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http://researchonline.ljmu.ac.uk/id/eprint/20280/

Article

Citation (please note it is advisable to refer to the publisher's version if you intend to cite from this work)

Melin, A, Areta, J, Heikura, I, Stellingwerff, T, Torstveit, M and Hackney, A (2023) Direct and indirect impact of low energy availability on sports performance. Scandinavian Journal of Medicine and Science in Sports. ISSN 0905-7188

Received: 23 December 2022 | Revised: 27 January 2023 | Accepted: 1 February 2023
DOI: 10.1111/sms.14327

SPECIAL ISSUE ARTICLE

WILEY

Direct and indirect impact of low energy availability on sports performance

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Abstract
Low energy availability (LEA) occurs inadvertently and purposefully in many athletes across numerous sports; and well planned, supervised periods with moderate LEA can improve body composition and power to weight ratio possibly enhancing performance in some sports. LEA however has the potential to have negative effects on a multitude of physiological and psychological systems in female and male athletes. Systems such as the endocrine, cardiovascular, metabolism, reproductive, immune, mental perception, and motivation as well as behaviors can all be impacted by severe (serious and/or prolonged or chronic) LEA. Such widely diverse effects can influence the health status, training adaptation, and performance outcomes of athletes leading to both direct changes (e.g., decreased strength and endurance) as well as indirect changes (e.g., reduced training response, increased risk of injury) in performance. To date, performance implications have not been well examined relative to LEA. Therefore, the intent of this narrative review is to characterize the effects of short-, medium-, and long-term exposure to LEA on direct and indirect sports performance outcomes. In doing so we have focused both on laboratory settings as well as descriptive athletic case-study-type experiential evidence.

KEYWORDS
illness, injuries, low carbohydrate availability, REDs

1 | INTRODUCTION

The energetic demands of training by athletes are often large within elite sports, although there can be marked variations across sport disciplines and the different phases of training during the season. Regardless of the training phase an adequate energy intake (EI) in relation to the energetics demands of training is fundamental to allow sufficient nutrient and energy availability (EA) to support optimal physiological function.[1] It can be challenging, however, for some athletes to maintain adequate EI to prevent themselves from developing a state of low energy availability (LEA). In some sports body mass (BM) and body composition or power to mass ratio affect performance,[2] and well planned, supervised periods with moderate LEA might enhance performance.[2,3] Unfortunately, many athletes undertake severe (serious and/or prolonged or chronic) LEA, which may lead to Relative Energy

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Deficiency in Sport (REDs), and thereby poses a risk to the health and performance potential of the athlete.[4] Most published literature on LEA has focused on the physiological or negative health specific aspects associated with its occurrence. To date, the effects of LEA on sports performance variables remain largely unexplored in a systematic fashion. As such, the main purpose of this narrative review is to characterize the effects of short-, medium-, and long-term exposure to LEA on direct (e.g. reduced strength and endurance) and indirect (e.g. reduced training response or lost training time due to injury) sports performance outcomes. In doing so we have focused both on laboratory settings as well as descriptive athletic case-study-type experiential evidence.

2 | LOW ENERGY AVAILABILITY

Operationally EA is defined as the difference in EI and exercise energy expenditure (EEE) relative to fat-free mass (FFM).[5] At present studies remain inconclusive as to what is the optimal EA for athletes by sex, yet current evidence from short-term laboratory-based studies suggests that an EA of 45 kcal/kg FFM/day for sedentary females[5] and 40 kcal/kg FFM/day for recreationally active males[6] appears to be congruent with normal physiological function. Unfortunately, evidence on what constitutes appropriate EA levels in female or male elite athletes (who can have variable clinical risk factors for REDs) is not firmly established. Furthermore, lack of agreement on what constitutes EEE makes EA calculations and interpretation of such calculations challenging.

2.1 | Low energy availability criteria

Early laboratory-based studies by Professor Anne Loucks on recreationally active eumenorrheic women reported that 3–6 days of EA $\leq$30 kcal/kg FFM/day causes clear endocrine and metabolic responses that mirror those of females with functional hypothalamic amenorrhea (FHA).[7,8] In much of the LEA research literature, this value ($\leq$30 kcal/kg FFM/day) is viewed as the threshold of LEA in athletes.[3,9,10] More recent studies have questioned this single cut-point for females suggesting that adverse consequences such as ovarian suppression and FHA are more likely to occur with decreasing EA but without a single universally applicable LEA criteria threshold value.[11–13] In males, the cut-point for LEA existence has been debated in the literature, and relative to REDs development a recent study have suggested values of 9 to 25 kcal/kg FFM as a criterion cut-point.[14] However, the validity and accuracy of this range of values remain to be determined, and the 40 kcal/kg FFM/day noted early[15] for adequate physiological function is hotly debated. It is important to recognize that LEA threshold values seems to be impacted by individual moderating factors (such as gynecological age, sex, type of sport, macronutrient profile of the energy deficit and genetics),[16,17] and it is also possible that the severity of LEA and its duration interact to induce a 'LEA dose or load' (i.e. LEA level multiplied by the number of days with LEA).[18,19] Therefore, a threshold based on studies of short duration exposure may be misleading, and as such, it has been suggested that an EA range might be more appropriate to use in both females[3] and males.[6,14,20,21]

Low energy availability can be produced by an intentional or inadvertent mismatch between inadequate EI to adequately compensate for an athlete's EEE. Intentional LEA is common among athletes who strive to achieve or maintain a lower competitive BM over a prolonged competitive season[3] or use it to achieve a rapid BM reduction in weight-category sports.[22,23] However, disordered eating (DE) behavior, as well as eating disorders (EDs), underpin some LEA situations, especially among athletes in leanness-demanding sports such as endurance, aesthetic, or weight category disciplines[24–27] (see *Potential neutral or positive effects of low energy availability on performance*). Nevertheless, it is important to keep in mind that athletes can develop LEA unknowingly, often due to the high or extreme EEE's of training (e.g., rowing, triathlon, cycling[18,28]) in combination with the inability of appetite to match daily energy requirements, and potential alimentary limits to the caloric absorption via the digestive system.[29]

3 | LOW ENERGY AVAILABILITY AND SPORTS PERFORMANCE

Sports performance is incredibly complex, but success in some sports is reliant, at least to some extent, on the physique and body composition of the athlete. Accordingly, it is not uncommon for an athlete to feel the pressure to alter their body composition (e.g., reduce fat mass) via manipulations of nutrition and exercise to decrease EA in the pursuit of further enhancing performance.[2] Health consequences from severe LEA are well established in terms of endocrine and metabolic disruptions[8,30] as well as psychological (e.g., mood disturbances and symptoms of DE behavior)[31,32] and clinical (e.g., premature osteoporosis) problems.[4,10,33] As such, DE behavior and EDs poses a potential risk to the health and performance potential of the athlete.[24] Therefore, matching EI to support training, recovery, as well as health and ultimately performance goals, is crucial to all athletes.[3,34]

Evidence suggests the emerging responses to short-, medium-, and long-term LEA centre around: reduced glucose concentrations, skeletal muscle glycogen, and protein synthesis, reduced circulating reproductive and anabolic hormones, disruption in markers of iron and bone metabolism, increased risk of mood disturbances and injuries all of which have potential direct or indirect performance implications.[19,30,35–37] Thus, the following sections describe key aspects of direct and indirect effects or associations between some of these parameters and sports performance. Specific details on representative performance-based research studies addressing short-, medium-, and long-term LEA consequences are shown in Tables 1, 2, and 3.

3.1 | Potential negative effects of low energy availability on performance

3.1.1 | Macronutrient issues

Decreased EI (absolute or relative to EEE) comes with a concomitant reduction in the availability of some or all energy substrates [i.e., carbohydrates (CHO), fat, or protein]. Indeed, one of the most significant indirect effects of LEA on sports performance may be via reduced CHO availability, given its key role as a fuel substrate during high-intensity exercise. Research, starting in the early 1960s, has highlighted the importance of sufficient daily CHO intake for high-intensity performance, and to improve prolonged endurance events.[38–40] This effect is most likely due to the contributions of CHO to endogenous (skeletal muscle and liver glycogen) and exogenous (blood glucose) fuel stores as the main source of energy during high-intensity exercise.[41,42] Not surprisingly, laboratory-based studies implementing LEA have reported concomitant reductions in skeletal muscle glycogen concentrations among the athletes.[43] Accordingly, at a restricted CHO intake, manipulation of EA via fat intake has shown to have no effect on skeletal muscle glycogen.[44] Therefore, a direct significant effect of LEA on performance may not necessarily be related to specific endocrine and physiological responses triggered by LEA alone, but due to the lack of CHO as substrate for metabolic function, particularly at high intensity.

Skeletal muscle glycogen potentially has a dual role, in that ample glycogen acts as an energy substrate while low muscle glycogen can act as an endurance phenotypic signaling agent. Thus, the negative effects of low muscle glycogen on performance may result in a contrasting effect of enhanced endurance adaptations to training due to stimulating skeletal muscle towards an oxidative phenotype.[45] However, while the evidence for these types of molecular adaptations is generally strong, a recent meta-analysis indicates that the risk for negative consequences of periodic low CHO availability (LCA) training might outweigh the rewards of such strategies.[46] LEA and LCA may also be a confounding factor in training-overload interventions, where the athlete's inability to adapt to training or increased fatigue levels may be mistaken as signs of overreaching or overtraining, rather than under-fuelling.[47] Therefore, when investigating the effect of LEA (acute or prolonged) and/or training overload on performance, a possible explanation for decreases in performance may be low muscle glycogen, if the experimental design does not include a strict dietary control performance pre-test scenario. Furthermore, short-term LCA (with and without LEA) has been reported to increase bone resorption biomarkers,[37,48] decrease markers of bone formation,[37] and increased post-exercise interleukin-6 (IL-6) and hepcidin concentrations[49] (see *Micronutrient issues* section) with negative effects on bone, immune- and iron biomarkers.[37,48–51] Hence, LCA may have indirect negative effects on performance (see *Injury and illness factors* and *Micronutrient issues*).

3.1.2 | Potential direct effects of low energy availability on performance decrements

Short-term low energy availability and performance decrements

Short-term LEA is defined as an exposure of a few days to weeks of inadequate EA.[18] Table 1 presents the findings of nine studies that addressed this form of LEA in association with direct and indirect performance outcomes. Most of these studies with a 3 days to 3 weeks duration of LEA report neutral or positive effects on sports performance variables[23,52–55] (see *Potential neutral or positive effects of low energy availability on sports performance*). However, in a randomized 14-day LEA intervention trial by Jurov et al.[14] well-trained and elite male endurance athletes ($n = 18$) had direct (counter movement jumps, agility test, power output, bicycle ergometer test to exhaustion), and indirect [lactate metabolism, and health outcomes (well-being, cognitive restriction, and eating behavior)] performance variables assessed while their EA was reduced by 25%, 50%, and 75% of prior EI[14] (Table 1). A minimum of a one-month wash-out period between EA treatments occurred for resting energy expenditure, body composition, blood values, and questionnaire responses to return to baseline values. No change in the bicycle ergometer test to exhaustion after the 14-days of 75% LEA (9 ± 3 kcal/kg FFM/day) was found, but it was reported that lactate levels were decreased after 14-days of 25% LEA (22 ± 6 kcal/kg FFM/day).[14] The latter was probably a consequence of decreased CHO (and muscle glycogen) availability.

TABLE 1 Short-term LEA (days to weeks) and performance aspects.

Reference	Athletic group	Study design	Energy availability	Weight-loss	Direct or indirect performance effects of LEA	Biomarkers of LEA
Jurov et al. 2022[14]	Male endurance trained athletes ($n = 18$), Tier 3	*Cross-sectional controlled study* 3 x 2 wks of varying levels of LEA with 1-mo washout in between each intervention	EA −25% (↑ EEE from ~1200 to ~2600 kcal/d; EA 22 ± 6 kcal/kg FFM/d), EA −50% (EA 17 ± 5 kcal/kg FFM/d) EA −75% (EA 9 ± 3 kcal/kg FFM/d)	75% LEA: ↓ BM	*Direct effects* 25% LEA: ↓ CMJ, ↔ agility T-test. 50% LEA: ↓ power output, CMJ, ↔ agility T-test. 75% LEA: ↓ power output, CMJ, ↔ agility T-test. *Indirect effects* 25% LEA: ↓ LA metabolism, well-being 50% LEA: ↓ LA metabolism, well-being 75% LEA: ↓ LA metabolism, well-being	25% LEA: ↓ Hb, ↑ cognitive restriction. 50% LEA: ↑ eating behavior score. 75% LEA: ↓ T_3, ↑ cognitive restriction and eating behavior score
Jurov et al. 2021[32]	Male endurance trained athletes ($n = 12$), Tier 3	*Cross-sectional controlled study* 2 wks of LEA	EA −50% (17 ± 5 kcal/kg FFM/d) via ↑ EEE	→ BM, ↓ FM	*Direct effects* ↓ CMJ, power output, relative power output, ↔ agility T-test. *Indirect effects* ↓ anaerobic threshold (↑ LA)	↔ RMR, Hb (↓ RF $n = 2$), T_3, testosterone (↓ RF $n = 1$), insulin, IGF-1, cortisol (↑ RF $n = 1$)
Kettunen et al. 2021[56]	Female cross-country skiers ($n = 19$), Tier 2–3	*Prospective observational study* 5 d of intensive training camp with calculation of EA based on 48-h food and training logs	LEA ($n = 7$) Subclinical LEA ($n = 4$) Optimal EA ($n = 8$)	NA	*Direct effects* ↓ explosive power (CMJ) Lower EA and CHO intake associated with signs of overreaching and ↓ muscular performance as demonstrated by RJ and CMJ. *Indirect effects* ↓ HR/RPE and lactate/RPE. ↑ RPE. EA correlated with changes in LA ($r = 0.54$, $p = 0.02$), LA/RPE ($r = 0.65$, $p < 0.01$), and RJ ($r = 0.47$, $p = 0.04$)	↓ HR, Hb, leptin, T_3, and insulin. LEAF-Q-score ≥ 8 ($n = 5$). FHA ($n = 3$)

TABLE 1 (Continued)

Reference	Athletic group	Study design	Energy availability	Weight-loss	Direct or indirect performance effects of LEA	Biomarkers of LEA
Kojima et al. 2020[55]	Male distance-runners ($n = 7$), Tier 3	*Randomized crossover study* 3 d of LEA and 3 d normal EA with 2 wks washout	LEA (19 ± 2 kcal/kg FFM/d). Normal EA (53 ± 5 kcal/kg FFM/d)	2%	*Direct effects* ↔ endurance capacity (time to exhaustion). *Indirect effects* ↔ LA	↓ 30% muscle glycogen, testosterone, IGF-1
Mettler et al. 2010[52]	Male resistance trained athletes ($n = 20$), Tier 1	*Randomized parallel study* 7 d of habitual diet followed by 7 d of WL diets with high and low protein intakes	EA not reported. EI −40% of habitual intake. Protein 2.3 g/kg or 1.0 g/kg (high and low protein group, respectively)	High protein: −1.5 kg Low protein: −3.0 kg	*Direct effects* ↔ squat jump, maximal isometric leg extension, (1RM) bench press, muscle endurance bench press, and 30-s Wingate test (both groups)	↔ glucose, NEFA, glycerol, urea, cortisol, fT, IGF-1, GH
Mourier et al. 1997[53]	Male elite wrestlers ($n = 25$), Tier 3	*Randomized parallel study design* 19 d intervention	Control ($n = 6$): 40 kcal/kg/d (12 E% protein). Hypocaloric control ($n = 6$): 28 kcal/kg/d (12 E% protein). Hypocaloric high-protein ($n = 7$): 28 kcal/kg/d (25 E% proteins). Hypocaloric high BCAA ($n = 6$): 28 kcal/kg/d (20 E% protein). Hypocaloric low protein ($n = 6$): 28 kcal/kg/d (15 E% protein). 19 d (28 kcal/kg/d): control ($n = 6$), ↑ protein ($n = 7$), ↑ BCAA, $n = 6$), ↓ protein ($n = 6$) and EB control ($n = 6$)	Range −2.5% to −5.4%. Highest WL (−4 kg, 5.4 %), with BCAA	*Direct effects* ↔ aerobic (VO_{2max}), PPO, anaerobic capacities (Wingate test) and muscular strength	

(Continues)

TABLE 1 (Continued)

Reference	Athletic group	Study design	Energy availability	Weight-loss	Direct or indirect performance effects of LEA	Biomarkers of LEA
Fogelholm et al. 1993[23]	Male combat sport athletes ($n = 10$), Tier 3–4	*Longitudinal intervention* A 3 wk gradual WL and 2.4 d rapid WL with 2 mo washout in between	EA not reported. Gradual WL: −1000 kcal/d Rapid WL: Severe dietary and fluid restriction	5%–6%	*Direct effects* (*rapid WL*) ↔ sprint (30-m run) and anaerobic (1-min Wingate test) performance, vertical jump height. *Direct effects* (*gradual WL*) ↑ vertical jump height 6%–8% ↔ sprint (30-m run) ↑ anaerobic (1-min Wingate test) performance	*N*
Wallberg et al. 1988[54]	Male weight lifters ($n = 19$), Tier 1–2	*Parallel study design* 7 d intervention	EA not reported. Control ($n = 5$; 35 kcal/kg/d and 1.1 g/kg/d protein) Moderate protein, high CHO (MPHC) ($n = 7$; 18 kcal/kg/d and 0.8 g/kg/d protein). High protein, moderate CHO (HPMC) ($n = 7$; 18 kcal/kg/d and 1.6 g/kg/d protein)	Control: −0.9 ± 0.2 kg HPMC: −3.6 ± 0.5 kg (−4.6%) MPHC: −4.0 ± 0.2 kg (−4.9%)	*Direct effects* Macronutrient mix: ↔ biceps endurance. HPMC: ↓ quadriceps endurance	

Note: Tier, sporting level; 0, Sedentary; 1, Recreationally active; 2, Trained; 3, National level/highly trained; 4, International level/Elite; 5, World class.[119]; ↓, reduced, ↔, unchanged, ↑, improved.

Abbreviations: Agility T-test, (measures the speed of movement, change of direction, strength and stability of lower extremities); BCAA, branched amino acids; BM, body mass; CMJ, counter movement jump (assesses explosive power of lower extremities); CSA, cross-sectional area; d, days; EA, energy availability; EB, energy balance; EEE, exercise energy expenditure; EI, energy intake; FFM, fat free mass; FHA, functional hypothalamic oligomenorrhea/amenorrhea; FM, fat mass; FSH, follicle stimulating hormone; fT, free testosterone; GH, growth hormone; Hb, hemoglobin; HR, heart rate; HRV, heart rate variability; IGF-1, insulin like growth factor 1; LA, lactate; LEA, low energy availability; LH, luteinizing hormone; mo, months; MPS, myofibrillar protein synthesis; NA, not assessed; NEFA, non-esterified fatty acids; PPO, peak power output; RCT, randomized controlled trial; RF, reference range; RJ, reactive jump test (assesses explosive power of lower extremities); RMR, resting metabolic rate; RPE, rated perceived exhaustion; RPE, rated perceived exhaustion; SHBG, sexual hormone binding protein; T_3, triiodothyronine; T_4, thyroxine; wk(s), week(s); WL, weight loss; yr, years.

TABLE 2 Medium-term LEA (weeks to months) and performance aspects.

Reference	Athletic group	Study design	Energy availability	Weight-loss	Direct or indirect performance effects of LEA	Biomarkers of LEA
Hammer et al. 2022[84]	Male elite wrestlers ($n = 67$), Tier 3	*Prospective longitudinal* (7 yr). WL before competition calculated by [(mid-season BM – pre-season BM)/ pre-season BM]	EA not reported	−7.0% ± 3.2% Injured athletes vs. −5.7% ± −3.3% in non-injured athletes	*Indirect effects* For every 1% of BM lost, wrestlers had an 11% increased hazard of injury (HR 1.11, 95% CI 1.03 to 1.19, $p = 0.005$)	
Stenqvist et al. 2020[57]	Male well-trained male cyclists ($n = 22$), Tier 2	*Longitudinal intervention* 4 wks of high intensity training for 32 min, 3 times a wk, superimposed on the athletes' background training	EA not reported	No change	*Direct effects* Aerobic performance-peak power output +4.8%. VO_{2peak} + 2.4%. Functional threshold power + 6.5%. A subgroup analysis of the $n = 5$ with the largest ↑ fT:cor ratio revealed a greater improvement in functional threshold power (9.5 vs. 2.5%), and higher relative RMR (0.6 vs. −4.2%) suggesting positive relationship between training adaptation and EA.	RMR (absolute −3.0%; relative RMR −2.6%; RMR_{ratio} − 3.3%) tT +8.1% fT +4.1% T_3–4.8% Cortisol +12.9% No change in the T:cortisol ratios.
Schaal et al. 2021[58]	Female competitive distance runners ($n = 16$), Tier 2	*Longitudinal observation* 4 wks of 30% increase in training volume with ad libitum EI	FOR runners ($n = 9$) maintained baseline EA during training overload. Failure to increase EI resulted in LEA in the NFOR group ($n = 7$)	1% WL in well adapted runners associated to ↑ RMR	*Direct effects* LEA subjects experienced NFOR characterized by absence of running performance supercompensation after a 2-wk recovery period	LEA: ↓ body temperature, leptin, oestradiol, luteal phase length, ↑ age of menarche

(Continues)

TABLE 2 (Continued)

Reference	Athletic group	Study design	Energy availability	Weight-loss	Direct or indirect performance effects of LEA	Biomarkers of LEA
Langan-Evans et al. 2021[67]	Male combat sport athlete ($n = 1$), Tier 4 or 5	*Longitudinal observation (case study)* 8 wks of WL	7 wks: EA 20 kcal/kg FFM/d 1 wk: EA <10 kcal/kg FFM/d	14%	*Direct effects* ↑ Absolute maximal dynamic strength 6%–9%, absolute and relative VO_{2peak} +13% and +19% respectively	After 7 wks → biomarkers. After 1 wk of severe LEA: ↓ RMR_{ratio}, testosterone, ↓↓ LH, FSH, SHBP, ↑↑ total cholesterol. All biomarkers reversed within 48 h (↑ EI) except for total cholesterol and fasting hyperinsulinemia suggesting insulin resistance
Schoenfeld et al. 2020[68]	Male body builder ($n = 1$), Tier 2 or 3	*Longitudinal observation (case study)* 8 mo competition preparation incl. WL + 4 mo post-contest recovery	EA not reported. EI 3000–1500 kcal/d during preparation	10%	*Direct effects* Explosive strength ↓ during pre-contest preparation that recovered quickly post-competition	Alterations in metabolism, hormonal status, and psychological aspects of eating observed during pre-contest preparation. All variables recovered quickly post competition
Tinsley et al. 2019[64]	Female physique athlete ($n = 1$), Tier 2 or 3	*Longitudinal observation (case study)* 6 mo competition preparation incl. WL + 2 mo recovery. High-volume resistance training throughout the 8 mo assessment period, with ↑ aerobic training during the 2 pre-competition phases	EA not reported. ↓ EI (25–16 kcal/kg/d, ↑ protein, ↓ CHO)	9%	*Direct effects* Concentric and eccentric peak forces ↓ 19% before the first competition after 4 mo and throughout the competition period and persisted 2 mo after the final competition	↓ RMR with partial recovery 5 d post the 1st competition. ↓↓RMR in the inter-competition period with total recovery 1 month after the final competition

TABLE 2 (Continued)

Reference	Athletic group	Study design	Energy availability	Weight-loss	Direct or indirect performance effects of LEA	Biomarkers of LEA
Kasper et al. 2019[69]	Male mixed martial arts athlete ($n = 1$) (rofessional MMA athlete), Tier 5	*Longitudinal observation* (*case study*) 7 wks of WL	EA not reported but likely low to extremely low (EI 1000–1900 kcal/d + 5 d of water loading +20 h fasting and dehydration + 32 h rehydration and refueling	18%	*Direct effects* Inability to complete performance tests suggests significant performance impairment	↑ cortisol, ↓ RMR, IGF-1, LH, FHS, ↓↓ testosterone and hypercholesterolemia. Severe dehydration induced hypernatremia and acute kidney injury. All variables except total cholesterol recovered post competition
Woods et al. 2017[59]	Male ($n = 5$) and female ($n = 5$) national team rowers, Tier 3–4	*Longitudinal observation* 4 wks of ~21% increase in training volume with ad libitum EI	EI unchanged with increased EEE resulted in decreased EA. A separate analysis estimated EA of 12 kcal/kg FFM/d	2%	*Direct effects* ↓ on-water 5 km rowing performance, pacing strategy. *Indirect effects* ↑ fatigue, ↑ total mood disturbance	↓ RMR
Woods et al. 2018[60]	Male trained cyclists ($n = 13$), Tier 3–4.	*Longitudinal observation* 1 wk of 20% increase in training load, 2 wks (+50%) + 2 recovery wks (~80% of baseline training load)	EA not reported but estimated to be significantly reduced due to unaltered EI in the presence of increased EEE	Yes	*Direct effects* ↓ anaerobic and aerobic performance after 3 wks of increased training load. *Indirect effects* ↑ HRV after 3 wks of increased training load. ↑ total mood disturbance. All variables returned to baseline after the recovery period	↓ RMR. → leptin, T_3 after 3 wks of increased training load.

(Continues)

TABLE 2 (Continued)

Reference	Athletic group	Study design	Energy availability	Weight-loss	Direct or indirect performance effects of LEA	Biomarkers of LEA
Stellingwerff 2018[70]	Female elite endurance athletes ($n = 1$), Tier 5	*Longitudinal observation* (*case study*) 9 yr regular assessment of skinfold thickness, BM, injury, illness, and performance indices	EA not reported. Qualitative description of implementation of 6–8 wks pre-race BM reduction (WL) with likely decreases in EA (unclear whether true LEA) + 8 mo general preparation phase with BM stability and optimal EA	2%–4%	*Direct effects* ↑ long-term training adaptation when training at a heavier BM during the general preparation phase, followed by body composition optimisation preceding the competition period. *Indirect effects* No significant injuries outside return to sport postpartum	EUM, normal BMD.
Hulmi et al. 2017[61]	Female fitness athletes dieting for competition ($n = 27$) and controls ($n = 23$), Tier 2 or 3	*Longitudinal observation* 4-mo competition preparation incl. WL with dietary recording mostly throughout the 4-mo time period	EA not reported. EI during the diet 22.9 ± 13.8% lower than at baseline and at ~30 kcal/kg BM combined with average daily activity MET of 9, was likely LEA	12%	*Direct effects* ↔ Isometric maximal strength and explosive strength of leg extensors ↓ isometric bench press *Indirect effects* ↔ mood	↓ Leptin, T_3, T, and oestradiol. ↑ Menstrual irregularities. BW and hormones except T_3 and testosterone reversed during a 3–4-month recovery period (↑ EI and ↓ aerobic exercise)
Pardue et al. 2017[71]	Male body builder ($n = 1$), Tier 2 or 3	*Longitudinal observation* (*case study*) 8 mo competition preparation incl. WL + 5 mo recovery with monthly assessments of nutrition and performance	EA not reported ↓ EI (3860 to 1724 kcal/d) likely resulted in LEA	10%	*Direct effects* ↓ anaerobic power (Wingate test) by end of competition preparation, no restoration with recovery	↓ RMR, testosterone, T_3, T_4, ↑ cortisol, and ghrelin. After 5 mo recovery all biomarkers except T_3, T_4, were reversed

TABLE 2 (Continued)

Reference	Athletic group	Study design	Energy availability	Weight-loss	Direct or indirect performance effects of LEA	Biomarkers of LEA
Robinson et al. 2015[72]	Male body builder ($n = 1$), Tier 2	*Longitudinal observation (case study)* 14 wks competition preparation incl. WL with dietary recording at baseline and wk 3, 8, 10 and 13	EA not reported. ↓ EI (−880 ± 430 kcal/d, ↑ protein and fat, ↓ CHO). EB from −500 up to nearly −1500 kcal/d	14%	*Direct effects* ↓ absolute $\dot{V}O_{2max}$ 19% and relative VO_{2max} 6%. ↓ hamstring concentric peak torque 22%. ↑ hamstring eccentric peak torque 27%. ↑ relative quadriceps concentric and eccentric peak torque. *Indirect effects* ↔ BRUMS scores throughout the diet period	↓ RMR
Rossow et al. 2013[63]	Male body builder ($n = 1$), Tier 2 or 3	*Longitudinal observation (case study)* 6 mo competition preparation incl. WL + 6 mo recovery with monthly assessments of nutrition and performance.	EA not reported ↓ EI (~2500 kcal/d during PREP; ~3500 kcal/d during REC) likely resulted in LEA during PREP	14%	*Direct effects* ↓ strength without full recovery during 6 mo of recovery. *Indirect effects* ↑ total mood disturbance	↓ HR, BP, testosterone. All variables returned to baseline during the 6-mo recovery period
Garthe et al. 2011[62]	Male and female elite athletes ($n = 24$), varied sports, Tier 3	*Longitudinal intervention* Slow WL vs fast WL for 4 to 12 wks (until target weight was achieved)	EA not reported. Slow WL: EI ↓ 19 ± 2% Fast WL: EI ↓ 30% ± 4%	6%	*Direct effects* Slow WL: ↑ CMJ, ↔ 1RM squat and 40-m-sprint. Fast WL: ↔ CMJ, 1RM squat and 40-m-sprint	A history of dieting and weight cycling was reported by 53% of the athletes in the slow-WL and 45% of the athletes in fast-WL group

(Continues)

TABLE 2 (Continued)

Reference	Athletic group	Study design	Energy availability	Weight-loss	Direct or indirect performance effects of LEA	Biomarkers of LEA
Koutedakis et al. 1994[65]	Female elite lightweight rowers ($n = 6$), Tier 4.	*Longitudinal observation* 2 mo vs. 4 mo habitual WL periods with 1 yr washout	EA not reported. Self-administered diets during WL ranging from ~2000 kcal/d to ~1000 kcal/d with approx. 60% and 20% energy from CHO and protein, respectively	6.0% (2 mo) 7.4% (4 mo)	*Direct effects* After 2 mo WL: ↓ isokinetic knee flexor peak torque. After 4 mo WL: ↑ VO_{2max} and peak power. *Indirect effects* After 2 mo: ↓ respiratory anaerobic threshold	
Ingjer & Sundgot-Borgen 1990[66]	Female elite endurance athletes with symptoms of EDs ($n = 33$), Tier 3–4	*Case–control study* 5 yr observation. 2 mo WL ($n = 7$) using pathogenic WL-methods and 1 yr follow up	EA not reported. +20% endurance training during a 4- to 6-week period before the first part of the preparation period (15–20 h/wk) during the WL period, and the same period of time the following year	9%	*Direct effects* Controls: ↑ Running speed (+3.6%) and VO_{2max} (+4.7%). WL: ↓$VO_{2\,max}$ (−9.4%), ↔running speed. Returned to baseline after 1 yr of likely EB	4 of the athletes in the WL group had clinical EDs

Note: Tier, sporting level; 0, Sedentary; 1, Recreationally active; 2, Trained; 3, National level/highly trained; 4, International level/Elite; 5, World class.[119]; ↓, reduced, ↔, unchanged, ↑, simproved.

Abbreviations: BM, body mass; BRUMS, The Brunel Mood Scale; CMJ, counter movement jump (assesses explosive power of lower extremities); d, days; EA, energy availability; EB, energy balance; EDs, eating disorders; EEE, exercise energy expenditure; EI, energy intake; EUM, eumenorrheic; FFM, fat free mass; FHA, functional hypothalamic oligomenorrhea/amenorrhea; FM, fat mass; FOR, functional overreaching; FSH, follicle stimulating hormone; fT, free testosterone; Hb, hemoglobin; HR, heart rate; HRV, heart rate variability; IGF-1, insulin like growth factor 1; LA, lactate; LEA, low energy availability; LH, luteinizing hormone; mo, months; MPS, myofibrillar protein synthesis; NA, not assessed; NFOR, non-functional overreaching; RCT, randomized controlled trial; RF, reference range; RPE, rated perceived exhaustion; RJ, reactive jump test (assesses explosive power of lower extremities); RMR, resting metabolic rate; RPE, rated perceived exhaustion; SHBG, sexual hormone binding protein; T, testosterone; T_3, triiodothyronine; T_4, thyroxine; tT, total testosterone; yr, years; WL, weight loss; wk(s), week(s).

TABLE 3 Long-term LEA (months to years) and performance aspects.

Reference	Athletic group	Study design	Manifestation of LEA	Direct or indirect performance effects of LEA	Biomarkers of LEA
Gillbanks et al. 2022[108]	Female ($n = 8$) and male ($n = 4$) light weight rowers with at least one REDs symptoms, Tier 2–4	*Qualitative cross-sectional assessment* Audio-recorded semi-structured individual telephone interviews	**Self-report** Recurrent injuries including stress fractures, menstrual dysfunction, low energy levels, prioritized leanness, excessive fatigue, muscle loss, inability to recover between sessions, diagnosis of REDs or the Female Athlete Triad	*Direct effects* Self-reported decreased performance and recovery, fatigue, and injury. *Indirect effects* Self-reported disrupted sleep, bowel disruption, menstrual dysfunction, musculoskeletal pain, and weakened immune system	
Langbein et al. 2021[112]	Female ($n = 10$) and male ($n = 2$) endurance athletes, Tier 2–3	*Qualitative cross-sectional assessment* Semi-structured interviews designed to explore contexts and mechanisms underpinning the onset of REDs; the subjective experience of REDs; contexts and mechanisms influencing “recovery” from REDs	**Self-report** Past or current experiences of REDs, associated with periods of LEA	*Direct effects* A multitude of physiological impairments predominantly consisted of BSI, a noticeable reduction in energy levels, and perceived reduced endurance capacity. *Indirect effects* Significant psychological distress	
Ihalainen et al. 2021[85]	Female elite middle and distance runners ($n = 13$), Tier 3, and EUM controls ($n = 8$), Tier 1	*Longitudinal observation* Assessments 4 times during 1 yr: Sept. post competition, Nov. post general preparation, May post specific preparation, Sept. post competition season	**Self-report** FHA ($n = 5$) EUM ($n = 8$)	*Direct effects* ↑ season best IAAF score in the EUM. ↔ season best IAAF score for FHA	FHA: ↑ risk of injuries
Heikura et al. 2018[81]	Female ($n = 27$) and male ($n = 21$) elite endurance athletes, Tier 3–5	*Cross-sectional assessment*	**Self-report** FHA ($n = 7$) EUM ($n = 20$)	*Indirect effects* ↓ baseline Hb mass in FHA suggests impaired hematological adaptation to training	↓ relative Hb mass pre-altitude exposure in FHA vs. EUM athletes

(Continues)

TABLE 3 (Continued)

Reference	Athletic group	Study design	Manifestation of LEA	Direct or indirect performance effects of LEA	Biomarkers of LEA
Ackerman et al. 2018[120]	Female athletes ($n = 1000$), Tier 1 or 2	*Cross-sectional assessment*	**Self-report** Positive response to BEDA-Q, ESP, or self-report history or current ED resulted in 47.3% with LEA	*Direct effects* ↓ training response, endurance performance. *Indirect effects* ↓ coordination, concentration, judgment, ↑ irritability	LEA: ↑ risk of FHA, poor bone health, metabolic issues, hematological detriments, psychological disorders, cardiovascular impairment, and GI dysfunction
Keay et al. 2018[75]	Competitive male road cyclists ($n = 50$), Tier 3	*Cross-sectional assessment*	**Clinical interview** Sport - specific Questionnaire and Clinical Interview (SEAQ)-I used to classify LEA (28%). 20% chronic LEA: ED/DE ($n = 5$) and long-term restrictive eating behavior ($n = 5$)	*Direct effects* ↓ cycling performance	Chronic LEA: ↓ testosterone levels
Tornberg et al. 2017[74]	Female endurance athletes ($n = 30$), Tier 3–4	*Cross-sectional assessment*	**Measured (biomarkers, ultrasound)** FHA (no bleeding for ≥3 mo) ($n = 14$) EUM ($n = 16$)	*Direct effects* ↓ (11%) Isokinetic and concentric extension and flexion strength (↔ when adjusted to leg FFM). ↓ 20% endurance in FHA	FHA: ↓ estradiol, T_3, glucose, RMR and ↑ cortisol
Vanheest et al. 2014[73]	Female elite junior swimmers ($n = 10$), Tier 3	*Longitudinal observation* 12 wk training period	**Measured (biomarkers)** Ovarian suppression (OVS; S-progesterone < 5 ng/mL) wk 0 and 2, + absence of cyclical increases in estradiol and progesterone ($n = 5$) EUM ($n = 5$)	*Direct effects* 400-m time trial swimming velocity. EUM↑ vs OVS↓	OVS: ↓ estradiol, progesterone, IGF-1 and RMR wk 0 and 2. ↓ T_3 wk 6 to 12, and IGF-1 wk 4–12
Harber et al. 1998[76]	Female competitive endurance athletes ($n = 19$) and EUM controls ($n = 13$), Tier 2	*Cross-sectional repeat assessments* Repeated assessments over a 4 mo period	**Self-report** FHA (no bleeding for ≥6 mo), ($n = 8$) EUM (≥9 menstrual cycles per yr) ($n = 11$)	*Indirect effects* ↓ CrP recovery rate in FHA. ↓ thyroid hormone concentrations in FHA may indirectly impair mitochondrial adaptations	FHA: ↓ estradiol, progesterone, T_3 and T_4

Note: Tier, sporting level; 0, Sedentary; 1, Recreationally active; 2, Trained, 3, National level/highly trained; 4, International level/Elite; 5, World class.[119] ↓, reduced; ↔, unchanged; ↑, improved.

Abbreviations: BEDA-Q, Brief Eating Disorder in Athletes Questionnaire; BM, body mass; BSI, bone stress injury; CrP, creatine phosphate; d, days; EA, energy availability; EB, energy balance; ED, eating disorder; EEE, exercise energy expenditure; EI, energy intake; ESP, eating disorder screen for primary care; EUM: eumenorrhea; FFM, fat free mass; FHA, functional hypothalamic oligomenorrhea/amenorrhea; FM, fat mass; FSH, follicle stimulating hormone; Hb, hemoglobin; HR, heart rate; HRV, heart rate variability; IAAF=International Association of Athletics Federations; IGF-1, insulin like growth factor 1; LA, lactate; LEA, low energy availability; mo, months; NA, not assessed; RMR, resting metabolic rate; T_3, triiodothyronine; T_4, thyroxine; wk(s), week(s); yr, years.

Interestingly, however, muscular power-related outcomes were reduced starting at 25% LEA only.[14] Reduced power has also been reported in female endurance athletes after 5 days of an intensive training camp where most athletes did not achieve adequate EI or CHO intake.[56]

Medium-term low energy availability and performance decrements

Medium-term LEA is defined as an exposure of a few weeks to months of inadequate EA.[18] Table 2 presents the findings of 17 studies that addressed this form of LEA in association with direct and indirect performance outcomes. Four studies have aimed to investigate the effects of training adaptation and performance after 3 - 4 weeks with a 20% - 50% increase in training volume with ad libitum EI[57–60] (see *Low energy availability or overtraining syndrome* section). Thirteen of the medium-term LEA studies have aimed to investigate the effects of various weight-loss programs on performance[61–72] of which eight are case studies.[63,64,67–72] Most of these weight-loss studies, report neutral or positive effects of LEA on sports performance variables (see *Potential neutral or positive effects of low energy availability on sports performance*). However, five of the eight case-studies report negative effects on performance variables (e.g., reduced strength and power)[63,64,68,69,71] (Table 2). Furthermore, in a study from the 1990 s, female endurance athletes ($n = 7$) with DE behavior involving pathogenic (e.g., vomiting, fasting) weight-loss methods were compared to weight-stable controls ($n = 26$). Each group was pre- and post-assessed a 2-month intensified endurance training period (+20% training amount).[66] The weight-reduction group lost 9% BM and failed to improve running speed or aerobic capacity, while the control athletes showed increases in both running speed and aerobic capacity. These findings support a failure of the weight-reduction group to respond to the training despite similar training as compared to the controls.[66]

Long-term low energy availability and performance decrements

Long-term LEA is defined as an exposure of inadequate EA during a few months to years.[18] Investigating the effects of such long periods of LEA in a controlled setting is near impossible and as such, most of the studies in this category rely on physiological or functional outcomes of LEA (e.g., DE behavior) or REDs symptoms (e.g., FHA or reduced resting metabolic rate (RMR)) as surrogate measures of likely prolonged exposure to LEA. Table 3 presents the findings of nine such studies that addressed this form of LEA in association with direct and indirect performance outcomes.

One of the few prospective studies that link long-term LEA to direct performance measures was conducted by Vanheest and colleagues.[73] They examined performance among junior elite swimmers during a 12-week competition season comparing the endocrine and performance effects in a group with suppressed oestradiol and progesterone (ovarian suppressed, $n = 5$) versus cyclic ($n = 5$) athletes across the season (Table 3). The results showed that the ovarian suppressed group had a lower concentration of T_3 and IGF-1 compared with the cyclic group, while the cyclic group had higher EI, EA, and $\mathrm{RMR}_{\mathrm{ratio}}$ (i.e., measured/predicted RMR) throughout the study period and experienced reduced fat mass compared with the ovarian suppressed group. Interestingly, 400-m swimming performance test performed fortnightly showed no difference within each group between the start and the end of the intervention. Importantly, however the ovarian-suppressed group reported a ~10% decrease in 400 m swimming performance after 12 weeks of training compared to an ~8% improvement in the cyclic group.[73]

In a cohort comparison study of female endurance athletes, no difference in $\mathrm{VO}_{2\mathrm{max}}$ (absolute or relative) was found between FHA and eumenorrheic athletes, despite a lower BM and fat mass in athletes with FHA[74] (Table 3). Furthermore, athletes with FHA had 20% reduced neuromuscular endurance compared with eumenorrheic athletes.[74] Reduced endurance performance (functional threshold power, W/kg) was also reported in four out of 10 male competitive male road cyclists with long-term LEA manifested as DE behavior or EDs.[75]

3.1.3 | Potential indirect effects of low energy availability on performance decrements

Exercise recovery

Harber et al.[7676] (1998) found that female athletes with FHA had slower restoration of creatine phosphate (also, associated with lower T_3 levels) compared with eumenorrheic athletes suggesting that recovery of skeletal muscle is reduced in FHA athletes, potentially affecting their ability to execute quality high-intensity exercise (Table 3).

Micronutrient issues

A poor iron status evolves from normal to subnormal or depleted iron stores with compromised synthesis of iron-containing proteins, such as hemoglobin (Hb) due to a low dietary iron intake, inadequate intestinal iron absorption and/or increased iron losses. This poor iron status can affect aerobic capacity in athletes through reduced tissue oxidative capacity and reduced oxygen-carrying capacity.[77] Iron absorption is impaired during the post-exercise period when hepcidin levels are elevated, and muscle glycogen availability, exercise intensity, and exercise-induced inflammatory stimulus determine the magnitude of the post-exercise hepcidin response, and therefore iron regulation.[78] LEA may also influence hepcidin concentration directly or indirectly via LCA, low estrogen or testosterone levels, and or IL-6 induced alterations in hepcidin levels post-exercise, highlighting the importance of maintaining adequate energy and CHO availability to avoid unnecessary elevations in hepcidin concentrations.[79]

Acutely lowered Hb levels have been reported after short-term LEA in male,[14,32] and female endurance athletes,[56] and is in general associated with a reduction of VO_{2max} and endurance performance due to reduction of the O_2-carrying capacity of blood.[80] Lower Hb levels have also been reported in female endurance athletes with severe LEA as manifested by the presence of FHA.[81] Altitude exposure typically invokes increases in Hb mass[80,82]; interestingly, Heikura et al.[81] reported no differences in the increases in Hb mass after a 3-week exposure to altitude (~2100 M) between females with and without FHA (LEA surrogate marker) (i.e. the altitude exposure did not induce a compensation for the LEA effects). However, females with FHA had significantly lower (~7%) baseline Hb mass upon arrival to the altitude, camp, and the subsequent altitude exposure did not induce a compensation for the LEA effects.[81]

Low energy availability or overtraining syndrome

Kettunen et al.[56] evaluated the associations between self-reported EA and macronutrient intake and sports performance in young female cross-country skiers during a 5-day intensive training camp and found that more than half of the skiers had suboptimal EA and CHO intake. Lower EA and CHO intake were associated with early signs of REDs, similar to the signs and symptoms of overreaching, including decreased muscular performance and submaximal lactate levels (rate of perceived exertion ratio)[56] (Table 1). A few studies have investigated the effects of 3–4 weeks of intensified training with ad libitum EI on performance. For example, Schaal et al.[58] compared the changes in ad libitum EI and EA among runners completing a 4-week training overload phase. The runners that adapted positively to the overload training phase increased their EI and thereby maintained baseline EA despite a large EEE, while runners not adapting to the training overload phase failed to maintain baseline EA, resulting in poor performance outcomes, and suppressed ovarian function (Table 2). These results support the findings of Woods et al.[59] who reported reduced rowing performance (on-water 5 km time trial) in male and female national team rowers during a 4-week intensified training program (+20% training volume) accompanied by weight-loss due to LEA. In a similar study with LEA (EA <40 kcal/kg FFM/day) and weight-loss among male cyclists completing a 3-week training overload phase, cycling performance did not improve after a 2-week recovery period.[60] In a 4-week intensified endurance training period designed to increase performance in well-trained male cyclists, BM, and body composition were unchanged, and aerobic performance, as well as total testosterone levels, increased from pre- to post-test although some biomarkers of REDs, such as a reduction in RMR and T_3, and an increase in cortisol were observed.[57] Similar findings were reported in a case report in a weigh-stable male elite track cyclist with lowered RMR and mid-range testosterone levels, although no negative effects on performance were found.[68] Indeed, a recent review looking at the signs, symptoms, and diagnostic complexities of non-functional overreaching training (NFOR) or the overtraining syndrome (OTS) as compared to REDs has definitely established that when LEA is present, it is the underlying etiology for a REDs diagnosis, and LEA therefore need to be excluded from an OTS/NFOR diagnosis.[47]

Injury and illness factors

Uninterrupted training and competition are crucial for training adaptation and enhanced performance. One of the concerns with severe LEA is an increased risk of injuries and illness,[4] and that performance may be impaired due to the loss of training.[83] To this end, a prospective study investigating international track and field athletes during five consecutive competitive seasons found that every week containing one or more days of modified training due to injuries and illnesses resulted in a 26% reduction in the odds of achieving key performance goals, and athletes who sustained <2 injuries or illnesses per season were three times more likely to achieve their performance goal than those who sustained ≥2 episodes of injuries or illness.[36] Therefore, it is imperative that sports medicine practitioners should direct their attention to the prevention of both injuries and illness via a multidisciplinary approach including, but not limited to, minimizing frequency of LEA in athletes.

In a recent study of 67 male U.S. Division I collegiate wrestlers, Hammer et al.[84] found that rapid weight cutting was associated with a higher risk of in-competition injuries. For every kilogram of BM lost, there was a 14% increased risk of injury during competition (hazard risk (HR) 1.14, 95% CI 1.04 to 1.25, $p = 0.004$), and for every 1% of BM lost, wrestlers had an 11% increased risk of injury (HR 1.11, 95% CI 1.03 to 1.19, $p = 0.005$)[84] (Table 1).

In a 1-year prospective study by Ihalainen et al.[85] EA, training load, and menstrual status were examined in regard to injuries and performance in young elite endurance athletes and controls (Table 3). The results revealed that runners with FHA had more injury days and less annual running distance compared with the eumenorrheic runners, and only the eumenorrheic runners improved their performance over the year.[85] This study confirms the results from other research[86,87] that FHA is a potential predisposing factor for musculoskeletal overload injuries. Other reported predisposing factors are restricted EI,[88,89] DE behavior/EDs,[88] and low BMD.[90]

Bone stress injuries (BSI), often referred to as stress fractures or stress reactions, are overuse injuries and may be related to low bone mineral density (BMD) and LEA.[91,92] Recovery from BSI may be further complicated by the presence of the Female Athlete Triad risk factors.[93] Using the Triad cumulative risk assessment tool, moderate or high-risk category collegiate runners had 4.0- and 5.7-fold risk for sustaining a BSI compared to low-risk category athletes, and a majority of elevated risk category running athletes sustained a BSI within an average of 1 year.[94] A BMD Z-score < -1.0 has been proposed as a cut-point for low BMD for both female and male athletes participating in land-based sports.[95,96] Risk factors for low BMD identified in athletes include LEA (low EI and/or prolonged distance running), low BMI and FFM, FHA/lowered testosterone levels, and a history of BSI.[91,96–98] Furthermore, a negative effect of short-term LCA on markers of bone formation and resorption independent of EA has also been reported.[48,50] Low BMD has been reported in adolescent female runners with a reported prevalence of nearly 40%,[97] and a prevalence of 43%–45% have been reported in female distance athletes.[99,100] In a study by Heikura et al. on EA status, blood hormone concentrations, BMD, and BSI history in a group of 59 elite female and male middle- and long-distance athletes, females with FHA and males with testosterone within the lowest quartile of the laboratory reference range had a ~4.5-fold higher frequency of career BSI compared to athletes with eumenorrhea/normal testosterone levels.[101]

There is limited evidence that LEA and/or LCA suppress immunity in athletes.[102] However, as mentioned earlier, negative effects of short-term LCA on immune response independent of LEA has been reported.[49,51] Furthermore, more upper respiratory symptoms and lower immunoglobulin-A secretion rates were found in FHA vs. eumenorrheic elite collegiate runners.[103] Similarly, in a study of female elite athletes in preparation for the 2016 Rio Olympic Games, an indication of LEA was the leading variable associated with illnesses.[83] There is a critical need for researchers to pursue this topic more fully before definitive conclusions can be reached on the relationship between LEA and the immune system.

Psychological disturbance

Measures of mood state are considered a reliable predictor of sports performance in competitive athletes across a wide variety of sports and athletic performance outcomes,[104] and while reduced vigor and increased fatigue are normal responses to hard training, other aspects of psychological disturbance, especially symptoms of depressed mood may indicate a maladaptive response.[105] Short-, medium-, and long-term LEA have all been reported to be associated with major mood disturbances such as anger, confusion, cognitive restriction, and tension in athletes,[58,67,106,107] sleep disturbances[71,108] and on long-term depressive symptoms,[109,110] as well as EDs.[111]

The athletes' subjective experiences

Elite athletes are part of a high-pressure environment that may exacerbate the psychological impact involving food choices and body image, affecting the amount of energy available for training and competition. This in turn may create short-term, medium-term, or long-term LEA that could affect performance. REDs is a highly complex condition, and moving beyond the laboratory-based, clinical studies, involving also quantitative and qualitative research where athletes may reveal different factors contributing to their experiences, may be a valuable addition towards an understanding of the impact LEA has on sports performance (Table 3). In the largest survey to date including 1000 adolescent and young adult female athletes, Ackerman et al.[109] assessed associations between LEA and performance factors. Here, athletes with LEA were more likely to report experiences with decreased training response, endurance performance, coordination, and concentration, impaired judgment, irritability, and depression. The authors concluded that LEA measured using self-report questionnaires is strongly associated with many performance consequences depicted in the REDs models[109] (Table 3). In a qualitative study, the physical and psychosocial impact of REDs, from the perspective of 12 current or former lightweight rowers at intermediate to international standards (67% females) was investigated[108] (Table 3). All athletes described restricting EI whilst increasing EEE through excessive exercise and/or weight-loss tactics to meet the body weight requirements. A range of implications by the athletes was described, such as impaired sleep, bowel disruption, FHA, fatigue, musculoskeletal pain, injury, and weakened immune systems. All participants reported that they felt that weight loss tactics and dehydration resulted in decreased performance and impaired recovery. For example, one elite international rower stated: 'It [dieting] really affected our race day performance, we could barely row four kilometres without people going dizzy… it was not healthy, and we were definitely not fuelled.'

A study by Langbein et al.[112] included 12 sub- or semi-elite male and female endurance athletes, and aimed to qualitatively explore athlete experiences of REDs (Table 3). The athletes described a multitude of health impairments, and factors contributing to reduced performance, predominantly consisting of BSI, a noticeable reduction in energy levels, and perceived endurance capacity. In terms of illness and injuries indirectly affecting performance, one athlete said: 'My immune system is shocking; I think I've had every disease or illness going', while another stated: 'We are all veterans of the MRI machine and DXA scan; I got a metatarsal stress fracture and then the tibial stress fractures started, of which I've had three'. Whilst representativeness and statistical generalizability are not key judgment criteria for these qualitative studies, their findings provide another knowledge important to incorporate when discussing the possible effects of LEA on sports performance in a bigger context.

3.2 | Potential neutral or positive effects of low energy availability on performance

It is noteworthy that despite an abundance of data demonstrating many indirect negative effects of severe LEA on performance (e.g., increased injury rates, poor training adaptation), there is still very limited research directly assessing the impact of LEA on direct performance outcomes and providing evidence for a causal, rather than correlative, linkage. Also, given the potential positive effect of LEA exposure (e.g., improving power to weight ratio), the possibility of performance benefits as a result of implementation of well-planned periods of moderate LEA in specific situations[113] should not be ignored.

There is some evidence, mainly from case-studies indicating that weight-loss with short-term (Table 1),[23,52–55] and medium-term (Table 2)[61,62,65,66] LEA report neutral or positive effects on performance variables. Hence, some elite athletes who manipulate their body composition prior to high-priority competitions[67,70,114] when they are likely to be in top form potentially could improve their physical performance.[67] A representative weight-loss study with short-term LEA was performed by Kojima et al.[55] These researchers reported no effects of LEA (<20 kcal/kg FFM/day) on endurance capacity assessed as time to exhaustion (~20 min, 19.0 ± 0.8 km/h, eliciting 90% VO_{2max}) in male long-distance runners in a 3-day randomized clinical trial study. However, considering a concomitant ~50% reduction in dietary CHO with LEA, not surprisingly, this research protocol decreased muscle glycogen content and lowered BM[55] (Table 1). A representative weight-loss study with medium-term LEA investigated female fitness athletes (n = 27) during a 4 months 12% weight-loss period for competition with lowered leptin, oestradiol and T_3 levels as well as increased menstrual and irregularities compared to weight-stable female fitness athletes (n = 23) acting as controls.[61] A decrease in isometric bench press was reported pre to mid intervention but not pre to post intervention, and no effects on isometric maximal strength and explosive strength of leg extensors. All endocrine changes and BM returned to baseline during the following 3–4-month recovery period with increased EI and reduced aerobic exercise.[61]

Short-term LEA and moderate energy restriction (80% of typical EI) in strength-trained males and females has been found to reduce muscle protein synthesis by 27% and 19%, respectively[115] (Table 1). The reduction in muscle protein synthesis was attenuated, at least acutely, by resistance training, and further increased by the ingestion of protein during the recovery from such training.[115] Indeed, supervised gradual weight-loss (0.5–1 kg/week) combined with strength training has been reported to be safe,[113] increase lean BM, and strength- and power-related performance in male and female athletes,[62] male combat-sport athletes[23] and in female elite rowers[65] (Tables 1 and 2). High or moderate volume resistance training during energy restriction appears to have similar effects of maintaining lean BM in resistance-trained men.[116] Moreover, a 40% reduction in EI over the course of 2 weeks showed retention of lean BM in resistance-trained men when following a high-protein (~2.3 g/kg/day) versus a normal protein (~1 g/kg/day) diet when training load was maintained constant.[52]

As mentioned earlier, it is difficult to disentangle the direct effects of acute reduced substrate availability (e.g., skeletal muscle glycogen) with LEA/LCA, from the physiological effect of LEA-triggered mechanisms of energy preservation (e.g., altered hormonal profile, decreased muscle protein synthesis and others) on physical performance. Therefore, from a mechanistic standpoint one can speculate that LEA could be an independent modulator of skeletal muscle metabolism. However, whether this translates to enhancing the oxidative capacity and muscle work capacity and thereby sports performance remains to be seen, as reported in the meta-analysis by Gejl and Nybo[46] on periodic low CHO (see *Macronutrient issues*).

Moderate and well-planned LEA may have a positive effect in the function of some tissues (i.e., skeletal muscle, neurons), but if sustained it may reach a point where the normal function of many other tissues and systems (e.g., bone and iron metabolism) may be disrupted and the likelihood of injury increased, therefore potentially negatively affecting performance. This dynamic interplay is complex and as such difficult to predict a final outcome. Dieting and restrictive eating is furthermore often associated with an increased risk of developing DE behavior and EDs,[113,117,118] and therefore, we are of the opinion that the importance of leanness should be de-emphasized within the sports environments, especially in young and developing athletes.[113] Hence, when considering BM and body composition changes, even if for short periods of time, careful planning with realistic goals is needed, and supervision from a sports dietitian is highly recommended.

4 | CONCLUSION

In conclusion, more research needs to be done to fully understand the effects of LEA on different physiological systems and how the interplay of these may ultimately affect physical capacity and athletic performance. Severe LEA exposure has the potential to be a serious problem leading to impaired sports performance, most likely mediated through direct/indirect health effects, hormonal alterations, and suboptimal levels of energy substrate (i.e., muscle glycogen). Therefore, athletes who desire to optimize BM and body composition (and use LEA to achieve those goals) to improve competitive performance should emphasize the use of well-planned and supervised gradual weight-loss methodologies with moderate LEA exposure to maintain health and performance. These athletes should also have baseline medical and psychological assessment to ascertain whether there is undue risk to even undertake BM or body composition changes. That said, the coach and athlete support team (e.g., physiotherapist, physician) must remain vigilant of the athletes' responses and health status to ensure the prevention of REDs.

5 | PERSPECTIVE

Severe LEA exposure in athletes increases the risk for the development of REDs and associated health consequences of both a physiological and psychological nature. Current nutritional status, as well as optimal muscle protein synthesis are important factors for maximizing training adaptations, as well as improving immune function, wound healing, and rehabilitation of musculoskeletal injuries in athletes. Hence, monitoring, detection, and treatment of athletes with exposure to severe LEA in the sports medicine setting will potentially improve athletes' health and ultimately performance, as well as their rehabilitation process from illness and injuries.

ACKNOWLEDGEMENT

The manuscript preparation was undertaken by A.K. Melin and A.C. Hackney in collaboration with all co-authors. J.L. Areta acknowledges disagreement with several of the points made in the article in relation to the risk of LEA on performance and interpretation of the literature, though acknowledges significant input in the manuscript. Nonetheless, all authors, including J.L. Areta approved of the final version of the paper.

CONFLICT OF INTEREST STATEMENT

None of the authors has any potential conflicts of interest.

DATA AVAILABILITY STATEMENT

Since this is a narrative review no dataset for sharing exists.

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How to cite this article: Melin AK, Areta JL, Heikura IA, Stellingwerff T, Torstveit MK, Hackney AC. Direct and indirect impact of low energy availability on sports performance. *Scand J Med Sci Sports*. 2023;00:1-23. doi:10.1111/sms.14327