Carbon monoxide haemoglobin mass measurement[360 361] *Used and recommended* ► Self-reported history of iron deficiency or anaemia *Potential* ► App-based self-assessment[362]
Urinary incontinence	*Preferred* ► Stress urinary incontinence: bladder stress test[363] ► International Consultation on Incontinence-Urinary Incontinence Short Form (ICIQ-UI-SF)[230 231] ► 3 Incontinence Questionnaire (3IQ)[364] *Potential* ► Pelvic Floor Dysfunction-ScrEeNing Tool IN fEmale athLetes (PFD-SENTINEL)[365]
Impaired glucose and lipid metabolism	*Preferred* ► Fasting blood glucose (serial measures)[366] ► Fasting insulin[366] ► Lipid panel: HDL, LDL, total cholesterol, triglycerides[299] *Used and recommended* ► Continuous glucose monitor[367]
Mental health issues	*Preferred* ► Clinical interview with psychiatrist or psychologist, DSM-5-TR[368] *Used and recommended* ► Depression: PHQ,[369] Centre for Epidemiological Studies Depression Scale,[370] Beck Depression Inventory[371] ► Generalised anxiety: GAD-7,[164 372] DASS-21[78 269 373 374] ► Stress: Perceived Stress Scale[375] ► Brunel Mood Scale[376] ► Profile of Mood States[377 378] ► Eating disorders: EDE-Q,[379–381] BEDA-Q,[382] Eating Disorder Inventory,[383] self-report
Impaired neurocognitive function	*Preferred* ► Clinical neuropsychological assessment *Used and recommended* ► Multiple domains: CogState assessment battery[384] ► Planning/cognitive flexibility: Wisconsin Card Sorting Test[242] ► Attention: Stroop Colour and Word Test[385–387] ► Decision making: Iowa Gambling Test[388 389] ► Verbal memory: California Verbal Learning Test-II[390] ► Executive function: Delis-Kaplan Executive Function System Color-Word Interference Test,[238] BRIEF-A[391]

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Table 6 Continued

Health outcome	Methods and notes
Sleep disturbances	*Preferred* ► Polysomnography[392] *Used and recommended*[392] ► Research-grade actigraphy ► Sleep diaries ► Numerous questionnaires, including Athlete Sleep Screening Questionnaire (ASSQ),[393] Athlete Sleep Behaviour Questionnaire (ASBQ),[394] Epworth Sleepiness Scale,[395] Pittsburgh Sleep Quality Index,[10 396] Insomnia Severity Index[164 397] *Potential* ► Sport wearables[398]
Impaired cardiovascular function	*Preferred* ► Conduction, rhythm abnormalities: ECG[313] ► Rate abnormalities: cardiac telemetry, Holter monitor ► Haemodynamics: sphygmomanometry, orthostatic sphygmomanometery (≥20 mm Hg drop in systolic pressure, ≥10 mm Hg drop in diastolic pressure on standing from supine)[313 399] ► Autonomic function: heart rate variability by Holter monitor,[400 401] baroreflex sensitivity testing,[402] bedside tests (eg, Valsalva, tilt testing) ► Structural abnormalities: transthoracic echocardiogram[313] ► Endothelial dysfunction: brachial artery flow-mediated dilatation[235 403] *Used and recommended* ► Heart rate: chest-mounted electrode-containing heart rate strap[404 405] ► Haemodynamics: self-reported episodes of orthostatic (pre-) syncope *Potential* ► Sport wearables[398 406]
Reduced skeletal muscle function	*Preferred* ► Muscle protein synthesis: isotopic amino acid labelling,[407] deuterated water ingestion[408 409] ► Muscle glycogen content: histochemical analysis of biopsy-derived muscle samples,[410] ^{13}C-magnetic resonance spectroscopy[48 411] *Used and recommended* ► None–exclude assessment if unable to directly measure as above
Impaired growth and development	*Preferred* ► Paediatric patients: clinical assessment with growth charts – Deviation from baseline growth trajectory, defined as a dynamic change with time (vs a single measurement) – Decrease in growth Z-score by >1[294 412] ► Growth hormone: overnight sampling[413] ► IGF-1: serum levels, IGFBP-3 levels[414] *Used and recommended* ► Paediatric patients: delayed markers of puberty (thelarche, menarche, spermarche)
Reduced immunity	*Preferred* ► To be determined *Used and recommended* ► Self-reported illness frequency[10 271 415] *Potential* ► CBC, with differential, immunoglobulin G glycome, leucocyte transcriptome and cytokine profile[272]
Performance outcome	Methods and notes
Decreased athlete availability	*Preferred* ► Self-reported days of training/competition lost or modified due to illness or injury[10 274 416]
Decreased training response	*Preferred* ► Longitudinal tracking of valid performance-related metric specific to athlete/sport (eg, sport-related time trial)[168 417 418] *Used and recommended* ► Self-reported plateauing of ability/performance despite training progression[419] ► Exercise lactate profile[420 421] ► Lactate: RPE ratio[422 423] ► Catecholamine concentrations[424]
Decreased recovery	*Preferred* ► To be determined *Used and recommended* ► Lab-based studies: – Creatine phosphate system: ^{31}P magnetic resonance spectroscopy[425] – Exercise-induced muscle damage: muscle biopsy[426] ► Field-based studies: – Questionnaires: Recovery-Stress Questionnaire (REST-Q),[10 427] self-reported perceptions of recovery, Profile of Moods State (POMS),[377] Hooper MacKinnon Questionnaire[428] – Creatine kinase (total, muscle)[429] ► Athlete's subjective report of readiness[430] *Potential* ► Wearable/commercialised recovery/readiness algorithms[431]
Decreased cognitive performance/skill	*Preferred* ► Skill: sport-specific measures (eg, Loughborough Soccer Passing Test)[432 433] *Used and recommended* ► Reaction time: consider sport-specific tests[434] ► Spatial awareness: mental rotation test[241]
Decreased drive/motivation	*Preferred* ► To be determined *Used and recommended* ► Motivation: Behavioural Regulation in Sport Questionnaire (BRSQ),[435] Psychological Need States in Sport-Scale (PNSS-S)[436] ► Athlete Burnout Questionnaire (ABQ)[437] ► Maslach Burnout Inventory[438]
Decreased muscle strength	*Preferred* ► Longitudinal tracking of valid performance-related metric specific to athlete/sport (eg, sport-related strength test, such as snatch or clean and jerk for weightlifting, or throw distance for shot put)[439] *Used and recommended* ► Isokinetic dynamometry[440 441] ► One repetition maximum, specific movement (eg, bench press)[442 443]

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Table 6 Continued

Performance outcome	Methods and notes
Decreased endurance performance	*Preferred* ► Longitudinal tracking of valid performance-related metric specific to athlete/sport (eg, sport-related time-trial)[168 417 418] *Used and recommended* ► Laboratory-based VO_2 max testing (via indirect calorimetry)[444] ► Laboratory-based lactate threshold testing[445] ► Multistage shuttle run[446 447] ► Cycling ramp test[448]
Decreased power performance	*Preferred* ► Wingate test[449] *Used and recommended* ► Counter-movement jump[73] ► Standing broad jump[450 451] ► Bosco test[452 453]

*While various methods have been used clinically and in research settings, many have not been validated or used in athletes or specifically used to assess the effects of REDs. Therefore, this table proposes methods that have been used for outcomes of interest and that the authors recommend to date.
ADAM-Q, Androgen Deficiency in Ageing Males Questionnaire; BEDA-Q, Brief Eating Disorder in Athletes Questionnaire; BRIEF-A, Behaviour Rating Inventory of Executive Function–Adult Version; CBC, complete blood count; DASS-21, Depression Anxiety Stress Scale-21; DSM-5 TR, Diagnostic and Statistical Manual of Mental Disorders– fifth edition, text revision; DXA, dual-energy X-ray absorptiometry; EDE-Q, Eating Disorder Examination Questionnaire; FSH, follicle stimulating hormone; GAD-7, General Anxiety Disorder-7; GERD, Gastro-oesophageal reflux disease; GerdQ, Gastro-oesophageal Reflux Disease Questionnaire; GI, gastrointestinal; HDL, high-density lipoprotein; HRpQCT, high-resolution peripheral quantitative computed tomography; IGF-1, Insulin-like growth factor 1; IGFBP-3, Insulin-like growth binding protein-3; LDL, low-density lipoprotein; LEA, low energy availability; LEAF-Q, Low Energy Availability in Females Questionnaire; LH, luteinising hormone; PHQ, Patient Health Questionnaire; RMR, Resting Metabolic Rate; RPE, rating of perceived exertion; SMA, superior mesenteric artery; T3, triiodothyronine; T4, thyroxine; TSH, thyroid stimulating hormone; VO_2 max, Maximal oxygen consumption.

LEA exposure (either problematic or adaptable), with associated moderating factors, leading to changes in health and/or performance outcomes in individual athletes. Our understanding of the outcomes of problematic LEA exposure causing REDs on athlete mental health and in male athletes has also been further refined.

In addition to the scientific advances, we have presented a summary of practical clinical guidelines for assessing LEA and for safe body composition measurement. We have also reviewed the scientific literature on the prevention and treatment of REDs and introduced an updated, validated IOC REDs CAT2 to aid in diagnosis and Severity/Risk Assessment. Finally, by providing standardised guidelines for research methodology, we look forward to high-quality REDs research outcomes in the future. Most importantly, our work aims to stimulate action by sports organisations, sports scientists, and the athlete health and performance team to protect the health and well-being of the many athletes at risk for developing this syndrome.

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Correction: *2023 International Olympic Committee's (IOC) consensus statement on Relative Energy Deficiency in Sport (REDs)*

Mountjoy M, Ackerman KE, Bailey DM, et al. 2023 International Olympic Committee's (IOC) consensus statement on Relative Energy Deficiency in Sport (REDs). *Br J Sports Med* 2023;57:1073–1097

Figure 6 and online supplementary file 5 have been updated. This has been updated in the online version only and not in print.

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