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REVIEW ARTICLE

Low Energy Availability in Athletes: A Review of Prevalence, Dietary Patterns, Physiological Health, and Sports Performance

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Abstract In a high-performance sports environment, athletes can present with low energy availability (LEA) for a variety of reasons, ranging from not consuming enough food for their specific energy requirements to disordered eating behaviors. Both male and female high-performance athletes are at risk of LEA. Longstanding LEA can cause unfavorable physiological and psychological outcomes which have the potential to impair an athlete's health and sports performance. This narrative review summarizes the prevalence of LEA and its associations with athlete health and sports performance. It is evident in the published scientific literature that the methods used to determine LEA and its associated health outcomes vary. This contributes to poor recognition of the condition and its sequelae. This review also identifies interventions designed to improve health outcomes in athletes with LEA and indicates areas which warrant further investigation. While return-to-play guidelines have been developed for healthcare professionals to manage LEA in athletes, behavioral interventions to prevent the condition and manage its associated negative health and performance outcomes are required.

Key Points

Advancements in research have revealed low energy availability (LEA) as an unfavorable factor involved in the disruption of physiological processes that may affect health and sports performance.

Research is required to establish a standardized method to measure energy availability and the identification of LEA cut-offs is warranted for both male and females athletes.

Investigations into health outcomes, injury, and illness in athletes with relative energy deficiency/LEA are needed to define potential negative effects and ensure optimal health and sports performance.

1 Introduction

Over the last 30 years, considerable research has been undertaken to understand the cause(s) of menstrual dysfunction and low bone mineral density (BMD), both of which are frequently observed amongst high-performance female athletes. It is widely acknowledged that low energy availability (LEA) is the main factor underpinning these unfavorable health outcomes. LEA occurs when an individual has insufficient energy to support normal physiological function after the cost of energy expended during exercise has been removed. This may occur with/without an eating disorder (ED) or disordered eating (DE) behavior and can have a negative effect on an athlete's health [1, 2]. The female athlete triad (TRIAD) [3] demonstrates the interrelationship between LEA (with/without ED),

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menstrual dysfunction, and poor bone health; it is characterized by a continuum, whereby an individual can move from optimal health to disease, with clinical EDs, functional hypothalamic amenorrhea (FHA), and impaired bone health considered the most harmful characteristics [3, 4].

Little is known about the physiological effects of LEA in male athletes, although it is widely acknowledged that investigation of the physiological issues associated with LEA in this sex group is necessary [3, 5, 6]. The sports medicine literature has documented that LEA has the potential to impair physiological function, beyond menstrual function and bone health (Fig. 1), and that LEA may occur in an energy-balanced state [1, 3, 4, 6, 7]. This concept has recently been referred to as relative energy deficiency in sport (RED-S). For example, an energy-deficient athlete may maintain normal body mass due to physiological adaptations such as decreased resting metabolic rate (RMR); thus, an athlete can be weight stable yet energy deficient. Irrespective of the terminology used, TRIAD or RED-S, both depict LEA as the primary causative factor [8, 9].

Previous research highlights the need to identify the prevalence of LEA, particularly among male athletes, and understand the consequences of LEA on physiological function [7]. LEA may promote susceptibility to respiratory tract infections and adversely affect blood lipid levels. This review discusses what LEA is, how it is currently measured, and the lack of research on potential biomarkers of energy deficiency. Furthermore, the physiological and health issues, dietary patterns, and potential impact on sports performance associated with LEA are examined, and interventions to minimize the deleterious effects of LEA on athletes' health are critically evaluated.

2 Methodology

This is a narrative review which was conducted using targeted internet searches, for example, PubMed, Google Scholar, and Web of Science. Combinations of the following key search terms were included: athlete, bone,

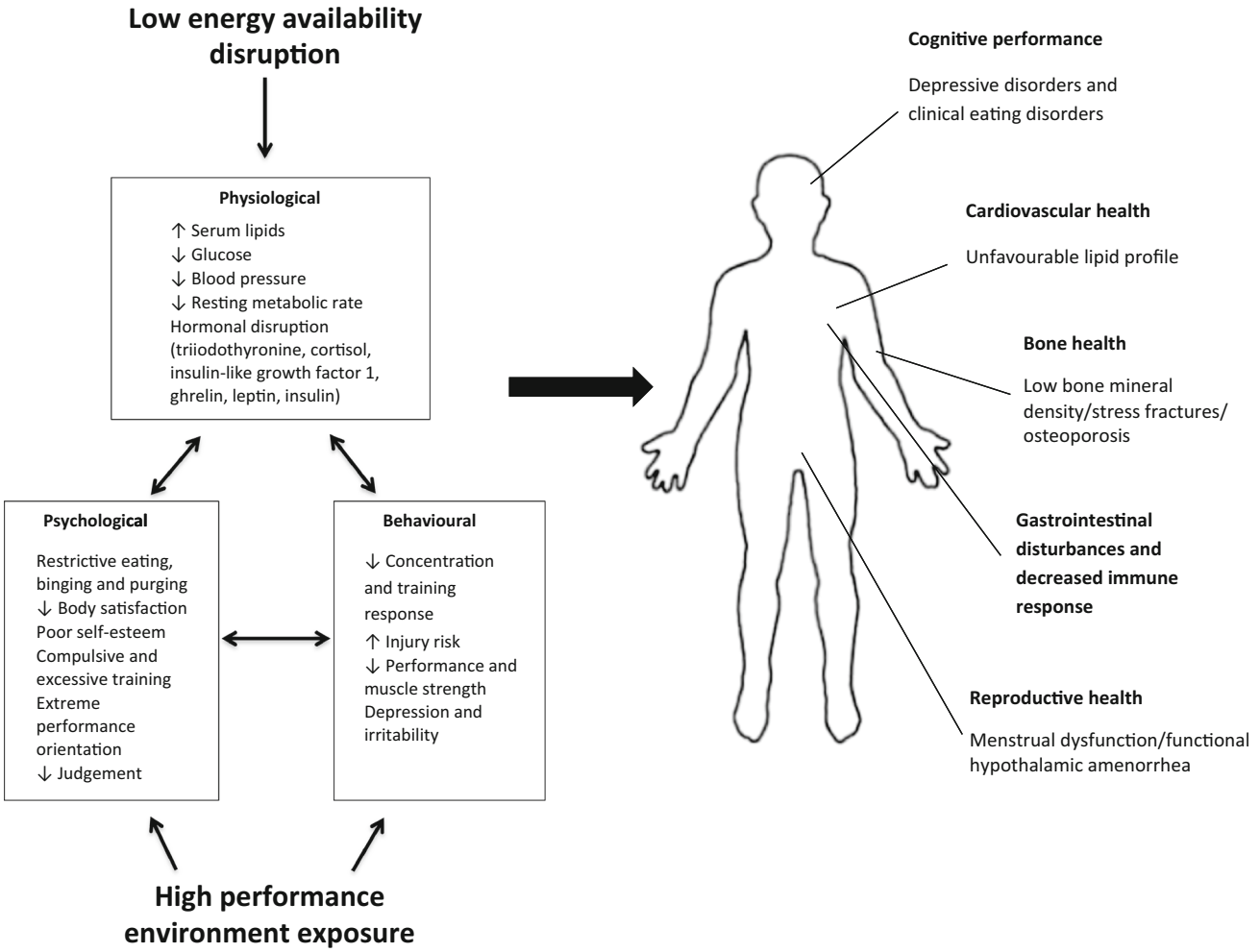

Fig. 1 Low energy availability disruption and high-performance environment exposure: the potential pathways to unfavorable health and performance outcomes

energy availability, energy intake, immune, injury, low energy availability, nutrition education/diet intervention, relative energy deficiency in sport, and weight loss. Articles were considered if they were available in full text, were written in English, and were conducted among trained or exercising human subjects. Only studies that quantified energy availability (EA) by assessing energy intake (EI), exercise energy expenditure (EEE), and body composition within the text of the manuscript were included in this review. Reference lists of articles retrieved were also reviewed. No time limit on retrieval of articles was set. Animal studies were not included. The quality and strength of the supporting evidence was graded according to the criteria of the Scottish Intercollegiate Guidelines Network (SIGN) [10].

3 Energy Availability

EA has been defined as the amount of ingested energy remaining for bodily function and physiological processes such as growth, immune function, locomotion, and thermoregulation after the energy required for exercise/training has been removed [3]. Figure 2 outlines how EA is calculated and the recommended EA thresholds for physically active females. These thresholds originated from experiments in small groups of untrained females that determined the effects of exercise stress and EA on luteinizing hormone (LH) pulsatility and markers of bone turnover [11–14]. Although prospective studies support a causal role of LEA on the suppression of reproductive function in physically active women and female athletes [12, 15, 16], a randomized controlled trial highlights that varying levels of energy deficiency predict the frequency, but not the severity, of menstrual disturbances [17]. Further research is warranted to accurately determine LEA cut-offs, particularly for the athletic population. Studies conducted outside the laboratory setting highlight the complexity in determining EA, which requires measures of EI, EEE, and body composition [2, 18, 19], which are notoriously difficult to measure accurately. The EA recommendation from Loucks and colleagues [11, 12] is particularly problematic when 'purposeful' exercise varies in type and intensity as calculation relies on consistent exercise behavior that is quantifiable in intensity and duration. Furthermore, non-purposeful physical activity expenditure needs to be accounted for to accurately reflect changes in the energy available for physiological processes. Moreover, current recommendations are only pertinent to females and, to our knowledge, no EA recommendations have been proposed for male athletes.

4 Low Energy Availability (LEA), Eating Disorders, and Disordered Eating Behaviors

There are many detrimental effects of decreased EA; those most widely acknowledged are the perturbation of reproductive function and bone metabolism when EA falls below 30 kcal/kg free fat mass (FFM)/day [12, 14]. LEA may be intentional, due to a clinical ED and/or DE behavior. It can also occur unintentionally, due to poor awareness of appropriate sport-specific fueling or re-

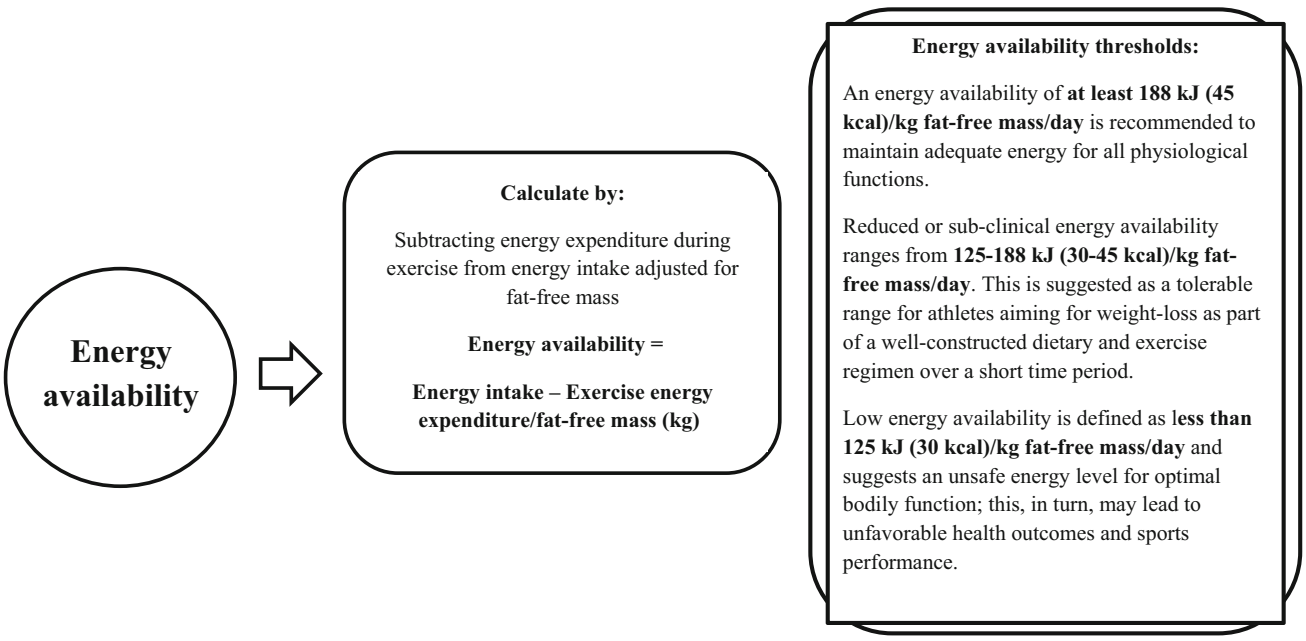

Fig. 2 Energy availability formula and current energy availability thresholds for physically active females [3, 6]

fueling requirements [3, 6]. Regardless of its etiology, LEA may contribute to macro- and micro-nutrient deficiencies and unfavorable physiological changes, potentially resulting in harmful health outcomes and suboptimal sports performance.

4.1 LEA

Table 1 summarizes the prevalence of LEA in a number of sports. Few studies have investigated the prevalence of LEA in male athletes [19, 20]; those doing so report similar prevalence in both sexes [20]. Indeed, the existence of widespread energy deficiency is evident across an array of sports, not just in those that specifically emphasize leanness [18, 20–22]. Nonetheless, accurate estimates of prevalence are problematic due to variability in the sports and groups of athletes (e.g., performance level and age) investigated, as well as small study sample sizes (range 10–352). On the basis of the best available evidence (Grade B: consistent, low-quality evidence), further research is needed to establish a better understanding of the prevalence of LEA in both sexes across all sports.

To date, no gold standard assessment of EA has been agreed. Different methods have been used to assess EI and EEE as part of an EA assessment and to investigate the links with DE, reproductive function, BMD, body composition, and biochemical variables. Some examples are outlined in Table 2. Unfortunately, many methodological issues prevail. Self-reported food and exercise logs lack accuracy, yet are widely used to estimate EA [19, 20, 23–30]. Reduced compliance with self-reported dietary intake has been documented after 4 days [31]. Some researchers have tried to overcome this difficulty by educating athletes on the importance of keeping accurate dietary logs and regularly checking these [18, 32]. Furthermore, the definition of 'exercise' varies, highlighting the need for a standardized definition. Few studies use adjusted EEE (i.e., subtracting the energy cost of sedentary behaviors during the exercise period from EEE) to avoid over-/under-estimating EEE and, thereby, over-/under-estimating EA [33]. Moreover, the majority of studies lack a non-athlete control group. This variability in study design makes it difficult to interpret study results and accurately estimate the extent of the problem. Only one study has assessed dietary information in situ [34]. In this study, all athletes were resident at the training center for the study duration and ate at the same food station each day. Innovative technologies may prove useful to reduce the burden of recording dietary intake and increase the accuracy of EI estimation within an EA assessment [33].

Few studies assessing EA included male athletes [19, 20, 26, 35]. One study, which did not assess EA but instead analyzed biomarkers of nutritional status and serum hormone levels, concluded that males competing in sports that emphasize leanness are characterized by a different body composition and endocrine status than those competing in non-lean sports [36]. In direct contrast to the study conclusion, biomarkers of nutritional status and serum hormone levels were within the normal range (i.e., showed no indication of hypothalamic suppression), thus providing no evidence for LEA in leanness sports. These findings indicate that physiological adaptations to LEA occur within males but they do not manifest as measureable, clinically recognizable changes. The use of different methodologies to determine EA, for example, EI and EEE vs. biomarkers of nutritional status, makes it difficult to compare study results, thus reinforcing the need for appropriate sex-specific tools/biomarkers to clearly identify the extent of LEA among athletes.

Over the last two decades, assessment of individual TRIAD components using questionnaires has occurred (Table 2). In 2014, a screening tool for female athletes, the LEA in Females Questionnaire (LEAF-Q), was devised and validated [37]. With 78% sensitivity and 90% specificity, this tool can be used to detect female athletes 'at risk' of the physiological symptoms associated with LEA. Although it can be used alone, its recommended use is in combination with a validated DE screening tool, for example the Female Athlete Screening Tool (FAST) [38]. Only one study has been published using the LEAF-Q in combination with the FAST in an athlete population [39]; over 40% (44.1%) of female ultra-marathon athletes were found to be 'at risk', with 32% demonstrating DE behaviors. Furthermore, it was demonstrated using six additional questions that 92.5% of the athletes lacked awareness of the TRIAD. The drive for thinness subscale from the Eating Disorder Inventory (EDI) can be considered a proxy indicator of LEA; exercising females with a high drive for thinness score exhibited metabolic adaptations to energy deficiency [40]. No screening tool is available for the assessment of males 'at risk' of LEA; such a tool is urgently required.

4.2 Eating Disorders and Disordered Eating Behaviors

Prevalence rates for EDs are high among elite athletes, particularly female athletes, and those competing in weight-class sports or sports that place emphasis on leanness [41, 42]. EDs also occur more frequently among male athletes than in non-athletic male controls [42]. Athletes most susceptible to developing DE are those who experience pressure to improve performance, to maintain a specific sporting appearance, or to have an 'ideal' physique [43]. Nearly one-quarter of male athletes competing in ED high-risk sports (weight-class sports; sports where leanness

Table 1 Estimated prevalence of low energy availability in various sporting groups

Study	Sex	Sample size	Athletes	Mean age (years)	% participants with LEA[a]	Comments
Observational studies						
Schaal et al. (2016) [34]	F	11	Synchronized swimmers	20.4	Baseline: 100 Intensive training week 2: 100 and week 4: 100	Low EA at each timepoint Significant lower EA at week 4 vs. baseline; $p < 0.05$
Viner et al. (2015) [19]	M/F	10 6 M 4 F	Endurance cyclists	M: 42 F: 38.4	Pre-season: 70 Competition: 90 Off-season: 80	EA did not change across the season ↓ EEE competition: 1133 ± 543 kcal/day vs. off-season: 811 ± 493 kcal/day
Vanheest et al. (2014) [23]	F	10 5 cyclic 5 ovarian suppressed	Elite swimmers	Cyclic: 16.2 Ovarian suppressed: 17	Ovarian suppressed across 12-week season: 100	EA in cyclic group significantly greater vs. ovarian suppressed Cyclic group only in positive energy balance weeks 2 and 4
Reed et al. (2013) [18]	F	19 pre-season 15 mid-season 17 post-season	Division 1 soccer players	19	Pre-season: 26 Mid-season: 33 Post-season: 12	
De Souza et al. (1998) [30]	F	35 24 exercising 11 sedentary Monitored and categorized over 3 menstrual cycles: SedOvul ExOvul ExLPD ExAnov	Exercising Sedentary	Exercising: 27.8 Sedentary: 26.2	Exercising: 100 Sedentary: N/A	EA lower in exercising vs. sedentary; $p < 0.05$ SedOvul vs. ExOvul, ExLPD, and ExAnov: 30 ± 1.2 vs. 23.3 ± 1.6, 26.5 ± 1.8, and 18.8 ± 3.2 kcal/kg FFM/day, respectively
Case–control study						
Schaal et al. (2011) [47]	F	10 5 EU 5 AM	Competitive endurance athletes	EU: 29.8 AM: 31	EU: N/A AM: 100	All AM had low EA
Cross-sectional studies						
Lagowska and Kapczuk (2016) [57]	F	52 31 athletes 21 ballet dancers	Athletes Ballet dancers Both with menstrual disorders	Athletes: 18.1 Ballet dancers: 17.1	Athletes: N/A Ballet dancers: 100	Higher EA in athletes vs. ballet dancers: 28.3 ± 9.2 vs. 21.7 ± 7.2 kcal/kg FFM/day; $p \leq 0.05$
Day et al. (2015) [115]	F	25	Division 1 track/field collegiate athletes	Athletes: 19.5	52 (13 of 25)	92% athletes (23 of 25) <45 kcal/kg FFM/day
Muia et al. (2015) [24]	F	110 61 athletes 49 non-athlete controls	Middle-/long-distance athletes	Athletes: 16 Non-athletes: 17	Athletes: 7.9 Non-athletes: 2.2	76% SC-EA EA lower in athletes vs. non-athletes: 36.5 ± 4.5 vs. 39.5 ± 5.7 kcal/kg FFM/day; $p = 0.003$
Silva and Paiva (2015) [25]	F	67	Rhythmic gymnasts	18.7	44.8	37.3% SC-EA
Melin et al. (2014) [2]	F	40 24 MD 16 EU	Elite endurance athletes	26.3	20	42.5% SC-EA, 37.5% O-EA

Table 1 continued

Study	Sex	Sample size	Athletes	Mean age (years)	% participants with LEA[a]	Comments
Melin et al. (2014) [55]	F	25	Elite endurance athletes	26.6	12	44% SC-EA, 44% O-EA
Gibbs et al. (2013) [46]	F	86 30 high dietary restraint 56 normal dietary restraint	Recreationally active	23	High dietary restraint: 26.7 Normal dietary restraint: 25	EA lower in high dietary restraint group: 35 ± 12.9 vs. 42 ± 12.9 kcal/kg FFM/day; $p = 0.018$
Koehler et al. (2013) [20]	M/F	352 167 M 185 F	Athletes from mixed sports	M: 16.2 F: 16.3	M: 55.6 F: 50.8	EA similar in both sexes
Woodruff and Meloche (2013) [22]	F	10	Volleyball players	20.9	20	60% SC-EA, 20% O-EA
Dolan et al. (2011) [26]	M	27 17 flat 10 hunt	Flat/hunt jockeys	27.3	Competitive race days: 100	EA reported for competitive race days only
Hoch et al. (2011) [61]	F	22	Professional ballet dancers	23.2	77	
Doyle-Lucas et al. (2010) [27]	F	30 15 dancers 15 sedentary controls	Professional ballet dancers	Dancers: 24.3 Sedentary: 23.7	Dancers: 100 Sedentary: 0	Lower EA in dancers vs. controls; $p < 0.01$
Hoch et al. (2009) [28]	F	160 80 athletes 80 sedentary controls	University athletes	Athletes: 16.5 Sedentary: 16.5	Athletes: 6 Sedentary: 4	30% athletes and 35% sedentary with SC-EA

AM amenorrheic, *EA* energy availability, *EEE* exercise energy expenditure, *EU* eumenorrheic, *ExAnov* exercising anovulatory, *ExLPD* exercising luteal phase deficiency, *ExOvul* exercising ovulatory, *F* female, *FFM* fat-free mass, *M* male, *MD* menstrual dysfunction, *N/A* not available, *O-EA* optimal energy availability (>45 kcal/kg FFM/day), *SC-EA* sub-clinical energy availability (30–45 kcal/kg FFM/day), *SedOvul* sedentary ovulatory

[a]<30 kcal/kg FFM/day

improves performance; aesthetic sports) displayed DE behaviors associated with body image dissatisfaction [44]. Similarly, a higher percentage of female athletes in ED high-risk sports (46.7%) had clinical EDs compared with athletes in other sports (19.8%) and non-athletic controls (21.4%) [45].

Few studies have investigated DE behaviors in combination with an assessment of EA (Table 2). Again, interpretation of study findings is difficult due to variability in the methods used to assess EDs/DE behaviors. It has been reported that male athletes demonstrating dietary restraint practices consciously restricted EI as a method of weight control [19]. Extreme weight-loss methods such as use of saunas (86%), excessive exercising to the point of sweating (81%), and dieting (71%) [26] were the most commonly reported behaviors practiced by male jockeys. Lower EA among exercising females and professional dancers with high dietary restraint compared with those with normal dietary restraint is apparent [27, 46]. Greater body dissatisfaction in female soccer players with LEA [18] and in amenorrheic compared with eumenorrheic endurance athletes has been reported [47]. Furthermore, more than 75% of endurance runners were identified as having DE behaviors [24].

In contrast, one study reported that adolescent athletes and sedentary students with LEA had satisfactory eating-attitude test scores, suggesting that those with LEA do not necessarily display ED characteristics [28]. Furthermore, the gold standard ED assessment, Eating Disorder Examination 16 (EDE-16), a semi-structured interview exploring

Table 2 Methods used to assess energy intake and exercise energy expenditure as part of an assessment of energy availability, disordered eating, reproductive function, bone mineral density, body composition, and biochemical variables

Study	Participants (*n*)	Methods used						Biochemical parameters	Other parameters
		Energy intake	Exercise energy expenditure	DE	Reproductive health	BMD	Body composition		
Crossover trials									
Koehler et al. (2016) [35]	6 exercising M	Assigned a 4-day diet depending on condition: Condition 1 = low EA: 15 kcal/kg FFM/day; Condition 2 = energy balance: 40 kcal/kg FFM/day	Accelerometer	N/A	N/A	N/A	BIA	Total testosterone, free T_3, insulin, leptin, ghrelin, glucose, glycerol, FFA	Peak oxygen uptake assessed using incremental exercise test on a bicycle ergometer
Observational studies									
Schaal et al. (2016) [34]	11 synchronized swimmers	Prospective dietary record	Heart rate monitor	N/A	N/A	N/A	7-site skinfold measurements	Salivary samples: cortisol, ghrelin, leptin	Fatigue rating using 7-point RPE scale
Viner et al. (2015) [19]	10 endurance cyclists 6 M 4 F	Prospective dietary record	Activity log	TFE-Q (CRS)	N/A	DEXA	DEXA	N/A	N/A
Vanheest et al. (2014) [23]	10 elite swimmers 5 cyclic ovarian 5 suppressed	Prospective dietary record	Activity log	N/A	Daily diary: questions on menstruation. Menstrual status determined by circulating E2, P4	N/A	4-site skinfold measurements	IGF-1, total T_3	Maximal swim performance time trial: 400 m swim velocity
Reed et al. (2014) [32]	Division 1 F soccer players 19 pre-season 15 mid-season 17 post-season	Prospective dietary record	Heart rate monitor; activity log	N/A	N/A	N/A	DEXA	N/A	VO_{2max} measured on a treadmill using indirect calorimetry
Reed et al. (2013) [18]	Division 1 F soccer players 19 pre-season 15 mid-season 17 post-season	Prospective dietary record	Heart rate monitor; activity log	EDI-2	Health questionnaire	N/A	DEXA	T_3	VO_{2max} measured on a treadmill using indirect calorimetry
De Souza et al. (1998) [30]	24 exercising F 11 sedentary F	Prospective dietary record	Heart rate monitor; activity log	N/A	Menstrual history; urine samples analyzed for FSH, estrone conjugates, pregnanediol-3-glucuronide	N/A	5-site skinfold measurements	Creatinine	VO_{2max} measured by expired metabolic gases during treadmill test

Table 2 continued

Study	Participants (*n*)	Methods used						Biochemical parameters	Other parameters
		Energy intake	Exercise energy expenditure	DE	Reproductive health	BMD	Body composition		
Case–control study									
Schaal et al. (2011) [47]	10 endurance athletes 5 EU 5 AM	Prospective weighed dietary record[a]	Heart rate monitor; activity log	EDE-Q	Menstrual history verified by physician	N/A	DEXA	Glucose, lactate, epinephrine, norepinephrine, cortisol	Maximal treadmill test F/B 30-min recovery with submaximal running test, heart rate, BP, RPE, POMS questionnaire
Cross-sectional studies									
Lagowska and Kapczuk (2016) [57]	52 F athletes/ ballet dancers 31 athletes 21 ballet dancers	Prospective dietary record under dietetic supervision and photographic diary	Heart rate monitor; activity log	N/A	Menstrual history questionnaire; gynecological U/S; sex hormones: LH, FSH, E2, PRL, P4, TSH, testosterone, sex hormone-binding globulin	N/A	BIA	N/A	N/A
Day et al. (2015) [115]	25 division 1 track/field collegiate athletes	24-h food recall	Accelerometer activity log	EAT-26	Menstrual history questionnaire	Stress fracture history	Skinfold measurements	N/A	Nutrition knowledge questionnaire
Muia et al. (2015) [24]	110 middle- and long-distance athletes 61 athletes 49 non-athletes	Prospective weighed dietary record[a]	Activity log	EDI-3 (BBD and DFT); TFE-Q (CRS)	Menstrual history questionnaire	Sahara Clinical Bone Sonometer using calcaneus U/S	Skinfold measurements	N/A	Socio-demographic data: training hours, medically diagnosed stress fractures
Silva and Paiva (2015) [25]	67 rhythmic gymnasts	24-h food recall	Training questionnaire	N/A	Medical and gynecological history	N/A	BIA	N/A	N/A
Melin et al. (2014) [2]	40 elite endurance athletes 24 MD 16 EU	Prospective weighed dietary record[a]	Heart rate monitor; activity log	EDI-3; EDE-16	Menstrual history using LEAF-Q; U/S, sex hormones: E2, P4, LH, FSH, sex hormone-binding globulin, PRL, dehydroepiandrosterone sulfate, androstendione, total testosterone	DEXA	DEXA	Cholesterol: TC, LDL, HDL, TG; blood glucose, cortisol, IGF-1, insulin, leptin, T_3	BP, RMR
Melin et al. (2014) [55]	25 elite endurance athletes	Prospective weighed dietary record[a]	Heart rate monitor; activity log;	N/A	N/A	N/A	DEXA	N/A	N/A
Gibbs et al. (2013) [46]	86 recreationally active F 30 high dietary restraint 56 normal dietary restraint	Prospective weighed dietary record[a]	Heart rate monitor; activity log	TFE-Q	Menstrual history; urinary samples analyzed for: LH, estrone-1-glucuronide, pregnanediol glucuronide	N/A	DEXA	N/A	N/A

Table 2 continued

Study	Participants (*n*)	Methods used						Biochemical parameters	Other parameters
		Energy intake	Exercise energy expenditure	DE	Reproductive health	BMD	Body composition		
Koehler et al. (2013) [20]	352 athletes from mixed sports 167 M 185 F	Prospective dietary record	Activity log	N/A	N/A	N/A	BIA	Leptin, insulin, IGF-1, T_3	N/A
Woodruff and Meloche (2013) [22]	10 volleyball players	Prospective dietary record	Accelerometer	N/A	Menstrual history; all participants EU	N/A	Air-displacement plethysmography:(Bod Pod)	N/A	N/A
Dolan et al. (2011) [26]	27 jockeys 17 flat 10 hunt	Prospective dietary record (during a 'typical race week')	Accelerometer	N/A	N/A	N/A	DEXA	N/A	Diet, health, and lifestyle questionnaire: weight control methods and timeframes, perceived negative effects of making weight, habitual sensations of hunger and thirst
Hoch et al. (2011) [61]	22 professional ballet dancers	Prospective dietary record	Accelerometer	EDE-Q	Menstrual history questionnaire; sex hormones: FSH, LH, P4, E2, thyrotropin, PRL, beta-human chorionic gonadotropin	DEXA	DEXA	N/A	Endothelial function: brachial artery flow-meditated vasodilation and velocity measured by high-frequency U/S
Doyle-Lucas et al. (2010) [27]	30 professional ballet dancers 15 dancers 15 sedentary controls	Prospective dietary record	Activity log	TFE-Q, EAT-26	Menstrual history questionnaire	DEXA	DEXA	N/A	RMR
Hoch et al. (2009) [28]	80 university athletes 80 sedentary controls	Prospective dietary record	Activity log	EAT-26	Menstrual history questionnaire; sex hormones: PRL, TSH, FSH, E2, LH	DEXA	DEXA	N/A	N/A

Table 2 continued

Study	Participants (*n*)	Methods used						Biochemical parameters	Other parameters
		Energy intake	Exercise energy expenditure	DE	Reproductive health	BMD	Body composition		
Thong et al. (2000) [29]	39 elite athletes/ recreationally active F grouped according to menstrual status 5 EAA 8 ECA 13 RCA 13 ROC	Prospective dietary record	Activity log	N/A	Sex hormones: E2, P4, 17a-ethinylestradiol	N/A	Underwater weighing	Leptin, insulin, total T_3, total thyroxine	N/A

AM amenorrheic, *BBD* Bulimia and Body Dissatisfaction subscale, *BIA* bio-impedance analysis, *BMD* bone mineral density, *BP* blood pressure, *CRS* Cognitive Restraint subscale, *DE* disordered eating, *DEXA* dual-energy X-ray absorptiometry, *DFT* Drive for Thinness subscale, *E2* estradiol, *EA* energy availability, *EAA* elite amenorrheic athletes, *EAT-26* Eating Attitudes Test, *ECA* elite cyclic athlete, *EDE-16* Eating Disorder Examination 16, *EDE-Q* Eating Disorder Examination Questionnaire, EDI Eating Disorder Inventory, *EU* eumenorrheic, *F* female, *F/B* followed by, *FFA* free fatty acids, *FFM* fat-free mass, *FSH* follicle-stimulating hormone, *HDL* high-density lipoprotein, *IGF-1* insulin-like growth factor, *LEAF-Q* Low Energy Availability in Females Questionnaire, *LDL* low-density lipoprotein, *LH* luteinizing hormone, *M* male, *MD* menstrual dysfunction, *N/A* not available, *P4* progesterone, *POMS* Profile of Mood States, *PRL* prolactin, *RCA* recreationally active woman who are cyclic, *ROC* recreationally active woman taking oral contraceptives, *RMR* resting metabolic rate, *RPE* rate of perceived exertion, T_3 triiodothyronine, *TC* total cholesterol, *TFE-Q* Three Factor Eating Questionnaire, *TG* triglycerides, *TSH* thyroid-stimulating hormone, *U/S* ultrasound examination, VO_{2max} maximal oxygen consumption

[a]Weighed dietary records provide a detailed estimate of intake for individuals which can be used for estimation of actual portion sizes

key psychopathological and behavioral features of EDs, identified ED/DE in only 7 of 25 female athletes with clinical or sub-clinical LEA [2]. Identifying athletes with EDs and/or DE behaviors does not appear to be sufficiently sensitive to indicate LEA. This emphasizes the need to investigate excessive exercise as an indicator of LEA.

Validated screening tools such as the EDE-16 are available to screen for DE in the general population, some of which have been used with athletes (Table 2). Recent emphasis has been on the development of athlete-specific DE screening tools. Along with the validated DE screening tool (FAST) mentioned in Sect. 4.1, the latest screening tool developed for female athletes, the Brief ED in Athletes Questionnaire (BEDA-Q), complements the LEAF-Q and can be used to identify female athletes with or without an ED [48]. No athlete-specific screening tool has yet been developed to assess EDs/DE behaviors or the physiological symptoms of LEA in the athletic male population.

5 Biomarkers of Energy Deficiency

Identifying unintentional LEA can be problematic as the signs and symptoms are difficult to detect. The use of validated biomarkers associated with LEA could provide a quick method of monitoring energy status and identifying athletes potentially 'at risk' of energy deficiency. Biomarkers suggested include leptin, triiodothyronine (T_3), and cortisol [49]. Table 3 outlines the small number of studies that have investigated metabolic substrates and hormone levels in athletes who exhibited LEA. Evidence for an association between LEA (<30 kcal/kg FFM/day) and metabolic substrates/hormones is not particularly strong, with weak or conflicting data reported in studies in athletic populations.

5.1 Appetite Hormones: Leptin and Ghrelin

Leptin (appetite-suppressing hormone), a marker of low body fat and restricted food intake [49], appears to be reduced when EA is low, perhaps indicating inadequate recovery from exercise and relative energy deficiency. A study of healthy exercising females demonstrated that the pulsatility of leptin is dependent on EA and not exercise-induced stress; exercise had no suppressive effect on the diurnal rhythm of leptin when EI was adequate [50]. In contrast, another study showed no difference in leptin level between endurance female athletes, regardless of their EA status [2]. Studies investigating ghrelin (appetite-stimulating hormone) levels have also reported mixed findings, with both lower EA [34] and normal EA [35] associated with higher ghrelin level. One study of healthy females reported a significant increase in fasting ghrelin

concentrations following a decrease in EA during a 3-month diet and exercise intervention [51]. Furthermore, 3-month diet and exercise interventions in healthy females that elicited a significant decrease in body weight were associated with increases in fasting ghrelin [52–54]. Further research to determine if changes in leptin and ghrelin levels are sensitive enough to identify changes in EA are required.

5.2 Triiodothyronine

T_3 (involved in the hypothalamic–pituitary–thyroid axis and responsible for regulation of metabolism) levels have been explored as a biomarker of EA, with studies collectively presenting mixed results. Lower T_3 levels were observed in ovarian hormone-suppressed athletes [23] and among female athletes who had lost weight [20]. Although T_3 levels did not differ between endurance-trained females with a different EA status (optimal, sub-optimal, and LEA), lower T_3 levels were reported among athletes with menstrual dysfunction than in eumenorrheic athletes [2]. These study results indicate that T_3 levels decrease in female athletes with menstrual irregularities. LEA did not significantly influence T_3 levels in exercising men [35] or in female soccer players [18]. Further research is necessary to investigate whether T_3 levels can be used to reflect changes in EA.

5.3 Cortisol

Although cortisol (a steroid hormone released in response to stress) levels were similar among elite female endurance athletes regardless of EA status (optimal, sub-optimal and LEA respectively) [2], higher levels have been observed among those with menstrual dysfunction compared to eumenorrheic athletes [2, 47]. Moreover, exercising at different intensities appears to influence cortisol levels [47]. Although no significant changes in cortisol levels were observed in a group of elite synchronized swimmers across a 4-week intensive training period, direct correlations between cortisol levels and perceived fatigue suggest greater physiological stress among energy-deficient swimmers [34]. In summary, elevated cortisol levels suggest greater physiological stress during intensive training, with this being more pronounced in females with menstrual irregularities.

5.4 Insulin-Like Growth Factor 1

A marked decline in insulin-like growth factor 1 (IGF-1) (supports cell division and growth) was apparent in ovarian hormone-suppressed female swimmers compared with eumenorrheic swimmers. However, even in those with normal menstrual function, IGF-1 significantly declined over a 12-week season [23]. Furthermore, IGF-1 decreased in untrained females when EI was restricted to 10, 20, or 30 kcal/kg FFM/day [12]. This suggests that lower IGF-1 concentrations could indicate inadequate EA and excessive training in females. In contrast, no relationship between IGF-1 and EA has been established in males. Similar IGF-1 levels were reported in young elite male and female athletes with normal or LEA [20]. In summary, although there may be suggestive evidence for an association between IGF-1 and LEA, this needs to be investigated in highly trained male and female athletes before promotion of IGF-1 as a biomarker of LEA.

5.5 Insulin and Glucose

Similar insulin levels were reported in male and female athletes with low or normal EA [20], whilst insulin and fasting glucose levels were equivalent in female endurance athletes with low ($\leq$30 kcal/kg FFM/day), reduced (30–45 kcal/kg FFM/day) or optimal ($\geq$45 kcal/kg FFM/day) EA [2]. Following 4 days of LEA (15 kcal/kg FFM/day) in exercising men, reduced insulin levels were observed [35]. Increases in glycerol and free fatty acid concentrations and reductions in fasting glucose were also observed in this state of LEA (15 kcal/kg FFM/day). These findings suggest that insulin has increased sensitivity when EA is chronically low. Lower fasting glucose levels were also reported among those with menstrual dysfunction than in eumenorrheic athletes [2]. However, resting blood glucose levels were similar in athletes with amenorrhea and those who were eumenorrheic [47]. Currently, it can only be deduced that female athletes with menstrual irregularities appear to have lower blood glucose levels, which could be suggestive of greater overall physiological stress; further work is necessary to achieve consensus.

5.6 Summary

The viability of biomarkers of energy deficiency is unclear, with questions around appropriate assessment of EA, defined EA cut-offs, and standardized techniques impeding the quality of research in this area. These need to be considered in order to accurately determine the viability of biomarkers of energy deficiency. Although further research, especially with respect to the appetite hormones, is required, this represents an interesting area of investigation.

6 LEA and Dietary Intake in Athletes

Athletes should be encouraged to consume a wide variety of foods on a regular basis. Athletes with LEA reported lower energy density and lower percentage energy from fat

Table 3 Studies investigating associations between energy availability and biochemical parameters

Study	Participants (*n*)	Biochemical parameters
Crossover trials		
Koehler et al. (2016) [35]	6 exercising M	Testosterone, T_3, insulin, leptin, ghrelin, glucose, glycerol, free fatty acids
Observational studies		
Schaal et al. (2016) [34]	11 synchronized swimmers	Salivary samples: cortisol, ghrelin, leptin
Vanheest et al. (2014) [23]	10 elite swimmers 5 cyclic 5 ovarian suppressed	IGF-1, T_3
Reed et al. (2013) [18]	Division 1 F soccer players 19 pre-season 15 mid-season 17 post-season	T3
Case–control study		
Schaal et al. (2011) [47]	10 endurance athletes 5 EU 5 AM	Glucose, lactate, epinephrine, norepinephrine, cortisol
Cross-sectional studies		
Melin et al. (2014) [2]	40 elite endurance athletes 24 MD 16 EU	Cholesterol: TC, LDL, HDL, TG; blood glucose, cortisol, IGF-1, insulin, leptin, T_3
Koehler et al. (2013) [20]	352 athletes from mixed sports 167 M 185 F	Leptin, insulin, IGF-1, T_3
Thong et al. (2000) [29]	39 elite athletes and recreationally active F grouped according to menstrual status 5 EAA 8 ECA 13 RCA 13 ROC	Leptin, insulin, T_3, thyroxine

AM amenorrheic, *EAA* elite amenorrheic athletes, *ECA* elite cyclic athlete, *EU* eumenorrheic, *F* female, *HDL* high-density lipoprotein, *IGF-1* insulin-like growth factor, *LDL* low-density lipoprotein, *M* male, *MD* menstrual dysfunction, *RCA* recreationally active woman who are cyclic, *ROC* recreationally active woman taking oral contraceptives, *TC* total cholesterol, *TG* triglycerides, *T_3* triiodothyronine

(28%) than those with optimal EA (31%) [55]. Female soccer players who exhibited LEA consumed a lower EI at lunch during competition, pre-, and mid-season and at dinner mid-season than did those with optimal EA [18]. Methodological issues, such as reliance on self-reported food diaries, failure to compare dietary intakes with non-athletic controls who have optimal EA, and small sample sizes, make comparison between studies difficult [19, 25, 26, 32, 55].

6.1 LEA and Macronutrient Intakes

In individuals with LEA, total EI is reduced which, subsequently, negatively influences diet quality. As low carbohydrate intake (6–10 g/kg/day for athletes exercising at moderate to high intensity [56]) is commonly reported in athletes, it is not surprising that it has been observed in athletes identified with LEA [19, 26, 32, 55, 57]. A low-carbohydrate, high-fiber diet was reported among female endurance athletes with FHA when compared with eumenorrheic athletes; thus, a diet that exceeds the upper limit for dietary fiber may indicate risk of LEA [55]. The erratic restriction of carbohydrate observed among athletes may be influenced by media-driven fad diet trends such as the 'gluten-free' and 'paleo' diets that promote the elimination of carbohydrate-rich foods [19].

Although inadequate intake of all macronutrients has been observed in female gymnasts [25], the evidence for low protein intakes in athletes with LEA is not consistent. Jockeys were shown to meet their protein requirements

(1.3 g/kg/day) [26] and all but one female endurance athlete met or exceeded the recommended protein intake for endurance-trained athletes [55] of 1.2–1.7 g/kg/day [55]. Of these female athletes, 71% with LEA had protein intakes ranging from 1.8 to 2.0 g/kg/day, the recommended amount to minimize loss of FFM during energy deficiency [55]. Hence, excessive consumption of either fiber or protein may indicate increased risk of LEA [55].

6.2 LEA and Micronutrient Intakes

Inadequate intake of several essential micronutrients, such as vitamins A and C, riboflavin, folate, calcium, and zinc, have been documented in male jockeys and endurance-trained females [26, 55]. A mean consumption of 0.9 servings of fruit and vegetables per day was reported in male jockeys [26], indicating the need to educate athletes on appropriate nutritional strategies and the importance of meal timing and re-fueling following exercise. Micronutrient inadequacies are also common among female gymnasts, with low intakes of folate, pantothenic acid, vitamins D, E and K, calcium, iron, and magnesium reported [25]. Similar deficiencies were reported among cyclists [19]. Methodological problems, such as misreporting [58], make accurate measurement of dietary intake extremely difficult, particularly among athletes involved in weight- or lean-dependent sports who are more susceptible to misreport EI [59].

Nevertheless, it is essential to monitor EI and EEE to avoid a state of LEA, to allow for optimization of diet quality and to ensure athletes are meeting nutrient recommendations relative to their sport. Encouraging carbohydrate consumption for performance and recovery to ensure muscle glycogen stores are replenished is important [56]. Furthermore, personalized nutrition education is vital; for example, the low EI observed amongst jockeys is consistent with their need to maintain a low body mass for competition [26].

7 Physiological and Health Issues Associated with LEA

7.1 Reproductive Function

The frequency at which the pituitary gland secretes LH into the circulatory system is a proxy indicator of the central modulation of the reproductive axis [60]. Luteinizing pulsatility in an exercising woman is solely dependent on EA and is not affected by the stress of exercise itself. Prolonged LEA (10 kcal/kg FFM/day) reduces luteinizing pulsatility [11] and studies in athletes consistently report negative effects of LEA such as perturbed reproductive function [2, 23, 24, 57, 61, 62]. Such athletes have a lower RMR than athletes with good EA and normal menstrual function [2]. Endocrine changes, including high testosterone levels, have also been observed in female athletes (29%) and dancers (85.7%) with menstrual disorders which, furthermore, were associated with LEA and inadequate carbohydrate and EI [57]. Suppressed ovarian steroids (estradiol and progesterone), low metabolic hormones (T_3 and IGF-1), and low energy status markers (LEA and low EI) are highly correlated with a decrease in sports performance [23]. Although the impact of LEA on endocrine function in male athletes is not well-documented and warrants further research, male athletes who habitually engage in endurance exercise training exhibit persistently low/reduced testosterone levels [63].

Self-reported menstrual history is the most commonly used technique to diagnose a clinical menstrual disorder (Table 2). This can be used in combination with a single measurement of sex hormones [28, 61]. More recently, a number of studies have included a gynecological ultrasound examination in the diagnostic assessment [2, 47, 57, 62, 64]. The recent 2014 TRIAD Coalition Consensus Statement outlines an amenorrhea algorithm that recommends a diagnosis of exclusion, whereby a history and physical examination, a series of clinical and endocrine tests, and diagnosis by a physician are required to rule out pregnancy and endocrinopathies [4]. However, given the cost of gynecological function assessment, a standardized method to assess EA would be both clinically and economically advantageous.

7.2 Bone Health

The evidence supporting the benefits of vitamin D and calcium for bone health are widely accepted [65]; thus, it is critical that appropriate nutritional practices are adopted to ensure BMD is maintained. Bone formation is suppressed once EA decreases below 30 kcal/kg FFM/day [14]. Energy deficiency exerts a suppressive effect on bone formation whilst estrogenic deficiency contributes to up-regulation of bone reabsorption [14]; thus, both contribute independently and synergistically to bone loss. For females competing in weight-bearing sports, the American College of Sports Medicine (ACSM) has defined low BMD as a z-score of less than -1.0; however, a defined criterion has not been established in male athletes [3]. Prevalence of low BMD among female athletes ranges from 0 to 15.4% using a z-score of -2.0 or less. This increases to 39.8% when z-scores are defined as between -1.0 and -2.0 [21].

Studies in athletes have rarely assessed BMD using dual-energy X-ray absorptiometry (DEXA) in combination with EA assessment (Table 4). Although DEXA is expensive, it is acknowledged as the gold standard

assessment of BMD, is used to determine the extent and severity of osteoporosis and osteopenia, and can predict fracture risk. A best-practice DEXA protocol should be followed to accurately assess bone changes in athletes [66]. Male and female athletes competing in endurance sports and those sports that emphasize leanness appear to have low BMD. However, despite the persistence of LEA and presence of low BMD among a group of cyclists, BMD did not further reduce over a 10-month cycling season [19]. Although non-weight-bearing sports, such as cycling, can influence BMD, these findings highlight that poor bone health develops over a long period, suggesting that changes in BMD may not be detectable over a short timeframe and that previous exercise (jumping) and dietary (vitamin D and calcium supplements) interventions may stimulate bone mineralization sufficiently to maintain BMD over the period of LEA.

Although the positive impact that the mechanical loading from high-impact exercise has on bone health is irrefutable, irregular menstruation, running in five or more seasons, intentionally restricting dietary intake, and belief that thinness leads to improved performance were associated with low BMD in adolescent endurance runners [67–69]. With increased risk of stress fractures among female endurance athletes [70], it is vital to implement appropriate nutritional practices that meet individual energy needs as a means of optimizing bone health as well as achieving healthy hormonal status and menstrual function and improving body composition [71]. Although the impact of LEA on reproductive function and BMD is not well-documented among male athletes [7], indicators such as low testosterone and estradiol levels were found to be associated with low BMD and indicative of stress fractures [72, 73]. Jockeys who engaged in extreme weight loss practices had an elevated rate of bone loss and reduced BMD, which appeared to be associated with disrupted hormonal activity, for example, elevated sex hormone-binding globulin; this causes a decrease in the availability of biologically active testosterone [74]. Evidence of LEA [26], together with disrupted hormonal activity and low BMD [74], suggest that male athletes can be energy deficient and demonstrate symptoms that reflect both the TRIAD and have been identified by the International Olympic Committee as indicative of RED-S [3, 6].

The potential for low BMD to increase the incidence of injury needs consideration. As stress fractures develop from recurring excessive strain caused by repetitive microtrauma to bone at a rate greater than repair [75], it is important to detect the strains and sprains that athletes experience as soon as possible. Increased injury risk associated with components of the TRIAD has been observed [76–78], particularly among younger athletes. In contrast, one study investigating overuse injuries found no associations between these, menstrual irregularity, and/or DE [79]. This study only observed associations between higher training load (higher mileage) and injury in males. These discordant results may be due to the methods used to assess DE and injury, the athlete type investigated and sample size. In studies on this topic, 'musculoskeletal injury' has either not been defined [80] or has been defined as "an injury from either overuse or direct trauma that occurred during participation in the current sport season" [78]. A standardized definition of 'injury' is required to enable accurate interpretation of future research studies and will permit work that can determine if links between LEA and injury exist. Furthermore, the recording of epidemiological data on injuries warrants attention; the majority of previous surveillance studies have focused on the etiology of 'medical-attention' and/or 'time-loss' injury/illness. Few studies have related these to athletes' subsequent training limitations; this has resulted in the underreporting of 'performance restriction'-type injuries, whereby athletes continue training yet incur performance detriments [81]. Further research is needed to accurately quantify injury incidence; this will help to determine associations between LEA and injury and inform injury/illness prevention initiatives in sport.

7.3 Immune Function

Eating a varied diet that meets athletes' energy needs can help maintain an effective immune system [82]. Normal immune response can be suppressed by a variety of factors such as, but not limited to, insufficient nutrient intake, lack of sleep, psychological and environmental stress, and prolonged bouts of high-intensity exercise; hence, the cause of symptoms of illness among athletes is inevitably multifactorial [82–84]. Particularly when EEE is high, athletes are more susceptible to infectious agents [84]. From a health perspective, and as sports performance is influenced by days and weeks lost to injury and illness [85], preventative measures need to be implemented to ensure adequate energy to minimize these adverse health events in an elite performance environment.

Catecholamines regulate immune and inflammatory responses and are released by the sympathetic nervous system and the adrenal medulla. They cause an increase in the contraction and conduction velocity of cardiomyocytes, resulting in increased cardiac output and a rise in blood pressure, ultimately increasing vascular tone and resistance [86]. It has been speculated that the reduced catecholamine (epinephrine and norepinephrine) response, observed in amenorrheic athletes, could be an adaptive mechanism that preserves energy in order to promote survival by suppressing non-essential physiological processes in a state of LEA [47]. Norepinephrine and epinephrine are key

Table 4 Methods used to assess bone mineral density among athletes in studies investigating energy availability

Study	Participants (*n*)	BMD assessment method	Comment on prevalence of low BMD
Viner et al. (2015) [19]	10 endurance cyclists 6 M 4 F	DEXA	All cyclists with low EA had low BMD, lumbar spine ($n = 4$), femoral neck ($n = 1$)
Day et al. (2015) [115]	25 division 1 track and field collegiate athletes	Stress fracture history	8 had a history of stress fractures
Muia et al. (2015) [24]	110 middle- and long-distance athletes 61 athletes 49 non-athletes	Sahara Clinical Bone Sonometer using U/S calcaneus	No difference in BMD between groups. Reported stress fractures similar in both groups (16 vs. 10%)
Melin et al. (2014) [2]	40 elite endurance athletes 24 MD 16 EU	DEXA	Impaired bone health ($n = 18$): osteoporosis ($n = 3$), low BMD ($n = 15$), menstrual dysfunction ($n = 12$), ED/DE ($n = 6$)
Hoch et al. (2011) [61]	22 professional ballet dancers	DEXA	Low BMD (z-score ≤ -1.0) ($n = 7$) Low BMD in >1 location ($n = 5$)
Doyle-Lucas et al. (2010) [27]	30 professional ballet dancers 15 dancers 15 sedentary controls	DEXA	Spine z-scores for dancers with menstrual dysfunction showed signs of low BMD
Hoch et al. (2009) [28]	80 university athletes 80 sedentary controls	DEXA	16% athletes vs. 30% of sedentary controls had low BMD

BMD bone mineral density, *DE* disordered eating, *DEXA* dual-energy X-ray absorptiometry, *EA* energy availability, *ED* eating disorders, *EU* eumenorrheic, *F* female, *M* male, *MD* menstrual dysfunction, *U/S* ultrasound

hormones that prepare the body for one of its most primeval reactions: the 'fight or flight' response [86]. As reductions in catecholamine responses also correlate with lower peak blood lactate, fewer menstrual cycles and higher EEE, catecholamine responses to maximal exercise (and/or reduced lactate) may be suitable as biomarkers of inadequate EI [87].

Table 5 summarizes the studies that examined the effects of short-term dieting/rapid weight loss and exercise training on immunological parameters. Despite the lack of conclusive evidence of the effects of LEA on immune function, mucosal immunity appears to be altered in weight class sports, in which athletes intermittently use rapid weight loss methods in combination with intensive training [88–90]. The alterations in immunoglobulins, in combination with rapid weight loss, suggest that these athletes are more susceptible to infectious illnesses. Disrupted neutrophil function, reactive oxygen species production, and increased phagocytic activity are observed, demonstrating compensatory mechanisms in order to maintain immunological homoeostasis [91–93]. Furthermore, repetitive weight cycling appears to alter levels of salivary immunoglobulin A (IgA) during training, competition, and recovery periods [94]. Salivary IgA prevents attachment of external pathogens and toxic molecules to mucosal surfaces and, thus, plays a key role in mucosal immunity [95]. It is not surprising that the incidence of upper respiratory tract infection was significantly increased after competition in taekwondo athletes with low salivary IgA [94, 96]. Recently, it has been suggested that monitoring salivary IgA secretion can identify athletes at risk of upper respiratory symptoms [83]. Further research is recommended to more precisely identify the relationship between LEA, IgA, and other immunological markers.

7.4 Cardiovascular Health

Endothelial dysfunction can be classed as the earliest detectable stage of cardiovascular disease (CVD) [97]. Normal vascular endothelium is essential for the production of nitric oxide (NO). Cardiovascular health is influenced by NO, which acts as a vascular protector by playing a key role in preventing platelet aggregation, leukocyte adhesion, and vascular smooth muscle proliferation and migration [98]. Estrogen also plays a key role in the vascular endothelial NO signaling system. Associations between reduced flow-mediated dilation (FMD) and amenorrhea [99, 100] have been observed. However, investigations carried out in dancers suggest that FMD is not simply a function of circulating estrogen concentrations

as reduced FMD was observed in amenorrheic dancers as well as in some eumenorrheic and hormonal contraceptive-using dancers [61]. Of those dancers identified with LEA, 71% had reduced FMD. These findings suggest that in a state of LEA, regardless of estrogen levels, endothelial dysfunction can occur [61, 99].

There has been intense discussion around the etiology of hypercholesterolemia in patients with anorexia nervosa; it has been suggested that low total T_3 and high cortisol levels in a state of undernutrition may be contributory factors for the increase in certain pro-inflammatory markers such as interleukin (IL)-6 and apolipoprotein (Apo)-B, which are known to predict increased CVD risk [101]. Other researchers speculate that starvation results in an increased synthesis of lipoproteins, contributing to an unfavorable lipid pattern in patients with anorexia nervosa [102]. Increases in Apo-A1, Apo-C2, Apo-E, and cholesterol ester transfer protein (CETP) activity have been observed, suggesting accelerated cholesterol synthesis which indicates a metabolic basis for hypercholesterolemia among anorexic patients compared with age-matched controls.

In amenorrheic athletes compared with other athlete groups [100], unfavorable lipid profiles [higher total cholesterol and low-density lipoprotein (LDL) cholesterol] have been reported. This supports the premise of a relationship between LEA and development of CVD risk factors.

Furthermore, energy deficiency may accelerate changes in cholesterol synthesis. High total cholesterol levels were recently observed among endurance athletes with LEA and/or EDs/DE behavior (73%) [2]. Although a 7-day dietary restriction in male judo players did not influence changes in total, LDL or high-density lipoprotein (HDL) cholesterols, it negatively influenced triglyceride and free fatty acid levels [103]. Similarly, higher free fatty acid concentrations were reported in exercising males when subjected to 15 kcal/kg FFM/day for 4 days [35]. From the evidence reviewed, it is probable that the type of sport (endurance vs. weight class) and the length of time spent in an energy deficient state may influence lipid levels.

8 Potential Impact of LEA on Sports Performance

The maintenance of dietary restriction for a long period appears to detrimentally affect sports performance through the depletion of glycogen stores. This, in turn, causes a premature reduction in physical, psychological, and mental capacity, including increased risk of dehydration and higher circulatory lactate, both of which can produce muscular pain, cramps, and/or a reduction in FFM, leading to a reduction in muscular strength and aerobic performance [104, 105]. Thus, LEA can contribute to poor sports performance due to the loss of fat and lean body mass, electrolyte abnormalities, and dehydration [105]. A decrease in performance by 9.8% was observed in swimmers with LEA in contrast to an 8.2% increase in performance in those with adequate EA [23]. These results support previous literature that indicates that long-term energy restriction in athletes increases their risk of compromised sporting performance [106–108].

9 Nutrition Interventions to Improve Health Issues Associated with LEA

As consensus statements have previously addressed the treatment and return to play of athletes with health issues associated with LEA in great depth [4, 6], Table 6 goes beyond this by exploring specific interventions conducted to help minimize the deleterious effects of LEA on athletes' health and performance [62, 109–113]. Current data on the nutritional practices of athletes highlight the need to educate them about the suppressive effects of acute exercise on food intake and its relationship with well-being. The evidence for the effectiveness of interventions on dietary pattern in athletes presents a mixed picture (Table 6). Some studies have reported improvements [62, 110, 111, 113, 114] and others no improvement [115] in EI. Furthermore, increased EI did not always translate into improved EA [111]. The methods used to measure and calculate EEE and, hence, to determine EA, may, at least in part, contribute to the equivocal results reported [111]. It is worth noting that solely educating athletes on nutrition may not always translate into behavioral changes that optimize EI. Although improvements in nutritional knowledge were observed in athletes with low and sub-optimal EA following six interactive nutritional education group sessions which focused on the TRIAD and healthy body image, this did not translate into increased caloric intake [115]. In contrast, significant improvements in EI were reported following individualized nutrition intervention [114]. The educational strategies employed in these studies may also contribute to the conflicting results reported. Nutritional counselling, in combination with strength training, has been recommended as a method of increasing lean body mass or achieving weight gain as it appeared to minimize some practical challenges, including planning and timing of dietary intake and the appropriate amount of food needed to avoid excess body fat [109, 116]. Thus, EI as part of a weight gain plan should be carefully considered to increase lean body mass [109].

A 3-month dietary intervention did not achieve resumption of menses in female athletes with menstrual dysfunction [62], although resumption of menses was

Table 5 Effects of short-term dieting/rapid weight loss and exercise training on immunological parameters

Study	Sex	Participants (*n*)	Duration	Study purpose	Methods	Outcomes
Randomized controlled trial						
Abedelmalek et al. (2015) [88]	M	11 judo	7-day CR	Effect of CR on immune and hormonal responses	Fitness testing: SJFT Blood biomarkers: Hormones: growth hormone, testosterone, cortisol Inflammatory mediators: IL-6, TNF-α White blood cells: leukocytes, lymphocytes, neutrophils	CR outcomes: ↓ BW, performance, testosterone ↑ SJFT index, heart rate, TNF-α, IL-6, cortisol, growth hormone, macronutrient intake ET outcomes: ↑ testosterone, cortisol, growth hormone, leukocytes, neutrophils, TNF-*a*, IL-6
Observational studies						
Shimizu et al. (2011) [89]	M	6 judo	Approx. 1 month pre-competition and 1 day post-competition	Effects of WL on immune function	Illness symptoms: URTI symptoms Blood biomarkers: Monocyte and T cell subpopulations: $CD3^+$, $CD4^+$, $CD8^+$, $CD56^+CD3^-$, $CD28^+CD4^-$, $CD28^+CD8^+$, (TLR-4) CD14 cells	WL period: ↓ $CD3^+$, $CD4^+$, $CD8^+$, $CD28^+$CD4 cell counts, (TLR-4) CD14 cells
Tsai et al. (2011) [94]	M	16 taekwondo	Approx. 1 month pre- and post-competition	Effects of prolonged intensive training and RWL on immunological parameters and antioxidant activity	Incidence of URTI Salivary parameters: sIgA, cortisol, lactoferrin, FRSA	↓ BW before competition ↓ sIgA intermittently ↑ Risk of infection
Tsai et al. (2011) [96]	F	10 taekwondo 5 RWC 5 non-RWC	Approx. 1 month pre- and post-competition	Effects of prolonged intensive training with/without RWL on immunological parameters	Salivary parameters: sIgA, cortisol, lactoferrin	↓ sIgA levels and cortisol in RWC group before competition Non-RWC showed ↓ lactoferrin after competition
Kowatari et al. (2001) [91]	M	18 judo	Approx. 2 weeks pre- and 1 week post-competition	Effects of WR as the result of exercise training and ER on neutrophil function	Blood biomarkers: Subpopulations of neutrophils: CD16, CD11b White blood cells: leukocytes, lymphocytes, neutrophils PA and neutrophil oxidative burst activity measured by flow cytometry	Leukocytes, neutrophils, and lymphocytes not affected by WR No effect of ER on oxidative burst activity

Table 5 continued

Study	Sex	Participants (*n*)	Duration	Study purpose	Methods	Outcomes
Case–control studies						
Yaegaki et al. (2007) [93]	F	16 judo 8 WR 8 controls	20-day pre-competition period	Changes in capability of ROS production by neutrophils following WR	Blood biomarkers: Blood leukocytes: neutrophils, serum immunoglobulins, complement, myogenic enzymes PA, SOA, and ROS production capability measured by flow cytometry	↑ ROS production in both groups ↓ PA in WR group ↑ SOA in controls
Suzuki et al. (2003) [92]	F	16 judo 8 WR 8 controls	Before and immediately after match and 8 days later	Effects of short-term WR on neutrophil functions	Blood biomarkers: White blood cells: total leukocyte, neutrophil, lymphocyte counts PA and neutrophil oxidative burst activity measured by flow cytometry	↓ PA per cell in WR group ↑ Rate of neutrophils producing ROS/ oxidative burst activity per cell in both groups
Imai et al. (2002) [90]	M	18 amateur wrestlers 9 WR 9 no WR	1 month intensive training	Effects of WL on immune function during intensive exercise training	Blood biomarkers: White blood cells: total leukocyte counts, leukocyte subsets.	↑ Natural killer cells and T cells in both groups ↓ Anti-CD3 Ab-stimulated proliferation and interferon-γ production of lymphocytes in WR group

Ab antibody, *approx*. approximately, *BW* body weight, *CD* type of white blood cell, *CR* calorie restriction, *ER* energy restriction, *ET* exercise training, *F* female, *FRSA* free radical scavenging activity, *IL* interleukin, *M* male, *PA* phagocytic activity, *ROS* reactive oxygen species, *RWC* rapid weight changes, *RWL* rapid weight loss, *sIgA* salivary immunoglobulin A, *SJFT* Special Judo Fitness Test, *SOA* serum opsonic activity, *TLR* Toll-like receptor, *TNF* tumor necrosis factor, *URTI* upper respiratory tract infection, *WL* weight loss, *WR* weight reduction, ↑ increase, ↓ decrease

attained in a 6-month intervention [110]. A continuous, controlled dietary intervention, potentially greater than 6 months, may be necessary to allow for favorable menstrual changes. This supports previous research that showed non-pharmacological treatment (a sports nutrition beverage providing an additional 360 kcal/day), in combination with a reduced amount of exercise training, can contribute to re-establishing the hormonal profile necessary for resumption of menses [112].

Despite the lack of conclusive evidence, partially due to the small sample size and variation in the type of interventions used, there appears to be sufficient support for implementation of individualized dietary interventions, in conjunction with appropriate exercise training. Such interventions should increase awareness of the nutritional practices necessary to meet energy needs [117]. Further opportunities to improve athletic health and performance, such as screening for symptoms associated with LEA, may also be beneficial for the athletic population. Two research groups have shown that screening active females for symptoms of LEA effectively identified those at increased risk who would benefit from diet and exercise interventions [39, 118]. The benefits of regular screening among female athletes needs further exploration as does the development of screening tools that can be used with male athletes.

10 Conclusion

This review highlights the impact of LEA on a range of physiological functions that can potentially negatively affect athlete health and sports performance. Athletes need to be screened and educated individually by an appropriate healthcare professional about EA and potential health consequences associated with LEA. A recurrent theme in the literature is the lack of standardized methods for assessing EA in athletes. Small sample size in research studies is compounded by 'exercise' and athlete groups (e.g., performance level) being poorly defined, creating difficulty and confusion when making comparisons

Table 6 Intervention studies to improve health issues associated with low energy availability

Study	Sex	Mean age (years)	Participants (*n*)	Study length (months)	Change in mean EA (kcal/kg FFM/day)	Outcome measures	Comments
Dietary interventions							
Lagowska et al. (2014) [62]	F	18.1	31 professional athletes with menstrual dysfunction	3	Baseline: 28 3 months: 36	Dietary intake and body composition Serum concentrations: LH, FSH, 17-estradiol, and progesterone	↑ EI, EA, LH and LH:FSH ratio No resumption of menses Positive correlation between EA and LH
Cialdella-Kam et al. (2014) [110]	F	EU: 23.1 ExMD: 22.6	17 endurance trained 9 EU 8 ExMD	6	EU baseline: 38 ExMD baseline: 37 ExMD 6 months: 45	VO_{2max} Fasting bloods: iron, vitamin B_{12}, folate, vitamin D Reproductive hormones: estradiol, LH, FSH, prolactin, progesterone Bone health: BMD, bone mineral content, bone markers Muscle strength and power POMS	↑ EI, EA, and energy balance (N/S) ExMD resumed menses ExMD for >8 months took longer to resume menses/lower spine and hip BMD Improvements in spinal BMD in 2 ExED athletes Although N/S, POMS fatigue, and depression scores were 15% lower and 8% higher in ExMD vs. EU
Guebels et al. (2014) [111]	F	EU: 24.6 ExMD: 22.6	17 endurance trained 9 EU 8 ExMD	6	ExMD EA when EEE adjusted at 0 and 6 months using 4 methods: *Month* *0* *6* Method 1: 34 43 Method 2: 28 39 Method 3: 34 44 Method 4: 37 45	RMR EA assessed using 4 different methods to quantify EEE	↑ weight with + 360 kcal/day for 6 months No change in energy balance, EA, or RMR Assessment of EA varied (~30%) by method used
Diet, training, and nutritional counselling interventions							
Garthe et al. (2013) [109]	M F	NCG: 19.1 ALG: 19.6	39 athletes from mixed sports	Nutritional guidance given during a 2- to 3-month weight-gain period	N/A	BW, body composition 1RM, 40 m sprint, counter-movement jump	↑ EI higher in NCG vs. ALG (3585 ± 601 vs. 2964 ± 884 kcal/day) ↑ BW in NCG vs. ALG FFM similar in both groups ↑ 1RM in both groups (6–12%) ↓ 40 m sprint in NCG

Table 6 continued

Study	Sex	Mean age (years)	Participants (*n*)	Study length (months)	Change in mean EA (kcal/kg FFM/day)	Outcome measures	Comments
Garthe et al. (2011) [116]	M F	NCG:18.5 ALG:19.6	Athletes from mixed sports: 31 completed intervention 21 completed follow-up	Nutritional guidance for 2- to 3-month weight-gain period	N/A	BW, body composition	EI in NCG normalized after 12 months EI in ALG unchanged ↑ BM more in the NCG vs. ALG ↑ FFM in NCG, unchanged in ALG NCG maintained ↑ BM and FFM after intervention period
Diet and exercise training interventions							
Kopp-Woodroffe et al. (1999) [117]	F	N/A	4 AM athletes	5	N/A	Vitamin B_{12}, folate, zinc, magnesium, protein-bound calcium, iron status parameters Thyroid hormones: T_3 and T_4	↑ EI and energy balance ↑ Micronutrient intakes of vitamin B_{12}, folate, zinc, iron, and ferritin
Dueck et al. (1996) [112]	F	19	4 endurance trained 3 EU 1 AM	15 weeks	N/A	Body composition, BMD, estradiol, progesterone, LH, FSH, cortisol	AM athlete: ↑ Energy balance: baseline (−155) vs. week 4 (+683) ↑ Body fat by 6%; ↓ fasting LH and cortisol EU athletes: Minimal loss BF ↓ Follicular phase LH No change in cortisol
Nutrition education interventions							
Day et al. (2015) [115]	F	19.5	25 division 1 track and field runners	6 interactive sessions of nutrition education	Baseline: 31 After intervention: N/A	Body composition, nutrition knowledge, DE risk, menstrual history, stress fracture history	40% participants AM; 32% had history of ≥1 stress fracture ↑ Nutrition knowledge post-nutrition education program; $p = 0.001$ No increase in EI
Molina-Lopez et al. (2013) [113]	M	22.9	14 handball players	4	Week 0: 34 Week 8: 39 Week 16: 39	Blood glucose, transferrin, albumin, pre-albumin, creatinine, HDL, LDL, TG, TC, iron, nutrition knowledge	Post nutritional intervention: ↑ In total EI at weeks 8 and 16 vs. week 0; $p \leq 0.01$

Table 6 continued

Study	Sex	Mean age (years)	Participants (*n*)	Study length (months)	Change in mean EA (kcal/kg FFM/day)	Outcome measures	Comments
Valliant et al. (2012) [114]	F	19.5	11 volleyball players	4	Baseline: 24 Post intervention: 29	Body composition, nutrition knowledge	Post-dietary intervention: ↑ EI, carbohydrate, protein ↑ Sports nutrition knowledge

1RM one repetition maximum, *ALG* ad libitum group, *AM* amenorrheic, *BF* body fat, *BM* body mass, *BMD* bone mineral density, *BW* body weight, *DE* disordered eating, *EA* energy availability, *EEE* exercise energy expenditure, *EI* energy intake, *EU* eumenorrheic, *ExMD* exercise-induced menstrual dysfunction, *F* female, *FFM* fat-free mass, *FSH* follicle-stimulating hormone, *HDL* high-density lipoprotein cholesterol, *LDL* low-density lipoprotein cholesterol, *LH* luteinizing hormone, *M* male, *N/A* not available, *N/S* not statistically significant, *NCG* nutritional counselling group, *POMS* Profile of Mood States, *RMR* resting metabolic rate, T_3 triiodothyronine, T_4 thyroxine, *TC* total cholesterol, *TG* triglycerides, VO_{2max} maximum oxygen capacity, ↑ increase, ↓ decrease

between studies and impairing clear demonstration of the prevalence and consequences of the problem. Furthermore, consideration of study design is vital as much of the current research provides low-quality evidence. Nonetheless, an association between LEA and unfavorable health and sports performance outcomes is apparent. A standardized method for measuring EA is a priority. The lack of information on injury, illness, and CVD risk factors in a state of relative energy deficiency, and on effective diet and exercise interventions for use within this group, implies the need for further research to ensure that athletes achieve optimal health and sports performance.

Compliance with Ethical Standards

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Conflict of interest Danielle Logue, Sharon Madigan, Eamonn Delahunt, Mirjam Heinen, Sarah-Jane McDonnell, and Clare Corish declare that they have no conflicts of interest relevant to the content of this review.

References

1. Loucks AB, Kiens B, Wright HH. Energy availability in athletes. J Sports Sci. 2011;29(Suppl 1):S7–15.
2. Melin A, Tornberg AB, Skouby S, et al. Energy availability and the female athlete triad in elite endurance athletes. Scand J Med Sci Sports. 2015;25(5):610–22.
3. Nattiv A, Loucks AB, Manore MM, et al. American College of Sports Medicine position stand. The female athlete triad. Med Sci Sports Exerc. 2007;39(10):1867–82.
4. De Souza MJ, Nattiv A, Joy E, et al. 2014 Female Athlete Triad Coalition consensus statement on treatment and return to play of the female athlete triad: 1st International Conference held in San Francisco, CA, May 2012, and 2nd International Conference held in Indianapolis, IN, May 2013. Clin J Sport Med. 2014;24(2):96–119.
5. Otis CL, Drinkwater B, Johnson M, et al. American College of Sports Medicine position stand. The Female Athlete Triad. Med Sci Sports Exerc. 1997;29(5):i–ix.
6. Mountjoy M, Sundgot-Borgen J, Burke L, et al. The IOC consensus statement: beyond the Female Athlete Triad-Relative Energy Deficiency in Sport (RED-S). Br J Sports Med. 2014;48(7):491–7.
7. Tenforde AS, Barrack MT, Nattiv A, et al. Parallels with the female athlete triad in male athletes. Sports Med. 2016;46(2):171–82.
8. Slater J, Brown R, McLay-Cooke R, et al. Low energy availability in exercising women: historical perspectives and future directions. Sports Med. 2017;47(2):207–20.
9. Mountjoy M, Sundgot-Borgen J, Burke L, et al. Authors' 2015 additions to the IOC consensus statement: Relative Energy Deficiency in Sport (RED-S). Br J Sports Med. 2015;49(7):417–20.
10. Harbour R, Miller J. A new system for grading recommendations in evidence based guidelines. BMJ. 2001;323(7308):334–6.
11. Loucks AB, Verdun M, Heath EM. Low energy availability, not stress of exercise, alters LH pulsatility in exercising women. J Appl Physiol. 1998;84(1):37–46.
12. Loucks AB, Thuma JR. Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. J Clin Endocrinol Metab. 2003;88(1):297–311.
13. Loucks AB. Physical health of the female athlete: observations, effects, and causes of reproductive disorders. Can J Appl Physiol. 2001;26(Suppl):S176–85.
14. Ihle R, Loucks AB. Dose-response relationships between energy availability and bone turnover in young exercising women. J Bone Miner Res. 2004;19(8):1231–40.
15. Bullen BA, Skrinar GS, Beitins IZ, et al. Induction of menstrual disorders by strenuous exercise in untrained women. N Engl J Med. 1985;312(21):1349–53.
16. Williams NI, Helmreich DL, Parfitt DB, et al. Evidence for a causal role of low energy availability in the induction of menstrual cycle disturbances during strenuous exercise training. J Clin Endocrinol Metab. 2001;86(11):5184–93.
17. Williams NI, Leidy HJ, Hill BR, et al. Magnitude of daily energy deficit predicts frequency but not severity of menstrual disturbances associated with exercise and caloric restriction. Am J Physiol Endocrinol Metab. 2015;308(1):E29–39.

18. Reed JL, De Souza MJ, Williams NI. Changes in energy availability across the season in Division I female soccer players. J Sports Sci. 2013;31(3):314–24.
19. Viner RT, Harris M, Berning JR, et al. Energy availability and dietary patterns of adult male and female competitive cyclists with lower than expected bone mineral density. Int J Sport Nutr Exerc Metab. 2015;25(6):594–602.
20. Koehler K, Achtzehn S, Braun H, et al. Comparison of self-reported energy availability and metabolic hormones to assess adequacy of dietary energy intake in young elite athletes. Appl Physiol Nutr Metab. 2013;38(7):725–33.
21. Gibbs JC, Williams NI, De Souza MJ. Prevalence of individual and combined components of the female athlete triad. Med Sci Sports Exerc. 2013;45(5):985–96.
22. Woodruff SJ, Meloche RD. Energy availability of female varsity volleyball players. Int J Sport Nutr Exerc Metab. 2013;23(1):24–30.
23. Vanheest JL, Rodgers CD, Mahoney CE, et al. Ovarian suppression impairs sport performance in junior elite female swimmers. Med Sci Sports Exerc. 2014;46(1):156–66.
24. Muia EN, Wright HH, Onywera VO, et al. Adolescent elite Kenyan runners are at risk for energy deficiency, menstrual dysfunction and disordered eating. J Sports Sci. 2016;34(7):598–606.
25. Silva MR, Paiva T. Low energy availability and low body fat of female gymnasts before an international competition. Eur J Sport Sci. 2015;15(7):591–9.
26. Dolan E, O'Connor H, McGoldrick A, et al. Nutritional, lifestyle, and weight control practices of professional jockeys. J Sports Sci. 2011;29(8):791–9.
27. Doyle-Lucas AF, Akers JD, Davy BM. Energetic efficiency, menstrual irregularity, and bone mineral density in elite professional female ballet dancers. J Dance Med Sci. 2010;14(4):146–54.
28. Hoch AZ, Pajewski NM, Moraski L, et al. Prevalence of the female athlete triad in high school athletes and sedentary students. Clin J Sport Med. 2009;19(5):421–8.
29. Thong FS, McLean C, Graham TE. Plasma leptin in female athletes: relationship with body fat, reproductive, nutritional, and endocrine factors. J Appl Physiol. 2000;88(6):2037–44.
30. De Souza MJ, Miller BE, Loucks AB, et al. High frequency of luteal phase deficiency and anovulation in recreational women runners: blunted elevation in follicle-stimulating hormone observed during luteal-follicular transition. J Clin Endocrinol Metab. 1998;83(12):4220–32.
31. Magkos F, Yannakoulia M. Methodology of dietary assessment in athletes: concepts and pitfalls. Curr Opin Clin Nutr Metab Care. 2003;6(5):539–49.
32. Reed JL, De Souza MJ, Kindler JM, et al. Nutritional practices associated with low energy availability in Division I female soccer players. J Sports Sci. 2014;32(16):1499–509.
33. Burke L, Deakin V. Clinical sports nutrition. North Ryde: McGraw-Hill Education; 2015.
34. Schaal K, Tiollier E, Le Meur Y, et al. Elite synchronized swimmers display decreased energy availability during intensified training. Scand J Med Sci Sports. 2016;1(27):925–34.
35. Koehler K, Hoerner NR, Gibbs JC, et al. Low energy availability in exercising men is associated with reduced leptin and insulin but not with changes in other metabolic hormones. J Sports Sci. 2016;34(20):1921–9.
36. Hagmar M, Berglund B, Brismar K, et al. Body composition and endocrine profile of male Olympic athletes striving for leanness. Clin J Sport Med. 2013;23(3):197–201.
37. Melin A, Tornberg AB, Skouby S, et al. The LEAF questionnaire: a screening tool for the identification of female athletes at risk for the female athlete triad. Br J Sports Med. 2014;48(7):540–5.
38. McNulty KY, Adams CH, Anderson JM, et al. Development and validation of a screening tool to identify eating disorders in female athletes. J Am Diet Assoc. 2001;101(8):886–92 **(quiz 93–4)**.
39. Folscher LL, Grant CC, Fletcher L, et al. Ultra-marathon athletes at risk for the female athlete triad. Sports Med Open. 2015;1(1):29.
40. De Souza MJ, Hontscharuk R, Olmsted M, et al. Drive for thinness score is a proxy indicator of energy deficiency in exercising women. Appetite. 2007;48(3):359–67.
41. Byrne S, McLean N. Elite athletes: effects of the pressure to be thin. J Sci Med Sport. 2002;5(2):80–94.
42. Sundgot-Borgen J, Torstveit MK. Prevalence of eating disorders in elite athletes is higher than in the general population. Clin J Sport Med. 2004;14(1):25–32.
43. Sundgot-Borgen J, Torstveit MK. Aspects of disordered eating continuum in elite high-intensity sports. Scand J Med Sci Sport. 2010;20(2):112–21.
44. Goltz FR, Stenzel LM, Schneider CD. Disordered eating behaviors and body image in male athletes. Rev Bras Psiquiatr. 2013;35(3):237–42.
45. Torstveit MK, Rosenvinge JH, Sundgot-Borgen J. Prevalence of eating disorders and the predictive power of risk models in female elite athletes: a controlled study. Scand J Med Sci Sports. 2008;18(1):108–18.
46. Gibbs JC, Williams NI, Mallinson RJ, et al. Effect of high dietary restraint on energy availability and menstrual status. Med Sci Sports Exerc. 2013;45(9):1790–7.
47. Schaal K, Van Loan MD, Casazza GA. Reduced catecholamine response to exercise in amenorrheic athletes. Med Sci Sports Exerc. 2011;43(1):34–43.
48. Martinsen M, Holme I, Pensgaard AM, et al. The development of the brief eating disorder in athletes questionnaire. Med Sci Sports Exerc. 2014;46(8):1666–75.
49. Warren MP. Endocrine manifestations of eating disorders. J Clin Endocrinol Metab. 2011;96(2):333–43.
50. Hilton LK, Loucks AB. Low energy availability, not exercise stress, suppresses the diurnal rhythm of leptin in healthy young women. Am J Physiol Endocrinol Metab. 2000;278(1):E43–9.
51. Scheid JL, De Souza MJ, Leidy HJ, et al. Ghrelin but not peptide YY is related to change in body weight and energy availability. Med Sci Sports Exerc. 2011;43(11):2063–71.
52. Hill BR, De Souza MJ, Wagstaff DA, et al. The impact of weight loss on the 24-h profile of circulating peptide YY and its association with 24-h ghrelin in normal weight premenopausal women. Peptides. 2013;49:81–90.
53. Leidy HJ, Gardner JK, Frye BR, et al. Circulating ghrelin is sensitive to changes in body weight during a diet and exercise program in normal-weight young women. J Clin Endocrinol Metab. 2004;89(6):2659–64.
54. Leidy HJ, Dougherty KA, Frye BR, et al. Twenty-four-hour ghrelin is elevated after calorie restriction and exercise training in non-obese women. Obesity (Silver Spring). 2007;15(2):446–55.
55. Melin A, Tornberg AB, Skouby S, et al. Low-energy density and high fiber intake are dietary concerns in female endurance athletes. Scand J Med Sci Sports. 2016;26(9):1060–71.
56. Burke LM, Hawley JA, Wong SH, et al. Carbohydrates for training and competition. J Sports Sci. 2011;29(Suppl 1):S17–27.
57. Lagowska K, Kapczuk K. Testosterone concentrations in female athletes and ballet dancers with menstrual disorders. Eur J Sport Sci. 2016;16(4):490–7.

58. Poslusna K, Ruprich J, de Vries JH, et al. Misreporting of energy and micronutrient intake estimated by food records and 24 hour recalls, control and adjustment methods in practice. Br J Nutr. 2009;101(Suppl 2):S73–85.
59. Dwyer J, Eisenberg A, Prelack K, et al. Eating attitudes and food intakes of elite adolescent female figure skaters: a cross sectional study. J Int Soc Sports Nutr. 2012;9(1):53.
60. Martin B, Golden E, Carlson OD, et al. Caloric restriction: impact upon pituitary function and reproduction. Ageing Res Rev. 2008;7(3):209–24.
61. Hoch AZ, Papanek P, Szabo A, et al. Association between the female athlete triad and endothelial dysfunction in dancers. Clin J Sport Med. 2011;21(2):119–25.
62. Lagowska K, Kapczuk K, Friebe Z, et al. Effects of dietary intervention in young female athletes with menstrual disorders. J Int Soc Sports Nutr. 2014;11:21.
63. Hackney AC. Effects of endurance exercise on the reproductive system of men: the "exercise-hypogonadal male condition". J Endocrinol Investig. 2008;31(10):932–8.
64. Hagmar M, Berglund B, Brismar K, et al. Hyperandrogenism may explain reproductive dysfunction in olympic athletes. Med Sci Sports Exerc. 2009;41(6):1241–8.
65. Cashman KD. Diet, nutrition, and bone health. J Nutr. 2007;137(11 Suppl):2507s–12s.
66. Nana A, Slater GJ, Hopkins WG, et al. Importance of standardized DXA protocol for assessing physique changes in athletes. Int J Sport Nutr Exerc Metab. 2016;26(3):259–67.
67. Barrack MT, Rauh MJ, Barkai H-S, et al. Dietary restraint and low bone mass in female adolescent endurance runners. Am J Clin Nutr. 2008;87(1):36–43.
68. Barrack MT, Rauh MJ, Nichols JF. Prevalence of and traits associated with low BMD among female adolescent runners. Med Sci Sports Exerc. 2008;40(12):2015–21.
69. Tenforde AS, Fredericson M, Sayres LC, et al. Identifying sex-specific risk factors for low bone mineral density in adolescent runners. Am J Sports Med. 2015;43(6):1494–504.
70. Duckham RL, Peirce N, Meyer C, et al. Risk factors for stress fracture in female endurance athletes: a cross-sectional study. BMJ Open. 2012;2(6):e001920.
71. Mallinson RJ, Williams NI, Hill BR, et al. Body composition and reproductive function exert unique influences on indices of bone health in exercising women. Bone. 2013;56(1):91–100.
72. Vanderschueren D, Laurent MR, Claessens F, et al. Sex steroid actions in male bone. Endo Rev. 2014;35(6):906–60.
73. Moran JM, Martin RR, Pedrera-Canal M, et al. Low testosterone levels are associated with poor peripheral bone mineral density and quantitative bone ultrasound at phalanges and calcaneus in healthy elderly men. Biol Res Nurs. 2015;17(2):169–74.
74. Dolan E, McGoldrick A, Davenport C, et al. An altered hormonal profile and elevated rate of bone loss are associated with low bone mass in professional horse-racing jockeys. J Bone Miner Metab. 2012;30(5):534–42.
75. Pepper M, Akuthota V, McCarty EC. The pathophysiology of stress fractures. Clin Sports Med. 2006;25(1):1–16, vii.
76. Rauh MJ, Nichols JF, Barrack MT. Relationships among injury and disordered eating, menstrual dysfunction, and low bone mineral density in high school athletes: a prospective study. J Athl Train. 2010;45(3):243–52.
77. Thein-Nissenbaum JM, Rauh MJ, Carr KE, et al. Associations between disordered eating, menstrual dysfunction, and musculoskeletal injury among high school athletes. J Orthop Sports Phys Ther. 2011;41(2):60–9.
78. Thein-Nissenbaum JM, Rauh MJ, Carr KE, et al. Menstrual irregularity and musculoskeletal injury in female high school athletes. J Athl Train. 2012;47(1):74–82.
79. Tenforde AS, Sayres LC, McCurdy ML, et al. Overuse injuries in high school runners: lifetime prevalence and prevention strategies. PM R. 2011;3(2):125–31 **(quiz 31)**.
80. Beals KA, Manore MM. Disorders of the female athlete triad among collegiate athletes. Int J Sport Nutr Exerc Metab. 2002;12(3):281–93.
81. Palmer-Green D, Fuller C, Jaques R, et al. The Injury/Illness Performance Project (IIPP): a novel epidemiological approach for recording the consequences of sports injuries and illnesses. J Sports Med (Hindawi Publ Corp). 2013;2013:523974.
82. Walsh NP, Gleeson M, Pyne DB, et al. Position statement. Part two: maintaining immune health. Exerc Immunol Rev. 2011;17:64–103.
83. Gleeson M, Pyne DB. Respiratory inflammation and infections in high-performance athletes. Immunol Cell Biol. 2016;94(2):124–31.
84. Gleeson M. Immunological aspects of sport nutrition. Immunol Cell Biol. 2016;94(2):117–23.
85. Raysmith BP, Drew MK. Performance success or failure is influenced by weeks lost to injury and illness in elite Australian track and field athletes: a 5-year prospective study. J Sci Med Sport. 2016;19(10):778–83.
86. Flierl MA, Rittirsch D, Huber-Lang M, et al. Catecholamines-crafty weapons in the inflammatory arsenal of immune/inflammatory cells or opening Pandora's box? Mol Med. 2008;14(3–4):195–204.
87. Zouhal H, Jacob C, Delamarche P, et al. Catecholamines and the effects of exercise, training and gender. Sports Med. 2008;38(5):401–23.
88. Abedelmalek S, Chtourou H, Souissi N, et al. Caloric restriction effect on proinflammatory cytokines, growth hormone, and steroid hormone concentrations during exercise in judokas. Oxid Med Cell Longev. 2015;2015:809492.
89. Shimizu K, Aizawa K, Suzuki N, et al. Influences of weight loss on monocytes and T-cell subpopulations in male judo athletes. J Strength Cond Res. 2011;25(7):1943–50.
90. Imai T, Seki S, Dobashi H, et al. Effect of weight loss on T-cell receptor-mediated T-cell function in elite athletes. Med Sci Sports Exerc. 2002;34(2):245–50.
91. Kowatari K, Umeda T, Shimoyama T, et al. Exercise training and energy restriction decrease neutrophil phagocytic activity in judoists. Med Sci Sports Exerc. 2001;33(4):519–24.
92. Suzuki M, Nakaji S, Umeda T, et al. Effects of weight reduction on neutrophil phagocytic activity and oxidative burst activity in female judoists. Luminescence. 2003;18(4):214–7.
93. Yaegaki M, Umeda T, Takahashi I, et al. Change in the capability of reactive oxygen species production by neutrophils following weight reduction in female judoists. Br J Sports Med. 2007;41(5):322–7.
94. Tsai ML, Chou KM, Chang CK, et al. Changes of mucosal immunity and antioxidation activity in elite male Taiwanese taekwondo athletes associated with intensive training and rapid weight loss. Br J Sports Med. 2011;45(9):729–34.
95. Marcotte H, Lavoie MC. Oral microbial ecology and the role of salivary immunoglobulin A. Microbiol Mol Biol Rev. 1998;62(1):71–109.
96. Tsai ML, Ko MH, Chang CK, et al. Impact of intense training and rapid weight changes on salivary parameters in elite female Taekwondo athletes. Scand J Med Sci Sports. 2011;21(6):758–64.
97. Schachinger V, Britten MB, Zeiher AM. Prognostic impact of coronary vasodilator dysfunction on adverse long-term outcome of coronary heart disease. Circulation. 2000;101(16):1899–906.
98. Dusting GJ, Fennessy P, Yin ZL, et al. Nitric oxide in atherosclerosis: vascular protector or villain? Clin Exp Pharmacol Physiol Suppl. 1998;25:S34–41.

99. Zeni Hoch A, Dempsey RL, Carrera GF, et al. Is there an association between athletic amenorrhea and endothelial cell dysfunction? Med Sci Sports Exerc. 2003;35(3):377–83.
100. Rickenlund A, Eriksson MJ, Schenck-Gustafsson K, et al. Amenorrhea in female athletes is associated with endothelial dysfunction and unfavorable lipid profile. J Clin Endocrinol Metab. 2005;90(3):1354–9.
101. Misra M, Miller KK, Tsai P, et al. Uncoupling of cardiovascular risk markers in adolescent girls with anorexia nervosa. J Pediatr. 2006;149(6):763–9.
102. Ohwada R, Hotta M, Oikawa S, et al. Etiology of hypercholesterolemia in patients with anorexia nervosa. Int J Eat Disord. 2006;39(7):598–601.
103. Filaire E, Maso F, Degoutte F, et al. Food restriction, performance, psychological state and lipid values in judo athletes. Int J Sports Med. 2001;22(6):454–9.
104. Fogelholm M. Effects of bodyweight reduction on sports performance. Sports Med. 1994;18(4):249–67.
105. El Ghoch M, Soave F, Calugi S, et al. Eating disorders, physical fitness and sport performance: a systematic review. Nutrients. 2013;5(12):5140–60.
106. Lacey JM. Disordered eating among athletes: a comprehensive guide for health professionals. Middletown: American Library Association CHOICE; 2005. p. 2021.
107. Armstrong LE, VanHeest JL. The unknown mechanism of the overtraining syndrome: clues from depression and psychoneuroimmunology. Sports Med. 2002;32(3):185–209.
108. Mackinnon LT. Overtraining effects on immunity and performance in athletes. Immunol Cell Biol. 2000;78(5):502–9.
109. Garthe I, Raastad T, Refsnes PE, et al. Effect of nutritional intervention on body composition and performance in elite athletes. Eur J Sport Sci. 2013;13(3):295–303.
110. Cialdella-Kam L, Guebels CP, Maddalozzo GF, et al. Dietary intervention restored menses in female athletes with exercise-associated menstrual dysfunction with limited impact on bone and muscle health. Nutrients. 2014;6(8):3018–39.
111. Guebels CP, Kam LC, Maddalozzo GF, et al. Active women before/after an intervention designed to restore menstrual function: resting metabolic rate and comparison of four methods to quantify energy expenditure and energy availability. Int J Sport Nutr Exerc Metab. 2014;24(1):37–46.
112. Dueck CA, Matt KS, Manore MM, et al. Treatment of athletic amenorrhea with a diet and training intervention program. Int J Sport Nutr. 1996;6(1):24–40.
113. Molina-Lopez J, Molina JM, Chirosa LJ, et al. Implementation of a nutrition education program in a handball team; consequences on nutritional status. Nutr Hosp. 2013;28(4):1065–76.
114. Valliant MW, Emplaincourt HP, Wenzel RK, et al. Nutrition education by a registered dietitian improves dietary intake and nutrition knowledge of a NCAA female volleyball team. Nutrients. 2012;4(6):506–16.
115. Day J, Wengreen H, Heath E, et al. Prevalence of low energy availability in collegiate female runners and implementation of nutrition education intervention. Sports Nutr Ther. 2015;1:101.
116. Garthe I, Raastad T, Sundgot-Borgen J. Long-term effect of nutritional counselling on desired gain in body mass and lean body mass in elite athletes. Appl Physiol Nutr Metab. 2011;36(4):547–54.
117. Kopp-Woodroffe SA, Manore MM, Dueck CA, et al. Energy and nutrient status of amenorrheic athletes participating in a diet and exercise training intervention program. Int J Sport Nutr. 1999;9(1):70–88.
118. Slater J, McLay-Cooke R, Brown R, et al. Female recreational exercisers at risk for low energy availability. Int J Sport Nutr Exerc Metab. 2016;26(5):421–7.