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

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Scientific Contributions

All authors were involved in the conception, drafting, voting, in-person discussion, revising, and approval of the final manuscript prior to submission. MM was responsible for leading the consensus project and for coordinating the consensus statement manuscript. AUJ represented the athlete's voice, and DB represented the coach's voice. RB, UE and LE represented the International Olympic Committee's medical and scientific department.

Competing Interests

Margo Mountjoy is a Deputy Editor of the BJSM and a member of the BJSM IPHP Editorial Board.
Kathryn Ackerman is a Deputy Editor of the BJSM and an Associate Editor of the BJSM IPHP.
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Richard Budgett is the IOC Medical and Scientific Director
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Uğur Erdener is an IOC member and the Chair of the IOC Medical and Scientific Commission.

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ABSTRACT

Relative Energy Deficiency in Sport (REDs) was first introduced in 2014 by the International Olympic Committee's (IOC) 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.

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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)². 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:

$$EA \text{ [Energy Availability]} = \{EI \text{ [Energy Intake (kcal)]} - EEE \text{ [Exercise Energy Expenditure (kcal)]}\} / FFM \text{ [Fat-Free Mass (kg) / day]}^{2-4}$$

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 2014⁵, and was updated in 2018⁶. 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 appendix 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 to 79.5%⁷⁻¹⁶), and male (15% to 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

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(e.g., inaccurate EA measurements), variation in physiological demands amongst the study populations, and participant study volunteering biases^{21 22}.

Compared with previous REDs consensus statements, this updated IOC REDs consensus is more robust in its methodology including *i*) outlining criteria for consensus panel inclusion, thresholds for reaching consensus via voting statements, and the provision for dissent^{23 24}; *ii*) being supported by a dedicated edition of related reviews and editorials providing detailed context to facilitate further understanding^{22 25-31}; and *iii*) featuring a blend of science and knowledge translation (implementing an athlete- 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^{26 28 30}. 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 methodologies²². The outcomes of this consensus are focussed on the developing to world-class level athlete (Tiers 2-5)³².

The goals of this consensus statement are to *i*) summarise the recent scientific advances in the field of REDs; *ii*) introduce a novel REDs Physiological Model template and validated REDs Clinical Assessment Tool - Version 2 (IOC REDs CAT2); and *iii*) 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: A) What is REDs?, B) Methodology and Consensus Results, C) Key Scientific Advances since the 2018 REDs Consensus Statement, D) Clinical Applications, and E) Research Methodology Guidelines.

A. 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 circumstances³³⁻³⁵. In sports science literature, EA to meet various biological

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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 maintenance³³. In particular, such evolutionary selective pressures have favoured adaptations that allocate limited energy supplies during periods of LEA (e.g., famines) to biological processes that support immediate survival, as well as long-term reproductive success³³. Therefore, humans, like other animals, are adapted to cope with periods of LEA by downregulating biological processes that are temporarily unnecessary or reducible³³. Some of these perturbations to body systems might be considered mild and/or transient, representing physiological plasticity³⁶ 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 programs³⁷ or sports-related practices. This is especially the case in endurance sports (often >30 hours of training/week)³⁸, which can sometimes result in extreme exercise energy expenditure (EEE) that exceed the capacity of the human alimentary tract for sustained energy absorption³⁹. Indeed, the spectrum of exposure to LEA can include scenarios (e.g., significant duration, magnitude, frequency – see Definitions Box 1), that in conjunction with moderating factors (e.g., 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)⁴⁰. In the real world, athletes experience exposure to LEA (purposefully or inadvertently) in various manners along the continuum from adaptable to problematic^{3 41}. 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 (e.g., the implementation is guided by experts, the athlete has the physical and psychological readiness, adequate recovery is included, and health is maintained)^{42 43}.

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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”.

*****Insert Box 1 - LEA definitions here*****

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:

$$EA [Energy Availability] = \{EI [Dietary energy Intake (kcal)] - EEE [Exercise Energy Expenditure (kcal)]\} / FFM [Fat-Free Mass (kg) / day]^2 \text{ }^4$$

Low Energy Availability (LEA)

Low energy availability is any mismatch between dietary energy intake and energy expended in exercise that leaves the body's total energy needs unmet, i.e., there is inadequate energy to support the functions required by the body to maintain optimal health and performance⁶. Low energy availability occurs as a continuum between scenarios in which effects are benign (*adaptable LEA*) and others in which there are substantial and potentially longterm impairments of health and performance (*problematic LEA*)

Adaptable Low Energy Availability

Adaptable low energy availability 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 (e.g., monitored, and mindful manipulation of body composition or scheduled period of intensified training or competition) might be associated with acute health or performance benefits (e.g., increased relative VO_{2max}). Adaptable low energy availability 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 Low Energy Availability

Problematic low energy availability is exposure to low energy availability 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 low energy availability exposure (e.g., duration, magnitude, frequency) may vary according to the body system and

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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 low energy availability exposure on various body systems. Relevant moderating factors (e.g., gender, age, genetics) vary according to the body system. They may offer protection or additional risk in the progression from low energy availability 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 [e.g., self-induced restricting food intake, preoccupation with body shape or weight, bingeing, and purging (self-induced emesis, laxative use, excessive exercise, diuretic use)]⁴⁴.

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) 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 performance⁵.

B. 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 utilised the RAND-UCLA Appropriateness Method (RAM)⁴⁵.

A diverse (i.e., 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

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REDs. From the entire group of authors, smaller working groups of content experts were tasked with preparing specific sub-topics prior to the in-person consensus in the form of *i*) a referenced summary of the existing scientific literature and *ii*) 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 method⁴⁶). 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: $\geq 80\%$ of authors agreeing on the voting statement, without any author disagreeing.
- (2) Agreement with minority disagreement: $\geq 80\%$ 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 (appendices 2, 3 & 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 (i.e., 80% agreement was reached, but one or more individuals disagreed with the statement).

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

	Total	Agreement [∞]	Minority disagreement ^β	Disagreement ^φ
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: $\geq 80\%$ agree without disagreement but potentially includes “undecided” votes

^β Minority disagreement: $\geq 80\%$ agree but with one or more disagreeing opinions

^φ Disagreement: $<80\%$ agreement

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 4 continents participated.

C. 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 emerging themes are *i) the*

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additive impact of low carbohydrate availability (LCA) with LEA in the development of REDs; *ii*) the overlap of REDs and Overtraining Syndrome (OTS) symptomology; *iii*) the time-course of biomarker responses to problematic LEA in the development of REDs; *iv*) improved understanding of mental health associations of REDs; *v*) advances in knowledge pertaining to REDs in male athletes; and *vi*) para athletes.

The magnifying impact of low carbohydrate availability in the context of REDs

Most LEA intervention studies are also accompanied by a substantial reduction (25 to 60%, depending on magnitude of LEA) in carbohydrate (CHO) ingestion, resulting in concurrent LCA⁴⁷⁻⁵⁰. In the real world, the magnitude of LCA is likely to be even greater considering the emphasis on protein intake during periods of calorie restriction⁵¹⁻⁵³. 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^{54,55} with a concomitant impairment in biomarkers of bone formation⁵⁵, as well as increased post-exercise concentrations of interleukin-6 (IL-6) and hepcidin after LCA⁵⁶. 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 to only a 69% increase in hepcidin with isocaloric low energy but higher CHO diet⁵⁷. 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 3h post-exercise)⁵⁸, and elevated post-exercise IL-6 concentrations compared to an isocaloric high CHO treatment⁵⁹. Six studies since 2019 have shown an energy-independent and/or magnifying impact of LCA in the accelerated development of REDs outcomes⁵⁴⁻⁵⁹. 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.

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Symptomology overlap between REDs and Overtraining Syndrome

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^{38 60}. 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 OTS³⁸. 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^{38 61}.

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 22}. 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^{38 41}. 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 statements⁶²⁻⁶⁴ and prevalence studies⁶⁵⁻⁶⁷ 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 highlighted⁵, albeit less well understood

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^{6 36 68}. Recent qualitative studies^{1 69} involving mainly sub-elite endurance athletes provide support for this premise, reporting that LEA from intentional (e.g., weight regulation) or unintentional (e.g., 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 culture¹. 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 cohorts⁷⁰. LEA and DE behaviours, which exist along the spectrum between optimised nutrition and clinical EDs, may occur in isolation or together⁷⁰. A prior history of DE behaviours or an ED might perpetuate a continued under-fuelling of energy¹ 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 dissatisfaction⁷¹. Given the potentially serious outcomes of DE behaviours and EDs, prevention, early identification, and timely interventions should be prioritised^{62 72}.

Psychological indicators associated with problematic LEA and REDs are mood disturbances/fluctuations^{8 73 74}, cognitive dietary restraint⁷⁵, drive for thinness^{76 77}, reduced sleep quality,^{52 78} and perfectionistic tendencies⁷⁹. Depressive symptoms and affective disorders^{8 80 81}, subjectively reported reduced well-being⁷⁵, primary or secondary exercise dependence/addiction^{82 83}, anxiety related to injury and/or recovery, sport-specific issues such as difficulty coping with weight requirements^{69 78}, and the development of EDs^{1 84} 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 competition⁸⁵, as well as in athletes with physical disabilities⁸⁶. Furthermore, studies are required to *i*) ascertain why many athletes experience few or no negative mental health consequences in the early stages of problematic LEA exposure^{20 74 87} and *ii*) to better understand the reciprocal function of the different psychological variables^{88 89}. As perceived stress appears to be common for many mental health concerns

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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 athletes⁶².

REDs in male athletes

Although the 2014 IOC REDs consensus statement⁵ and the 2018 update paper⁶ 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 - 2022 include male athletes as subjects (see literature search summary in appendix 1).

While a universal cut-off of 30 kcal/kg FFM/d as a threshold of LEA leading to some REDs outcomes in females is debated⁹⁰, such a cut-off or range at which males experience REDs-related symptoms is even less understood,⁹¹ but appears to be lower (e.g., ~9 to 25 kcal/kg FFM/d)^{17 48 74 92 93}. 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^{74 94-102}; changes in metabolic hormones^{48 103-105}; impairments to immune function¹⁰⁶; detriments to bone health¹⁰⁷; as well as negative performance outcomes^{18 92 106 108} and decreased lean body mass accrual¹⁰⁹. 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 appears 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 LEA¹¹⁰⁻¹¹³.

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 athletes¹¹⁴. Among United States para athletes preparing for Paralympic Games, 62% attempted to alter weight or body composition

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to enhance performance, 32% had elevated scores on the Eating Disorder Examination Questionnaire (EDE-Q), and 44% of the female athletes reported menstrual dysfunction¹¹⁵. 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 study¹¹⁶. 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 (i.e., 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)¹¹⁷. Additionally, the presence of central neurological injury may result in alterations of the HPG axis and baseline menstrual function, regardless of energy status^{118 119}. 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 osteoporosis¹²⁰. 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 (e.g., Z-scores); normative, reference datasets are determined from measurements in able-bodied populations and stratified by age-, sex-, and limited race/ethnicity-matched categories to determine diagnostic cut-offs for “low BMD for age” and “osteoporosis”^{121 122}. 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 population¹¹⁴.

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.

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Undoubtedly, these outcomes occur over different timeframes and with different severity and significance to the individual athlete due to various moderating factors²⁵.

*****Insert Figures 1 and 2 here*****

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 (i.e. physiological plasticity^{2 4}, 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 of reproductive function and bone health in female and male athletes¹²³⁻¹²⁵. 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 below).

Table 2. Potential REDs health outcomes resulting from problematic 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).

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	<p>Females Alteration in LH concentrations or pulsatility Reduced oestrogen and progesterone Reduced testosterone Primary amenorrhea Oligomenorrhea/menstrual irregularities Secondary amenorrhea (FHA) Luteal phase defects/deficiency Anovulatory cycles</p> <p>Males Reduced testosterone Sperm abnormalities Erectile dysfunction</p> <p>Females and Males Decreased libido</p>	<p>SF^{47 126-129}, FA¹³⁰⁻¹³³ SF⁹⁰, FA^{134-138 139} FA¹³² FA^{140 141} SF⁹⁰, FA^{138 142 143} FA^{135 136 138 142 144 145} SF^{90 128}, FA¹⁴² SF⁹⁰, FA¹⁴²</p> <p>MA^{18 92 100 104 146-148} MA¹⁴⁹ MA^{83 110 113}</p> <p>MA^{110 113 149}</p>	<p>Females <i>Primary amenorrhea:</i> Constitutionally delayed puberty, various genetic syndromes, anatomic abnormalities</p> <p><i>Secondary amenorrhea:</i> Pregnancy, PCOS, pituitary mass (e.g., prolactinoma), thyroid abnormalities</p> <p><i>Other menstrual dysfunction:</i> use of hormonal birth control methods, physiologic stress</p> <p>Males Primary hypogonadism (gonadal disease), Hypogonadism (e.g., hypothalamic/pituitary disease), toxic exposures, infection, psychosomatic neurological dysfunction</p>

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<p>Poor bone health</p>	<p>Longitudinal loss of BMD/lack of expected bone accrual or maintenance (younger populations) Lower BMD/low Z-score Impaired bone strength or micro-architecture Bone stress injuries Change/differences in bone remodelling biomarkers</p>	<p>ANF¹⁵⁰, FA¹⁵¹, MA¹⁵² FA^{51 153-155} MA^{18 51 155 156} FA^{157 158 159} MA¹⁶⁰ FA^{51 83 161-164}, MA^{51 83 165} SF¹⁶⁶, FA^{49 133 167 168}, MA^{55 169 170}</p>	<p><i>Low BMD:</i> Genetic bone disorders (e.g., osteogenesis imperfecta), hyperparathyroidism, poor micronutrient intake (e.g., calcium and vitamin D), malabsorption disorders (e.g., coeliac disease), malignancies (e.g., leukaemia, lymphoma, metastasis), renal diseases, medications (e.g., anabolic steroids)</p> <p><i>Bone Stress Injury:</i> External reasons (e.g., training errors, surface, shoes) or internal issues (e.g., body build, medical predispositions as above)</p>
<p>Impaired GI function</p>	<p>Abdominal pain/cramps/bloating/alteration in bowel movements</p>	<p>FA^{8 83 144 171}, MA⁸³</p>	<p>GI diseases (e.g., Coeliac disease, inflammatory bowel disease, Helicobacter-Pylori, gastroesophageal reflux, functional dyspepsia/constipation), medications (e.g., antidepressants, iron pills, narcotics, laxative/cathartic use in EDs)</p>
<p>Impaired energy metabolism/ regulation</p>	<p>Sub-clinically or clinically low T3 Low RMR/RMR ratio Reduced leptin Increased cortisol</p>	<p>SF^{127 172-174}, FA^{51 137 139 143 145 167 175 176}, MA^{51 147} FA^{136 144 145 175-180} MA^{105 146 181} SF^{47 182}, FA^{49 133 143 167 175}, MA^{48 183} SF^{127 129}, FA^{180 184 132 133 139}, MA^{82 104}</p>	<p>Primary or Central (secondary & tertiary) hypothyroidism, medications/supplements</p> <p><i>Increased cortisol:</i> physiologic stress, Cushing disease, steroid use</p>
<p>Impaired haematological status</p>	<p>Low iron status Increased hepcidin concentrations/response Reduced iron absorption Lower haemoglobin concentration/mass Reduced response to altitude training</p>	<p>FA¹⁸⁵ SF⁵⁷, MA^{186 187} MA¹⁸⁷ FA¹⁸⁸, MA⁷⁵ MA¹⁸⁹</p>	<p>Acute or chronic blood loss (e.g., menstrual cycle, GI bleeding), RBC destruction (e.g., haemolysis, hemoglobinopathy, splenomegaly), poor micronutrient intake (e.g., iron, vitamin B12, folate), bone marrow diseases</p>
<p>Urinary incontinence</p>	<p>Urinary incontinence</p>	<p>FA^{190 191 192}</p>	<p>Persistent urinary incontinence: Trauma (e.g., childbirth, surgery, radiation), anatomical abnormalities, neurological diseases.</p>

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			Temporary urinary incontinence: Pregnancy, urinary tract infection, constipation, certain foods & drugs
Impaired glucose and lipid metabolism	Reduced fasting/24-hour glucose Reduced fasting/24-hour insulin Elevated total cholesterol/LDL cholesterol	SF ¹²⁷ , FA ^{139 171 177} , MA ¹⁹³ SF ¹²⁷ , FA ⁴⁹ , MA ^{48 104 193 194} FA ^{135 195 196} , MA ^{74 147 148}	Impaired glucose metabolism: Insulinoma, critical illness, medications, adrenal insufficiency Impaired lipid metabolism: familial hyperlipidaemia
Mental health issues	Depression Exercise dependence/addiction DE behaviours/EDs	FA ^{8 80 81} , MA ⁸¹ FA ^{83 197} , MA ^{82 83} FA ^{83 136 177} , MA ^{82 83}	Primary psychologic/mood disorders
Impaired neurocognitive function	Reduced/impaired memory Reduced/impaired decision making Reduced/impaired spatial awareness Poor planning/cognitive flexibility Reduced executive function	FA ¹⁹⁸ , ANF ¹⁹⁹ ANF ²⁰⁰ FA ²⁰¹ ANF ²⁰² FA ¹⁹⁸	Dementia (e.g., Alzheimer's disease), vitamin deficiencies, infections, malignancies, ADHD, substance use disorder, primary psychologic/mood disorders, traumatic brain injury
Sleep disturbances	Sleep disturbances (self-reported)	FA ⁷⁸ , MA ⁵²	Primary psychologic/mood disorders, shift-work, obstructive sleep apnoea, chronic pain/injury, nocturia, medications/substance use, restless legs syndrome
Cardiovascular dysfunction	ECG abnormalities (e.g., sinus bradycardia, QT Prolongation and QT dispersion) Haemodynamic abnormalities (e.g. hypotension and orthostatic hypotension, syncope) Impaired endothelial function / reduced blood flow	FA ^{144 203} , MA ^{74 204} , ANM ²⁰⁵ , ANF ²⁰⁶ ²⁰⁷ FA ^{203 208} , ANF ²⁰⁹ , MA ²⁰⁴ FA ^{179 195 203 210-214} , MA ²¹⁵	<i>Bradycardia:</i> Genetic, ultra-endurance training, hypothyroidism, medications (e.g., beta-blockers), toxic exposures, electro-conductive disorders, electrolyte abnormalities <i>Hypotension:</i> illness, medications, dehydration

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	Cardiac abnormalities (e.g., MVP, decreased left ventricular mass, decreased left ventricular systolic function, myocardial fibrosis)	ANF ²¹⁶ , ANM ^{205 216}	
Reduced skeletal muscle function	Reduced rate of muscle protein synthesis Reduced rates of muscle glycogen restoration	FA ²¹⁷⁻²¹⁹ , SM ²²⁰ , MA ^{217 218} FA ²²¹ , MA ^{50 222}	Inadequate protein intake Inadequate CHO intake
Impaired growth and development	Reduced IGF-1 Increased GH/GH resistance Deviation from the expected growth curve	SF ^{127 172} , FA ^{137 167} , MA ^{147 194 223 224} SF ¹²⁷ , FA ¹³² , MA ^{104 224} FA ¹⁴¹ , ANF ^{225 226} , ANM ^{227 228}	Constitutional delayed puberty, chronic diseases, GH deficiency, congenital or acquired hypogonadotropic hypogonadism, genetic defects, hyperprolactinemia, long-term drug use (e.g., anabolic steroids, opioids, glucocorticosteroids)
Reduced immunity	Increased infection/illness susceptibility Change in immune biomarkers	FA ^{10 229-231} , MA ^{10 229 231} FA ²³² , MA ²³³	Primary or acquired immune deficiency (e.g., chemotherapy, viral infections,) Intensive exercise without LEA

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

Abbreviations: ADHD: Attention-deficit/hyperactivity disorder; CHO: carbohydrate; ECG: electrocardiogram; FHA: Functional hypothalamic amenorrhea; GI: Gastrointestinal; GH: Growth Hormone; IGF-1: Insulin-like Growth Factor-1; LDL: low density lipoprotein; LEA: Low Energy Availability; LH: Luteinizing Hormone; MVP: mitral valve prolapse; OCD: Obsessive Compulsive Disorder; PCOS: Polycystic Ovary Syndrome; RMR: Resting Metabolic Rate; T3: triiodothyronine

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

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)	<p>Increase in training days lost or modified due to illness or injury (e.g., impaired preparation)</p> <p>Inability to compete at key competitions due to illness or injury</p>	<p>Tier 4* FA (n=85) & MA (n=47) Olympic athletes from 11 different sports¹⁰ Tier 4 FA (n = 55) & MA (n = 26) Olympic athletes from 11 different sports²²⁹ Tier 4 FA endurance athletes (n=45)¹⁷¹ Tier 4 FA endurance athletes (n=13)²³⁴ Tier 3 FA college athletes (n=116) from endurance, power, and team sports²³⁵ Unspecified Tier FA high school athletes (n=163) from endurance, power, and team sports²³⁶ Unspecified Tier FA high school athletes (n=249) from aesthetic, endurance, and team sports²³⁷ A mix of Tier 1-4 FA (n = 833)²³⁰ Tier 2 FA figure skaters (n=137)²³⁸</p> <p>Tier 4 FA endurance athletes (n=13)²³⁴ Unspecified Tier FA high school athletes (n=163) from endurance, power, and team sports²³⁶</p>
Decreased training response	<p>Decreased rather than increased performance of treadmill protocol following 4 weeks intensified training plus 2 weeks recovery</p> <p>Reduced performance of 5 km on-water rowing following a period of intensified training</p> <p>Reduced swimming velocity in 400 m time trial after 12 weeks of training</p> <p>Self-reported reduction in training response</p>	<p>Tier 2 club level FA endurance runners (n = 16)⁷³</p> <p>Tier 4 national level MA (n=5) and FA rowers (n=5)²³⁹</p> <p>Tier 3 junior national level FA swimmers (n = 10)¹³⁷</p> <p>Unspecified mixed tier FA (n = 1000)⁸</p>

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	Decreased aerobic (4000 m time trial) and anaerobic (15 second) performance after 2 weeks intensified training including inadequate energy intake	Tier 3 MA road cyclists (n = 13) ¹⁰⁸
Decreased recovery	<p>Direct: Self-reported failure to recover between training sessions</p> <p>Indirect: Reduced glycogen synthesis</p> <p>Indirect: Reduced muscle protein synthesis</p> <p>Indirect: Reduced PCr recovery</p>	<p>Tier 4 FA (n = 8) and MA (n = 4) lightweight rowers⁷⁸</p> <p>Tier 3 MA endurance runners (n = 7)⁵⁰</p> <p>Tier 1 MA (n = 6) and FA (n = 7) endurance athletes²²¹</p> <p>Unspecified tier resistance-trained FA (n=7) and MA (n=8)²¹⁷</p> <p>Tier 2 FA (n = 19) endurance athletes²⁴⁰</p>
Decreased cognitive performance/skill	<p>Reduced reaction time</p> <p>Self-reported impaired judgement and decreased coordination and concentration</p>	<p>Tier 4 FA endurance athletes (n = 30)¹³⁹</p> <p>Unspecified tier FA (n = 1000)⁸</p>
Decreased motivation	<p>Decreased well-being</p> <p>Increase in total mood disturbance (e.g., fatigue, vigour)</p> <p>Self-reported increase in irritability and depression</p> <p>Emotional lability</p> <p>Increased irritability</p> <p>Increase in total mood disturbance and general stress</p>	<p>Tier 3 MA endurance athletes (n=18)⁹²</p> <p>Tier 4 national level MA (n = 5) and FA rowers (n = 5)²³⁹</p> <p>Unspecified tier FA (n = 1000)⁸</p> <p>Tier 2-4 Mix of sports FA (n=8)⁶⁹</p> <p>Tier 3 Endurance FA (n=10) and MA (n=2)⁶⁹</p> <p>Tier 3 M Road cyclists (n = 13)¹⁰⁸</p>

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	Self-reported decrease in mood, emotional self-regulation, concentration, social interaction, food anxiety	Tier 4 FA (n = 8) and MA (n = 4) lightweight rowers ⁷⁸
Decreased muscle strength	Decreased neuromuscular strength	Tier 4 FA endurance athletes (n = 30) ¹³⁹
	Decreased explosive power (countermovement jump)	Tier 3 MA endurance athletes (n = 18) ⁹² Tier 2-3 MA bodybuilder (n=1) ²⁴¹
	Decreased explosive power (countermovement jump, reactive jump)	Tier 2 junior elite FA cross country skiers (n = 19) ²⁴²
	Decreased concentric hamstring peak torque	Tier 2-3 MA bodybuilder (n=1) ⁸⁷
	Decreased isometric bench press	Tier 2-3 FA fitness competitors (n=27) ¹⁴³
	Decreased 1 rep max squat, bench press, deadlift	Tier 2-3 MA bodybuilder (n=1) ²⁰⁴
	Decreased concentric and eccentric peak force	Tier 2-3 FA physique athlete (n=1) ²⁴³
Decreased endurance performance	Decreased performance of treadmill run protocol	Tier 2 club level FA endurance runners (n = 16) ⁷³
	Reduced 5 km on-water rowing performance	Tier 4 national level MA (n=5) and FA rowers (n=5) ²³⁹
	Decreased neuromuscular endurance	Tier 4 FA endurance athletes (n=30) ¹³⁹
	Self-reported reduction in endurance performance	Unspecified Tier FA athletes (n = 1000) ⁵⁶
	Decreased VO _{2 max}	Tier 3-4 FA endurance athletes (n = 33) ²⁴⁴
	Apparent underperformance in 60-minute functional power threshold vs training load	Tier 3 MA road cyclists (n = 50) ¹⁸

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	Decreased performance of 4000 m time trial	Tier 3 MA road cyclists (n = 13) ¹⁰⁸
	Self-reported decrease in rowing performance	Tier 4 FA (n = 8) and M (n = 4) lightweight rowers ⁷⁸
Decreased power performance	Reduced velocity during 400 m swim time trial	Tier 3 junior national level FA swimmers (n = 10) ¹³⁷
	Decreased anaerobic (Wingate) performance	Tier 2-3 MA bodybuilder (n=1) ⁵²
	Decreased number of throws in a Judo Specific Fitness Test	Tier 2 MA 2 nd and 3rd Dan black belt Judo athletes (n = 11) ¹⁰⁶
	Decreased performance of 15 second cycling sprint	Tier 3 MA road cyclists (n = 13) ¹⁰⁸

*tiering system according to McKay et al.³²

Abbreviations: FA: female athlete; km: kilometres; m: metre; MA: male athlete; PCr: phosphorylated creatine; VO_{2 max} : maximal oxygen consumption

REDs physiological model

Experts in the field have long realised that applying LEA exposure (i.e., severity, duration, frequency) on subsequent REDs short, medium, and long-term outcomes is complex and dependent upon 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’²⁴⁵, 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.

*****insert Figure 3 here *****

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D. CLINICAL APPLICATIONS

Assessment of energy availability

Seminal research^{47 166} around EA in habitually sedentary females identified a continuum of zones ranging from low to high risk of harm (e.g., high EA for mass gain and growth = >45 kcal/kg FFM/d; adequate EA for weight maintenance and support of body function = ~45 kcal/kg FFM/d; reduced EA for body mass/fat loss = 30-45 kcal/kg FFM/d; and LEA causing health implications = <30 kcal/kg FFM/d)²⁴⁶. The concept of the LEA threshold (30 kcal/kg FFM/d), below which health problems occurred, was based on elegant but short-term laboratory studies that investigated stepwise changes in EA, perturbations of sex hormones^{47 127 247}, and changes in markers of bone turnover¹⁶⁶ 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 studies⁹⁰, theoretical constructs, and methodological challenges in assessment, around the frailty of a single, universal threshold²⁴⁸, 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 (e.g., 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 [e.g., FFM, resting metabolic rate (RMR)],^{41 51 248} but these can be better managed in the future by implementing a standardised approach²¹. 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.

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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 sports²⁴⁹. 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^{78 250}. 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^{27 251 252} (see Figure 4). Exceptional circumstances may exist where body composition assessment may be justified for athletes <18 years. Still, such decision warrants careful consideration and consensus amongst the athletes’ health and performance team and requires guardian consent.

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^{250 253}. Unfortunately, many members of the athlete entourage appear to *i)* lack the knowledge of safe regulation of body weight and composition and how it can be utilised to improve performance while maintaining health; *ii)* have ignorance of the suitability of various body composition methods and the possible negative health effects consequent to inappropriate assessment; and *iii)* 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^{251 254}. It is important, therefore, to identify valid and reliable body composition assessment methods and develop clear guidelines on how to interpret, manage, and communicate safely to athletic populations²⁵¹.

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 percent; they do not provide valid data, use spurious

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assumptions, and/or are influenced greatly by athlete presentation (e.g., hydration levels)²⁵⁵. 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 individuals²⁵⁶. Though costlier, DXA is a reliable method for assessing BMD and estimating fat and lean masses, provided standard test protocols are utilised²⁵⁷⁻²⁵⁹. 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 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^{27 251}. This should include pre-screening 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 results⁷⁰.

*****Insert Figure 4 here ****

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 2015²⁶⁰. 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 CAT2²⁶ (Figures 5, 6, and Tables 4, 5), which has

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undergone internal expert voting statement validation (see appendices 2-4) and external REDs expert clinical cross-agreement validation²⁶.

The IOC REDs CAT2 consists of a 3-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 [e.g., 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).

**** Insert Figure 5 here****

The IOC REDs CAT2²⁶ introduces a four-colour traffic-light severity/risk categorisation, in contrast to the three-colour stratification in the 2015 RED-S CAT²⁶⁰, 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.

*****Insert Box 2- REDs-CAT definitions here*****

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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 (i.e., 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 (i.e., 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 (i.e., 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 (e.g., pain from a BSI, amenorrhea, 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 (e.g., a significant change in sex hormones, resting metabolic rate, cholesterol). Note: some indicators can be both signs and symptoms (e.g., amenorrhea).

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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, utilising information collected from a multi-disciplinary team (e.g., 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.

*****Insert Figure 6 here *****

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. Every indicator below 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 (e.g., 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-and sex-appropriate and laboratory-specific reference ranges. Most REDs data and associated thresholds have been established in pre-menopausal/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 (e.g., 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.

REDs indicator	References
<i>Severe Primary Indicators (count as 2 Primary Indicators)</i>	
Primary amenorrhea (<i>Females:</i> Primary amenorrhea is indicated when there has been a failure to menstruate by age 15 in the presence of normal secondary sexual development (two standard deviations above the mean of 13 years), or within five years after breast development if that occurs before age 10; or prolonged secondary amenorrhea (absence of 12 or more consecutive menstrual cycles) due to FHA	6 260-263
Clinically low free or total testosterone (<i>Males:</i> below the reference range)	51 94 123 264-266

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<i>Primary Indicators</i>	
Secondary amenorrhea (<i>Females</i> : absence of 3 to 11 consecutive menstrual cycles) caused by FHA	6 260-262
Sub-clinically low total or free testosterone (<i>Males</i> : within the lowest 25% (quartile) of the reference range)	51 94 97 123 264-266
Sub-clinically or clinically low total or free T3 (within or below the lowest 25% (quartile) of the reference range)	51 177 265
History of ≥ 1 high-risk (femoral neck, sacrum, pelvis) <i>or</i> ≥ 2 low-risk BSI (all other BSI locations) within the previous 2 years <i>or</i> absence of ≥ 6 months from training due to BSI in the previous 2 years	161 261 267
<u>Pre-menopausal females and males <50 years old</u> : BMD Z-score* <-1 at the lumbar spine, total hip, or femoral neck or decrease in BMD Z-score from prior testing <u>Children/Adolescents</u> : 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).	121 122 125 268
A negative deviation of a paediatric or adolescent athlete's previous growth trajectory (height and/or weight)	269 270
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 (<i>only 1 primary indicator for either or both outcomes</i>)	70 82 236 271- 273
<i>Secondary Indicators</i>	
Oligomenorrhea caused by FHA (>35 days between periods for a maximum of 8 periods/year)	6 260-262
History of 1 low-risk BSI (see high vs low-risk definition above) within the previous 2 years <i>and</i> absence of <6 months from training due to BSI in the previous 2 years	161 261 267
Elevated total or LDL cholesterol (above reference range)	146 195 274
Clinically diagnosed depression and/or anxiety (<i>only 1 secondary indicator for either or both outcomes</i>)	271 275 276
<i>Potential Indicators (not scored, emerging) **</i>	
Sub-clinically or clinically low IGF-1 (within or below the lowest 25% (quartile) of the reference range)	11 137 265
Clinically low blood glucose (below the reference range)	11 82
Clinically low blood insulin (below the reference range)	47 127 265
Chronically poor or sudden decline in iron studies (e.g., ferritin, iron, transferrin) and/or haemoglobin	277-280
Lack of ovulation (via urinary ovulation detection)	262 281-283
Elevated resting AM or 24-hour urine cortisol (above the reference range or significant change for an individual)	47 127 133 265
Urinary incontinence (<i>Females</i>)	190 284 285
GI or liver dysfunction / adverse GI symptoms at rest and during exercise	8 171 286
Reduced or low RMR <30 kcal/kg FFM/d or RMR ratio <0.90	9 177 287 288
Reduced or low libido/sex drive (especially in males) and decreased morning erections	110-113
Symptomatic orthostatic hypotension	269 289 290
Bradycardia (HR < 40 in adult athletes; HR<50 in adolescent athletes)	269 270 289
Low systolic or diastolic BP (<90/60mmHg)	291 292
Sleep disturbances	52 78 293
Psychological symptoms (e.g., increased stress, anxiety, mood changes, body dissatisfaction and/or body dysmorphia)	8 70 271 275 276 294
Exercise dependence/addiction	70 82 295 296
Low BMI	261 269 270

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* BMD assessed via DXA within ≤ 6 months. In some situations, using a Z-score from another skeletal site may be warranted [e.g., 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]^{121 297}. A true BMD decrease (from prior testing) is ideally assessed in comparison to the individual facilities DXA's Least Significant Change (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^{122 297}

** Potential indicators are purposefully vague in quantification, pending further research to quantify parameters and cut-offs more accurately.

Abbreviations and definitions: Adolescent: <18 years of age; BP: Blood Pressure; BMD: Bone Mineral Density; BMI: Body Mass Index; BSI: Bone Stress Injuries; DXA: dual-energy X-ray absorptiometry; DSM-5-TR: Diagnostic and Statistical Manual of Mental Disorders, 5th edition, text revision; EDE-Q: Eating Disorder Examination Questionnaire; FFM: Fat-Free Mass; FHA: Functional Hypothalamic Amenorrhea; GI: Gastrointestinal; HR: Heart Rate; IGF-1: Insulin-like Growth Factor 1; kcal: kilocalories; LDL: Low-density lipoprotein; RMR: Resting Metabolic Rate; TBLH: Total Body Less Head; T: Testosterone; T₃: Triiodothyronine

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^{269 270 289 291 292 298 299}).

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
$\leq 75\%$ median BMI for age and sex
Electrolyte disturbances (e.g., hypokalemia, hyponatremia, hypophosphatemia)
ECG abnormalities [e.g., prolonged QTc interval or severe bradycardia (Adult: HR ≤ 30 bpm; Adolescent: HR ≤ 45 bpm)]
Severe hypotension: $\leq 90/45$ mmHg
Orthostatic intolerance (Adult & Adolescent: a supine to standing systolic BP drop >20 mmHg and a diastolic drop >10 mmHg)
Failure of outpatient ED treatment program
Acute medical complications of malnutrition (e.g., syncope, seizures, cardiac failure, pancreatitis)
Any condition that inhibits medical treatment and monitoring while training and/or competing

Abbreviations: BMI: body mass index; bpm: beats per minute; ECG: electrocardiogram; ED: eating disorder; HR: heart rate; QTc: corrected QT

Prevention and treatment of REDs

Prevention of REDs

Primary prevention includes tackling inadequate awareness and knowledge of the health and performance sequelae of REDs and sports nutrition among athletes^{115 300-302} and their entourage (e.g., coaches³⁰³⁻³⁰⁵, parents, athlete health and performance team^{300 306 307}). For example, less than half of

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coaches and physicians surveyed were able to identify the three components of the female athlete triad³⁰⁵
^{306 308 309}; other studies reported similar knowledge gaps among physiotherapists and athletic trainers³⁰⁰
³⁰³. Short-term education programs, 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^{72 310-316}. Furthermore, early
identification of symptoms using screening instruments, individual health interviews, and objective
assessment of REDs biomarkers may be useful as secondary prevention²⁶. However, the REDs education
and behaviour modification research field is underdeveloped, and specific REDs education programs
targeting athletes and other key personnel require further exploration and validation²⁸.

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 sequelae²⁸, sometimes requiring adjuvant treatment
of body system dysfunction(s) [e.g., low BMD, GI dysfunction, depression (see Figures 1 and 2)] while
reversing problematic LEA and its various underpinning causes⁷¹. 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
micronutrients³¹⁷.

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^{127 182}. 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 control
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 (e.g., menstrual function
resumption in select participants³¹⁸), but not all symptoms (e.g., inability to retard bone loss³¹⁹). Such
findings may indicate that optimal dietary interventions are not yet identified, dietary changes are

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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 REDs²⁸. One example is 17 β -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 results³²⁰.

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^{28 70 321}. 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.

E. REDs RESEARCH METHODOLOGY GUIDELINES

Although the seminal REDs research implemented randomised clinical trials with strict laboratory-controlled EA interventions in habitually sedentary females^{47 127 166 174 182}, 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 22 51 80 322 323}. 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 51 80 322 323}, as well as a lack of a universal EA threshold below which problems are observed⁹⁰. Cross-sectional studies are useful for clinical REDs assessment and prevalence, but an analysis of this literature reveals multiple limitations (e.g., lack of a classification of subject calibre/training status; lack of a standardisation of recruitment and assessment protocols; poor characterisation of menstrual status and

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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^{137 277}. 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^{48-50 55 56 167 186}. 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 comparisons²².

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 (i.e., variability and precision) and feasibility (e.g., availability, cost). Some tests have standards and diagnostic criteria for what is considered ‘normal’ vs. ‘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.

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	<p><i>Preferred</i></p> <ul style="list-style-type: none"> • Overnight sampling of LH and FSH³²⁴ • Menstruating females: phase-based hormonal approach using urinary ovulation kits (testing mid-cycle LH surge) and blood sampling³²⁵ • Post-pubertal males: morning total and free testosterone level^{326 327} <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> • Females: self-reported menstrual history, urinary ovulation testing^{325 328}, LEAF-Q¹⁷¹ • Males: self-reported libido/morning erection [e.g., LEAM-Q³²⁹ or ADAM-Q^{113 330}]
Impaired bone health	<p><i>Preferred</i></p> <ul style="list-style-type: none"> • DXA^{125 331} <ul style="list-style-type: none"> ○ Using age- and medically-appropriate body-site scanning³³¹ ○ Using age-, sex-, and activity-appropriate interpretation (e.g., Z-score vs. T-score) <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> • Bone stress injury and fracture history <p><i>Potential</i></p>

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	<ul style="list-style-type: none"> • HRpQCT
Impaired gastrointestinal function	<p><i>Preferred</i></p> <ul style="list-style-type: none"> • Oesophageal motility: oesophageal manometry, barium swallow • GERD: upper endoscopy • Gastric motility: electrogastrography^{332 333} • Gastroparesis: gastric emptying study • Pancreatitis: ≥ 2 of: (a) lipase $> 3x$ upper limit of normal; (b) imaging findings consistent with pancreatitis; (c) characteristic epigastric pain • Intestinal transit: radiopaque marker study,³³⁴ oro-caecal transit time test^{335 336} • SMA syndrome: upper GI oral contrasted study, MRI, or CT³³⁷⁻³³⁹ <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> • GERD: many questionnaires³⁴⁰, including GerdQ³⁴¹ • Constipation: Wexner Constipation Score³⁴², Bristol Stool Scale³⁴³ • Diarrhoea: Bristol Stool Scale³⁴³ • Irritable bowel syndrome: Rome IV Criteria³⁴⁴ • Elevated transaminases^{345 346} • Defecatory disorders, faecal incontinence:³⁴⁷ Faecal Incontinence Questionnaire^{8 348}, Faecal Incontinence Severity Index (FISI)³⁴⁹, Altomare's Obstructed Defecation Scale (ODS) score³⁵⁰ • Multiple GI symptoms: Rome II questionnaire³⁵¹ • GI symptoms during exercise^{352 353} • LEAF-Q GI subsection score ≥ 2 indicative of LEA^{171 354} • Athlete-specific GI symptom inventory³⁵⁵ • Feeding challenge during exercise^{335 356} <p><i>Potential</i></p> <ul style="list-style-type: none"> • Intestinal transit: wireless motility capsule • Gut bacterial profile • Faecal or plasma short-chain fatty acid concentration
Impaired energy metabolism/regulation	<p><i>Preferred</i></p> <ul style="list-style-type: none"> • Thyroid function tests: TSH, free T4, total and free T3¹⁷⁴ • Leptin: overnight sampling³⁵⁷ • Cortisol: overnight sampling,¹³³ 24-hour urinary free cortisol³⁵⁸ • Laboratory / expert-controlled measurements/estimates of all compartmentalized energetic intakes and total daily expenditures (exercise, non-exercise activity, basal metabolic rate, thermic effect of food)³⁵⁹ <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> • Cortisol: morning serum cortisol, late-night salivary cortisol³⁵⁸ • RMR: indirect calorimetry,³⁶⁰ room calorimetry²⁸⁷
Impaired haematological status	<p><i>Preferred</i></p> <ul style="list-style-type: none"> • CBC with differential • Iron studies (iron, ferritin, transferrin, total iron binding capacity) with age-, sex-, and laboratory-appropriate cut-offs • Carbon monoxide haemoglobin mass measurement^{361 362} <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> • Self-reported history of iron deficiency or anaemia <p><i>Potential</i></p> <ul style="list-style-type: none"> • App-based self-assessment³⁶³
Urinary incontinence	<p><i>Preferred</i></p> <ul style="list-style-type: none"> • Stress urinary incontinence: bladder stress test³⁶⁴ • International Consultation on Incontinence-Urinary Incontinence Short Form (ICIQ-UISF)^{190 191} • 3 Incontinence Questionnaire (3IQ)³⁶⁵ <p><i>Potential</i></p> <ul style="list-style-type: none"> • Pelvic Floor Dysfunction-ScrEeNing Tool IN fEmaLe athLetes (PFD-SENTINEL)³⁶⁶

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Impaired glucose and lipid metabolism	<p><i>Preferred</i></p> <ul style="list-style-type: none"> • Fasting blood glucose (serial measures)³⁶⁷ • Fasting insulin³⁶⁷ • Lipid panel: HDL, LDL, total cholesterol, triglycerides²⁷⁴ <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> • Continuous glucose monitor³⁶⁸
Mental health issues	<p><i>Preferred</i></p> <ul style="list-style-type: none"> • Clinical interview with psychiatrist or psychologist, DSM-5-TR³⁶⁹ <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> • Depression: PHQ,³⁷⁰ Center for Epidemiological Studies Depression scale,³⁷¹ Beck Depression Inventory³⁷² • Generalized anxiety: GAD-7^{321 373}, DASS-21^{80 229 374 375} • Stress: perceived stress scale³⁷⁶ • Brunel Mood Scale³⁷⁷ • Profile of Mood States^{378 379} • Eating disorders: EDE-Q,³⁸⁰⁻³⁸² BEDA-Q,³⁸³ Eating Disorder Inventory,³⁸⁴ self-report
Impaired neurocognitive function	<p><i>Preferred</i></p> <ul style="list-style-type: none"> • Clinical neuropsychological assessment <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> • Multiple domains: CogState assessment battery³⁸⁵ • Planning / cognitive flexibility: Wisconsin Card Sorting Test²⁰² • Attention: Stroop Color and Word Test³⁸⁶⁻³⁸⁸ • Decision making: Iowa Gambling Test^{389 390} • Verbal memory: California Verbal Learning Test-II³⁹¹ • Executive function: Delis-Kaplan Executive Function System Color-Word Interference Test¹⁹⁸, BRIEF-A³⁹²
Sleep disturbances	<p><i>Preferred</i></p> <ul style="list-style-type: none"> • Polysomnography³⁹³ <p><i>Used and Recommended</i>³⁹³</p> <ul style="list-style-type: none"> • Research-grade actigraphy • Sleep diaries • Numerous questionnaires, including Athlete Sleep Screening Questionnaire (ASSQ),³⁹⁴ Athlete Sleep Behavior Questionnaire (ASBQ),³⁹⁵ Epworth Sleepiness Scale,³⁹⁶ Pittsburgh Sleep Quality Index,^{10 397} Insomnia Severity Index^{321 398} <p><i>Potential</i></p> <ul style="list-style-type: none"> • Sport wearables³⁹⁹
Impaired cardiovascular function	<p><i>Preferred</i></p> <ul style="list-style-type: none"> • Conduction, rhythm abnormalities: ECG²⁸⁹ • Rate abnormalities: cardiac telemetry, Holter monitor • Haemodynamics: sphygmomanometry, orthostatic sphygmomanometry (≥ 20 mmHg drop in systolic pressure, ≥ 10 mmHg drop in diastolic pressure on standing from supine)^{289 400} • Autonomic function: heart rate variability by Holter monitor,^{401 402} baroreflex sensitivity testing,⁴⁰³ bedside tests (e.g., Valsalva, tilt testing) • Structural abnormalities: transthoracic echocardiogram²⁸⁹ • Endothelial dysfunction: brachial artery flow-mediated dilatation^{195 404} <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> • Heart rate: chest-mounted electrode-containing heart rate strap^{405 406} • Hemodynamics: self-reported episodes of orthostatic (pre-) syncope <p><i>Potential</i></p> <ul style="list-style-type: none"> • Sport wearables^{399 407}
Reduced skeletal muscle function	<p><i>Preferred</i></p> <ul style="list-style-type: none"> • Muscle protein synthesis: isotopic amino acid labelling,⁴⁰⁸ deuterated water ingestion^{409 410}

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	<ul style="list-style-type: none"> Muscle glycogen content: histochemical analysis of biopsy-derived muscle samples⁴¹¹, ¹³C-magnetic resonance spectroscopy^{50 412} <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> none— exclude assessment if unable to directly measure as above
Impaired growth and development	<p><i>Preferred</i></p> <ul style="list-style-type: none"> Paediatric patients: clinical assessment with growth charts <ul style="list-style-type: none"> Deviation from baseline growth trajectory, defined as a dynamic change with time (vs. a single measurement) Decrease in growth Z-score by > 1^{269 413} Growth hormone: overnight sampling⁴¹⁴ IGF-1: serum levels, IGFBP-3 levels⁴¹⁵ <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> Paediatric patients: delayed markers of puberty (thelarche, menarche, spermarche)
Reduced immunity	<p><i>Preferred</i></p> <ul style="list-style-type: none"> To be determined <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> Self-reported illness frequency^{10 416 417} <p><i>Potential</i></p> <p>Complete blood count with differential, immunoglobulin G glycome, leukocyte transcriptome, and cytokine profile²³²</p>

Performance Outcome	Methods and Notes
Decreased athlete availability	<p><i>Preferred</i></p> <ul style="list-style-type: none"> Self-reported days of training/competition lost or modified due to illness or injury^{10 234 418}
Decreased training response	<p><i>Preferred</i></p> <ul style="list-style-type: none"> Longitudinal tracking of valid performance-related metric specific to athlete/sport (e.g., sport-related time trial)^{137 419 420} <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> Self-reported plateauing of ability/performance despite training progression⁴²¹ Exercise lactate profile^{422 423} Lactate:RPE ratio^{424 425} Catecholamine concentrations⁴²⁶
Decreased recovery	<p><i>Preferred</i></p> <ul style="list-style-type: none"> To be determined <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> Lab-based studies: <ul style="list-style-type: none"> Creatine phosphate system: ³¹P magnetic resonance spectroscopy⁴²⁷ Exercise-induced muscle damage: muscle biopsy⁴²⁸ Field-based studies: <ul style="list-style-type: none"> Questionnaires: Recovery-Stress Questionnaire (REST-Q),^{10 429} self-reported perceptions of recovery, Profile of Moods State (POMS),³⁷⁸ Hooper MacKinnon Questionnaire⁴³⁰ Creatine kinase (total, MM)⁴³¹ Athlete's subjective report of readiness⁴³² <p><i>Potential</i></p> <ul style="list-style-type: none"> Wearable/commercialized recovery/readiness algorithms⁴³³
Decreased cognitive performance/skill	<p><i>Preferred</i></p> <ul style="list-style-type: none"> Skill: sport-specific measures (e.g., Loughborough Soccer Passing Test)^{434 435} <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> Reaction time: consider sport-specific tests⁴³⁶

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	<ul style="list-style-type: none"> Spatial awareness: mental rotation test²⁰¹
Decreased drive/motivation	<p><i>Preferred</i></p> <ul style="list-style-type: none"> To be determined <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> Motivation: Behavioral Regulation in Sport Questionnaire (BRSQ),⁴³⁷ Psychological Need States in Sport-Scale (PNSS-S)⁴³⁸ Athlete Burnout Questionnaire (ABQ)⁴³⁹ Maslach Burnout Inventory⁴⁴⁰
Decreased muscle strength	<p><i>Preferred</i></p> <ul style="list-style-type: none"> Longitudinal tracking of valid performance-related metric specific to athlete/sport (e.g., sport-related strength test, such as snatch or clean and jerk for weightlifting, or throw distance for shot put ⁴⁴¹) <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> Isokinetic dynamometry^{442 443} 1 repetition maximum, specific movement (e.g., bench press) ^{444 445}
Decreased endurance performance	<p><i>Preferred</i></p> <ul style="list-style-type: none"> Longitudinal tracking of valid performance-related metric specific to athlete/sport (e.g., sport-related time-trial)^{137 419 420} <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> Laboratory-based VO₂ max testing (via indirect calorimetry)⁴⁴⁶ Laboratory-based lactate threshold testing⁴⁴⁷ Multistage shuttle run^{448 449} Cycling ramp test⁴⁵⁰
Decreased power performance	<p><i>Preferred</i></p> <ul style="list-style-type: none"> Wingate test⁴⁵¹ <p><i>Used and Recommended</i></p> <ul style="list-style-type: none"> Counter-movement jump⁷⁵ Standing broad jump^{452 453} Bosco test^{454 455}

*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.

Abbreviations and definitions: ADAM-Q, Androgen Deficiency in Aging Males Questionnaire; BEDA-Q, Brief Eating Disorder in Athletes Questionnaire; BRIEF-A, Behavior Rating Inventory of Executive Function–Adult Version; CBC, Complete Blood Count; CT, Computerized Tomography; DASS-21, Depression Anxiety Stress Scale-21; DSM-5 TR, Diagnostic and Statistical Manual of Mental Disorders– 5th 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, Gastroesophageal reflux disease; GerdQ, Gastroesophageal 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; LEA, low energy availability; LDL, Low-density lipoprotein; LEAF-Q, Low Energy Availability in Females Questionnaire; LEAM-Q, Low Energy Availability in Males Questionnaire; LH, Luteinizing hormone; MRI, Magnetic resonance imaging; 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; VO₂ max, Maximal oxygen consumption

CONCLUSION

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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 statement⁶: 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 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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FIGURE LEGENDS

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).

*Psychological consequences can either precede REDs or be the result of REDs.

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).

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 4 steps to adapt and update the model as the future science of LEA/REDs evolves. Examples of moderating factors are also provided (Step 3).

Figure 4. A conceptual framework on the implementation of body composition assessments (e.g., height, weight, anthropometrics, skinfolds) within the context of athlete stage of development and their nutritional preparation skills ²⁵¹ (reprinted with permission from BJSM).

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.

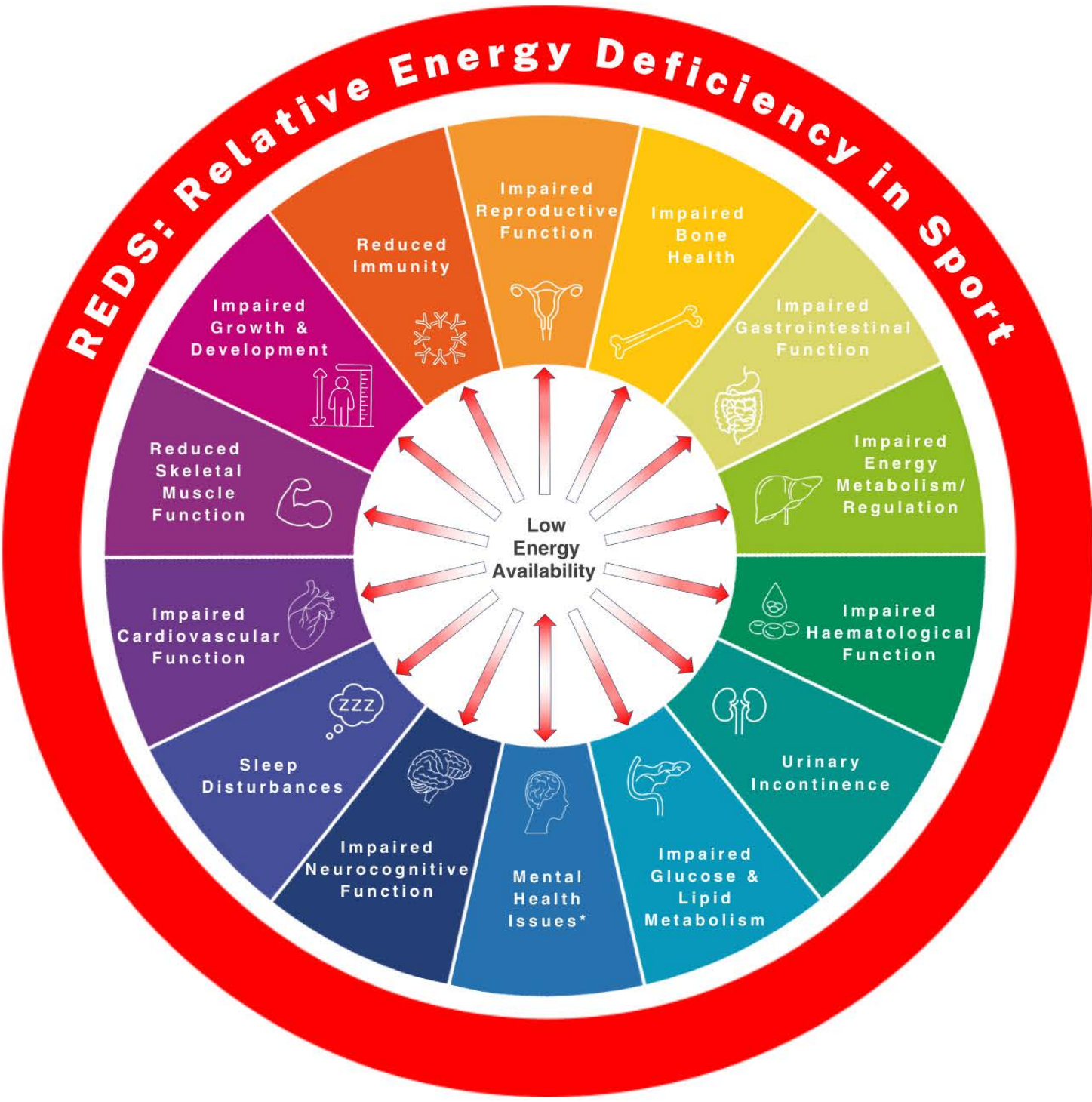
Abbreviations: REDs: Relative Energy Deficiency in Sport

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 appendix 5 for the IOC REDs CAT2 Scoring Tool.

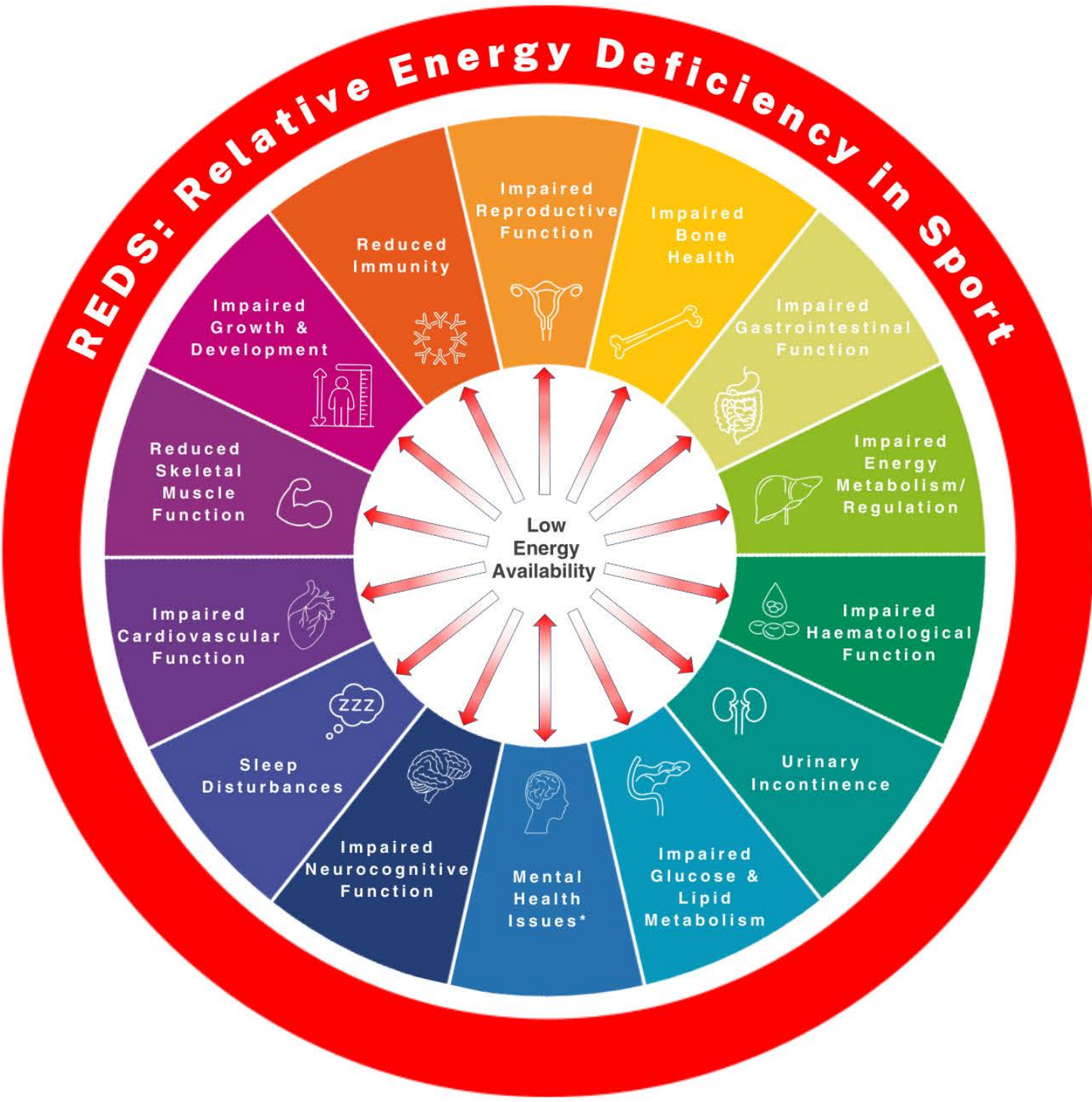
Abbreviations: bpm: Beats Per Minute; BMI: Body Mass Index; BP: Blood Pressure; ECG: Electrocardiogram; EDs: Eating Disorders; HR: Heart Rate; mmHg: milli metres Mercury; REDs: Relative Energy Deficiency

*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.*⁴⁵⁶

REDS: Relative Energy Deficiency in Sport



REDS: Relative Energy Deficiency in Sport



Body system

STEP 2: Identify the characteristics of the LEA exposure that are of most consequence to this body system – see list

STEP 3: Identify moderating factors that might alter the effects of LEA on the body system to affect health or performance outcomes – see list

REDs outcomes

STEP 1: Identify the specific impairments of health or performance by which this body system might be perturbed by LEA. This step should also include noting the criterion tests used to assess health and performance, and potential differential diagnoses – causes other than LEA

Characteristics of LEA exposure

- Severity (magnitude)
- Duration
- Consistency
- Origin
- Within Day Energy Balance
- Accumulated Dose (e.g., severity x duration/frequency)

Moderating factors

Categories	Potential moderating factors
Personal characteristics	Sex
	Age/gynaecological age
	Genetics/epigenetics
Medical history	Anatomical/ biomechanical features
	Co-existing medical disorders
	Medication use
	Past medical history
	Menstrual disturbances/low oestrogen (female)
	Low testosterone (male) PCOS/high androgen (female)
Training characteristics	Low impact exercise
	High impact exercise
	Training errors
	Resistance training
Dietary/ nutritional characteristics	Energy intake
	Carbohydrate availability
	Protein intake
	Vitamin D status
	Bioavailable iron intake
	Calcium intake
	Energy density
	Intake of caffeine and other stimulants
Other	Psychological/lifestyle stress
	Environmental stress

Explanation

Explanation

Explanation

Health outcomes

- Specific health outcome 1
- Specific health outcome 2
- Specific health outcome 3
- Specific health outcome 4... etc

Performance outcomes

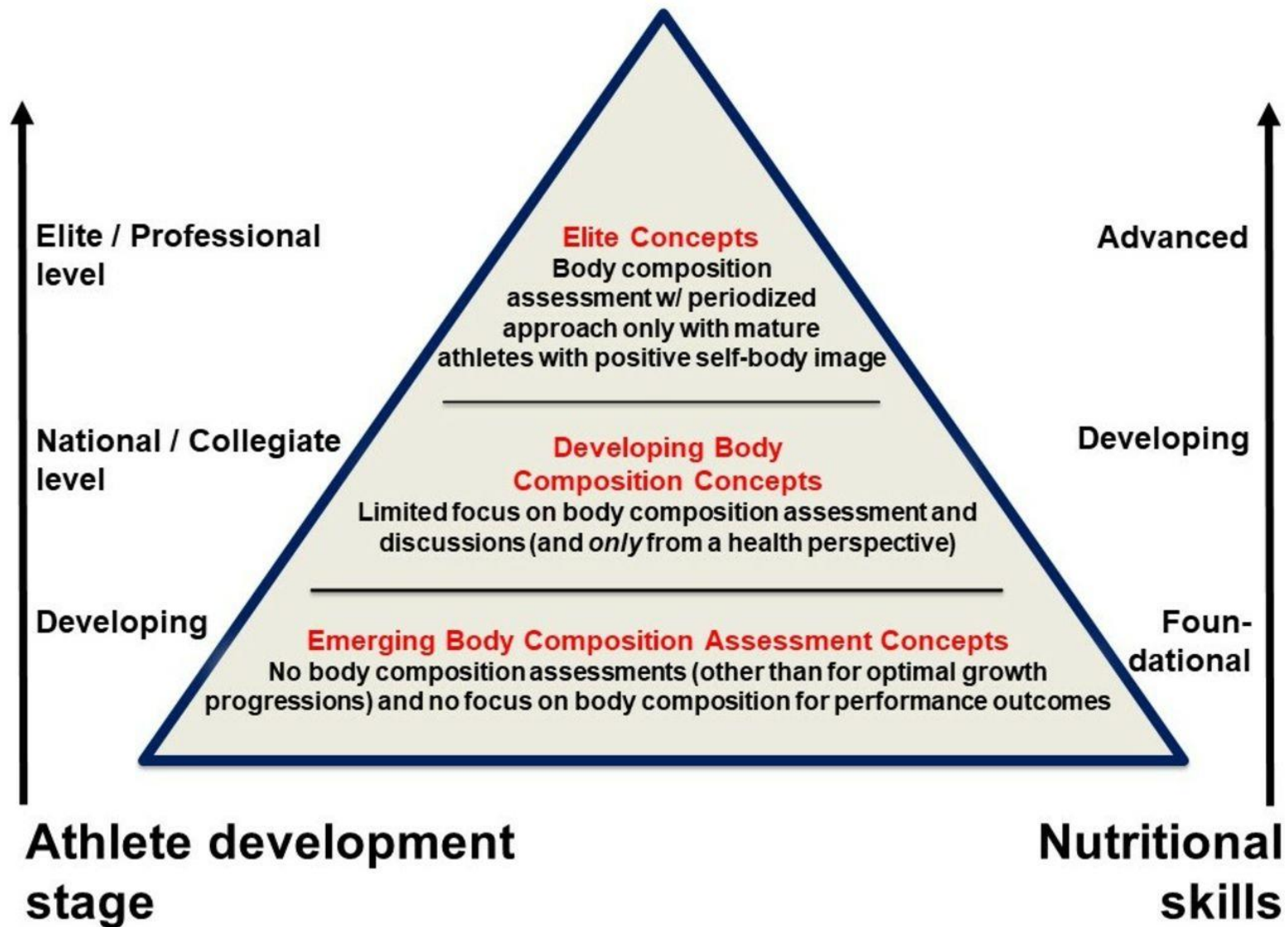
- Specific performance outcome 1
- Specific performance outcome 2
- Specific performance outcome 3
- Specific performance outcome 4... etc

Criterion tests to assess health and diagnose impairments

Criterion tests to assess performance outcomes

STEP 4: Identify the mechanisms/associations between moderating factors and the health/performance outcome using colour and the strength of the line to note the direction (+ve or -ve) of the effect and robustness of the evidence. Provide brief summary on arrow

A conceptual framework on the implementation of body composition assessments (e.g, height, weight, anthropometrics, skinfolds, etc.) within the context of athlete stage of development and their nutritional preparation skills.



Kathryn E Ackerman et al. Br J Sports Med 2020;54:369-370

Step 1

REDs Screening (Population-specific Questionnaires or Clinical Interview)

- Lower sensitivity and specificity
- Inexpensive and easy to use
- Questionnaires allow for large athlete group screening



greater than low
risk proceeds to
Step 2

Step 2

REDs Severity/Risk Assessment

- High sensitivity
- More expensive
- Clear scoring allows for easy and reliable implementation

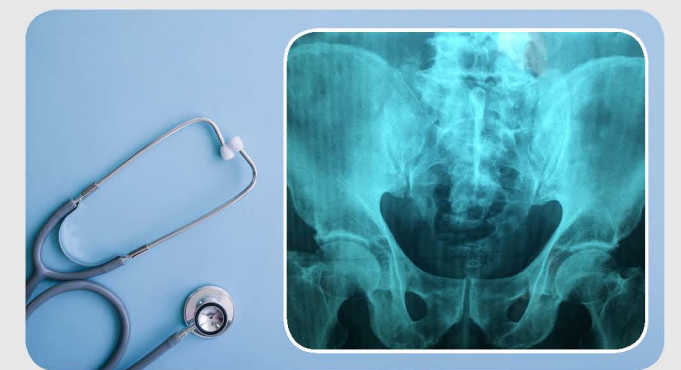


yellow, orange,
or red to proceed
to Step 3

Step 3

REDs clinical diagnosis and treatment

- Physician diagnosis based on information from Steps 1 and 2 along with clinical history and examination
- Individualized treatment plan implemented by the multi-disciplinary athlete health and performance team



REDS DIAGNOSIS WITH ↑ SEVERITY AND/OR RISK CATEGORISATION †

GREEN †

Severity/Risk

None to very low

Clinical Criteria

0 primary indicators
≤ 1 secondary indicator

Treatment, Training & Competition Recommendations

- No treatment required
- Full training and competition clearance

YELLOW †

Severity/Risk

Mild

Clinical Criteria

0 primary & ≥2 secondary indicators OR
1 primary & ≤2 secondary indicators OR
2 primary & ≤1 secondary indicator

Treatment, Training & Competition Recommendations

- Treatment, monitoring and regular follow-up at appropriate intervals.
- Full training and competition.

ORANGE †

Severity/Risk

Moderate to High

Clinical Criteria

1 primary & ≥3 secondary indicators OR
2 primary & ≥2 secondary indicators OR
3 primary & ≤1 secondary indicator

Treatment, Training & Competition Recommendations

- Treatment, close monitoring and follow-up required (e.g., ~monthly).
- Some aspects of training and/or competition may need to be modified.

RED †

Severity/Risk

Very High/Extreme

Clinical Criteria

3 primary and ≥2 secondary indicators OR
≥4 primary

Treatment, Training & Competition Recommendations

- Immediate treatment (± hospitalisation) required by frequent monitoring at ~daily to monthly intervals depending on severity.
- Significant training and competition modifications required, and in the majority of cases, removal from all training and competition is indicated.

† Serious medical indicators of REDs and/or EDs requiring immediate medical attention, potential hospitalization and removal from training and competition (please see table 3), include: ≤ 75% median BMI for age and sex; Electrolyte disturbances; ECG abnormalities (e.g., prolonged QTc interval or severe bradycardia (Adult: HR ≤ 30 bpm; Adolescent: HR ≤ 45 bpm)); Severe hypotension: ≤90/45 mmHg; Orthostatic intolerance (Adult & Adolescent a supine to standing systolic BP drop > 20 mmHg and a diastolic drop > 10 mmHg); Failure of outpatient ED treatment program; Acute medical complications of malnutrition; Any condition on that inhibits medical treatment and monitoring while training and/or competing.

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GLOSSARY OF ACRONYMS

ANF	Anorexia Nervosa Female
ANM	Anorexia Nervosa Male
BM	Body Mass
BMD	Bone Mineral Density
BMI	Body Mass Index
BP	Blood Pressure
BSI	Bone Stress Injury
CAT2	Clinical Assessment Tool 2
CHO	Carbohydrate
DE	Disordered Eating
DSM-5TR	Diagnostic & Statistical Manual-5 th edition Text Revision
DXA	Dual-energy X-ray Absorptiometry
E2	Oestrogen or Oestradiol
EA	Energy Availability
ED	Eating Disorder
EDE-Q	Eating Disorder Examination Questionnaire
EEE	Exercise Energy Expenditure
EI	Energy Intake
ECG	Electrocardiogram
EPO	Erythropoietin
FA	Female Athlete
FFM	Fat-Free Mass
FHA	Functional Hypothalamic Amenorrhea
GH	Growth Hormone
GI	Gastrointestinal
HPG	Hypothalamic-pituitary-gonadal
HR	Heart Rate
IGF-1	Insulin-like Growth Factor-1
IL-6	Interleukin-6
IOC	International Olympic Committee
kcal	Kilocalories
kg	Kilogram
LCA	Low Carbohydrate Availability
LCHF	Low Carbohydrate/High Fat
LDL	Low-Density Lipoprotein
LEA	Low Energy Availability
LH	Luteinising Hormone
MA	Male Athlete
MVP	Mitral Valve Prolapse
OCD	Obsessive-Compulsive Disorder
OTS	Overtraining Syndrome
PCOS	Polycystic Ovarian Syndrome
PCr	Phosphorylated Creatine
REDs	Relative Energy Deficiency in Sport
RAM	RAND-UCLA Method
RMR	Resting Metabolic Rate
SF	Sedentary Female
SM	Sedentary Male
T ₃	Triiodothyronine
TBLH	Total Body Less Head
T	Testosterone
VO ₂ max	Maximal oxygen consumption

IOC consensus statement on REDs

ONLINE SUPPLEMENTAL FILES

- Appendix 1. Summary of original studies in the literature review (2018-2022)
- Appendix 2: Results after the first round of voting on REDs statements
- Appendix 3: Results after the second round of voting on REDs statements
- Appendix 4: Results after two rounds of voting on REDs statements
- Appendix 5: REDs CAT2 Automatic Scoring Tool
- Appendix 6: Reference List

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