Sports Medicine (2021) 51:2251–2280
https://doi.org/10.1007/s40279-021-01491-0

REVIEW ARTICLE

Overtraining Syndrome (OTS) and Relative Energy Deficiency in Sport (RED-S): Shared Pathways, Symptoms and Complexities

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Accepted: 26 May 2021 / Published online: 28 June 2021

Abstract
The symptom similarities between training-overload (with or without an Overtraining Syndrome (OTS) diagnosis) and Relative Energy Deficiency in Sport (RED-S) are significant, with both initiating from a hypothalamic–pituitary origin, that can be influenced by low carbohydrate (CHO) and energy availability (EA). In this narrative review we wish to showcase that many of the negative outcomes of training-overload (with, or without an OTS diagnosis) may be primarily due to misdiagnosed under-fueling, or RED-S, via low EA and/or low CHO availability. Accordingly, we undertook an analysis of training-overload/OTS type studies that have also collected and analyzed for energy intake (EI), CHO, exercise energy expenditure (EEE) and/or EA. Eighteen of the 21 studies (86%) that met our criteria showed indications of an EA decrease or difference between two cohorts within a given study ($n = 14$ studies) or CHO availability decrease ($n = 4$ studies) during the training-overload/OTS period, resulting in both training-overload/OTS and RED-S symptom outcomes compared to control conditions. Furthermore, we demonstrate significantly similar symptom overlaps across much of the OTS ($n = 57$ studies) and RED-S/Female Athlete Triad ($n = 88$ studies) literature. It is important to note that the prevention of under-recovery is multi-factorial, but many aspects are based around EA and CHO availability. Herein we have demonstrated that OTS and RED-S have many shared pathways, symptoms, and diagnostic complexities. Substantial attention is required to increase the knowledge and awareness of RED-S, and to enhance the diagnostic accuracy of both OTS and RED-S, to allow clinicians to more accurately exclude LEA/RED-S from OTS diagnoses.

1 Introduction

Extreme training loads are required for elite-athlete success, especially in endurance sports [1–8]. At the elite/professional level, peak training volumes can exceed 30 h/week in weight-supported endurance sports such as swimming, cycling, triathlon and rowing [1–4], resulting in substantial exercise energy expenditures (EEE). Furthermore, performance constraints of many sports also favor an ideal power (or force) to weight ratio [9–11], incentivizing body mass/composition manipulations via adjustment of energy intake (EI) [12–14]. The prolonged combination of these pursuits creates a perfect storm for potentially adverse outcomes of training-overload [with or without the diagnoses of Overtraining Syndrome (OTS)] and/or under-fueling [with a potential diagnosis of Relative Energy Deficiency in Sport (RED-S)]. Accordingly, OTS and RED-S have many shared pathways and symptoms of under-recovery.

Undeniably, acute fatigue, caused by a single or series of training sessions, is part of a normal elite training program. However, when recovery is inadequate, this acute fatigue can eventually progress and manifest into a continuum of short-term performance decrements (days to weeks) ranging from Functional Overreaching (FOR), to unplanned fatigue termed Non-Functional Overreaching (NFOR), and then over time (weeks to months of performance decrements), to OTS [6]. Meeusen et al. [6] have defined OTS as follows: "an accumulation of training and/or non-training stress resulting in long-term decrement in performance capacity". Thus, the defining factor between OTS and NFOR/FOR is the time needed for performance restoration, not the specific type of training or life stress causing the performance decrement in the first place. For this review we will use the term training-overload/OTS, unless FOR or NFOR are specified

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Key Points

Energy availability (EA) is defined as [Energy Intake (EI)—Exercise Energy Expenditure (EEE)] corrected for fat free mass, and is disturbed both by training-overload (e.g. excessive EEE) and/or inadequate EI. Accordingly the EA concept potentially provides an elegant model that can unify many of the symptoms that present with both RED-S and many training-overload/OTS situations.

Both OTS and RED-S identification are based on a diagnosis of exclusion, with both lacking a single validated universal identifier but both syndromes feature progressive deterioration of quantitative and qualitative health and performance outcomes.

Although under-recovery resulting in OTS is multifactorial, there are significant and close parallels between OTS and RED-S, and in many instances the negative outcomes of training-overload (with, or without an OTS diagnosis) may primarily be due to misdiagnosed under-fueling via low EA.

within the original paper; appreciating that prolonged OTS is much more clinically significant than FOR or NFOR. We note that, by definition, only studies that quantify a significant and sustained decline in performance outcomes can be termed FOR, NFOR or OTS studies. Therefore, the current review will differentiate between these studies and others that involve a significant increase in training load, but not necessarily OTS, which should be defined as training-overload studies.

RED-S was first introduced by the International Olympic Committee (IOC) in 2014 [15] (updated by another IOC consensus in 2018 [16]). Within the RED-S umbrella, the negative health outcomes of low energy availability (LEA) on reproductive and bone health are known as the Triad [17]. RED-S has been defined as a complex syndrome caused by LEA that results in impaired physiological function, negatively impacting aspects of health and performance. The introduction of RED-S was made in response to emerging scientific literature supporting the premise that chronic LEA can result in negative health outcomes beyond the hypothalamic–pituitary–gonadal (HPG) axis and bone health, and because male athletes were also seen to suffer health and performance consequences. The IOC RED-S consensus authors have developed spoke and wheel descriptive figures [15, 16] to demonstrate how chronic LEA potentially affects various body systems resulting in negative health and performance outcomes and to exhibit the relationship to the Triad. It is relevant to note that psychological problems, specifically disordered eating or eating disorders, can precede LEA/RED-S, and also, in some athletes, LEA itself can result in psychological sequelae [15]. The cornerstone of treatment for RED-S is the restoration of EA, generally most optimally achieved through enhancing EI via nutritional interventions, but also potentially addressing EEE through exercise modifications (for review see: [18]).

The primary etiological factor underpinning RED-S is chronically poor energy availability (EA). EA is defined as [EI–EEE] corrected by fat-free mass (FFM; Fig. 1), and can be disturbed both by training-overload (e.g. excessive EEE) and/or inadequate EI. Regardless of source of LEA, chronic LEA has been shown to result in RED-S and many related OTS symptoms (Tables 1, 2). Certainly, the EA concept potentially provides an elegant model that can unify many of the symptoms that present with both RED-S and many training-overload/OTS situations (Fig. 1). To date, however, only a handful of the training-overload/OTS publications have specifically considered assessing EA in the underlying methodology [19–22], and despite a plethora of expert RED-S/Triad [23–27] and OTS [28–31] reviews over the previous decades, we are unaware of a single extensive review which conceptually highlights the many similarities of RED-S and OTS, as well as the diagnostic challenges of each syndrome. Furthermore, it is also important to note that despite diagnostic progress as elaborated below (Section 2), there is no singular validated diagnosis method/tool for either OTS (or FOR/NFOR) or RED-S–with both syndromes having a complex overlap of symptoms wherein a diagnosis of exclusion is recommended.

The premise of this narrative review is to showcase that many of the negative outcomes of training-overload (with, or without an OTS diagnosis) may be misdiagnosed due to a failure to recognize under-recovery from under-fueling, or a frank diagnosis of RED-S. Although under-recovery is clearly multifactorial (e.g. sleep, stress), this review will demonstrate that under-recovery specifically attributed to under-fueling, resulting in low EA and poor CHO availability, is especially prevalent and often overlooked and misdiagnosed in training-overload/OTS situations. We will showcase the complexity of diagnosis of OTS and RED-S due to the significant overlapping symptom similarities between training-overload/OTS and RED-S. Furthermore, we will establish that (1) humans do not always naturally increase ad libitum EI enough to match significantly increased EEE, resulting in many instances in inadvertent and non-intentional LEA; and (2) that there is a failure in some high- performance endurance athletes to intentionally achieve compensatory EI during chronically high EEEs. We propose that an analysis of the literature shows that many classic training-overload/OTS studies may be confounded with underlying LEA (due to inadequate EI (and primarily CHO macronutrients) coupled with high training loads), as

many training-overload/OTS studies do not even assess for LEA or mention it as a confounding factor that should be a diagnosis exclusion criteria. A secondary aim is to show that the symptoms of RED-S and training-overload/OTS significantly overlap, and as such, a definitive diagnosis of training-overload/OTS or RED-S is fraught with challenges. A final aim was to create increased awareness regarding the commonalities between training-overload/OTS and RED-S amongst the athlete, coaching, medical, physiology, psychology, and nutrition communities, who tend to work independently, thus emphasizing the fact that a multi-practitioner integrated approach is required when diagnosing and treating both OTS/NFOR and RED-S.

2 OTS vs. RED-S: Symptom Overlap and Diagnosis Complexities

It is beyond the scope of the current review to systemically highlight the step by step exclusion and diagnostic assessments of both OTS and RED-S, which have been extensively articulated in previous consensus statements [6, 16, 32]. Instead, we will demonstrate below that OTS and RED-S share a significant overlap of symptoms (Table 1) and diagnoses of exclusion, resulting in considerable difficulties in accurate diagnosis of either syndrome, where elements of LEA can be especially challenging.

2.1 Diagnosis Complexities Related to Overtraining Syndrome (OTS)

Despite extensive study and review over the past 40 years [6, 29–31, 33–36], there is still no single, readily available and validated diagnostic tool to identify OTS. Like RED-S (Sect. 2.2 below), OTS features a diagnosis of exclusion and a multi-factorial etiology in which performance decline is coupled with a broad and individually variable palette of psychological/emotional, physiological, immunological, and neuroendocrine decrements (Table 1), of which exercise/training may not be the sole causative factor. As highlighted in the OTS consensus statement, "the etiology of OTS involves the exclusion of organic diseases or infections and factors such as dietary caloric restriction (negative energy balance) and insufficient carbohydrate and/or protein intake, iron deficiency, magnesium deficiency, allergies, etc., together with identification of initiating events or triggers [6]." Therefore, LEA (or RED-S) should be ruled out from an NFOR/OTS diagnosis and once all confounding factors have been excluded: strictly speaking, an NFOR/OTS diagnosis would ultimately be based on just outstanding psychogenic and lifestyle stress factors being responsible for the unexplained prolonged fatigued and decreased performance, as outlined in Fig. 1.

Nevertheless, significant OTS diagnostic progress has been made by the Meeusen research group utilizing

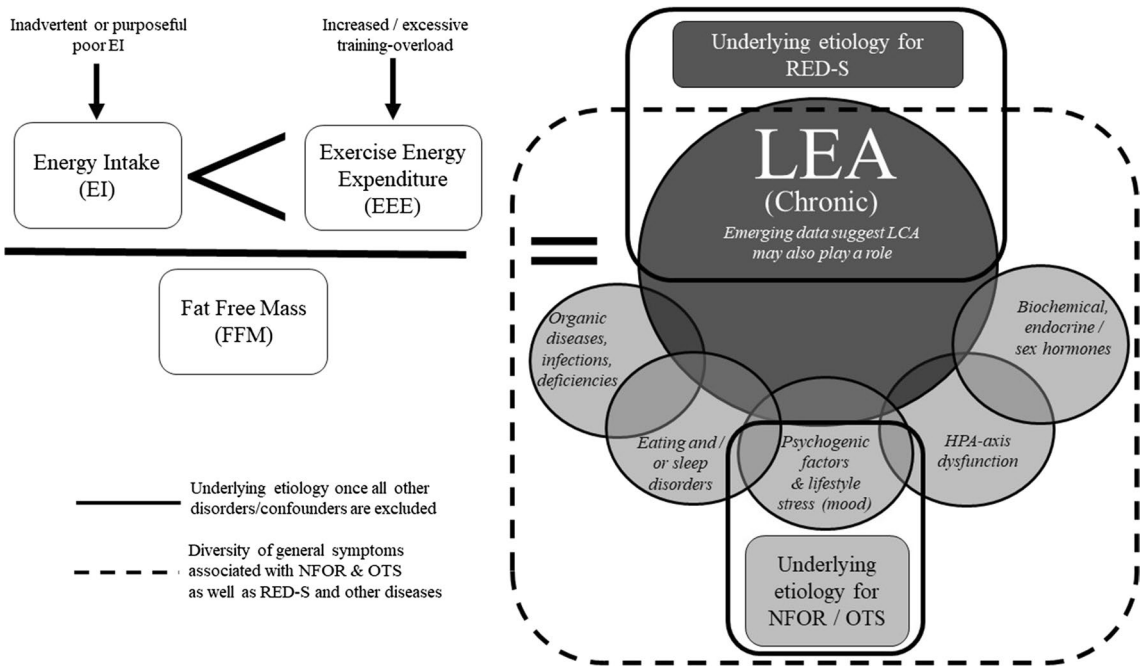

Fig. 1 Energy availability as a theoretical unifying framework for both RED-S and potentially many training-overload/OTS symptoms, while identifying the specific underlying etiology for RED-S and OTS, once all appropriate diagnostic exclusion criteria are met. EA, energy availability; EEE, exercise energy expenditure; EI, energy intake; FFM, fat free mass; FOR, functional overreaching; HPA, hypothalamic-pituitary-adrenal; LCA, low carbohydrate availability; LEA, low energy availability; NFOR, non-functional overreaching; OTS, overtraining syndrome; RED-S, relative energy deficiency in sport

Table 1 A comparison of the significant symptom/outcome similarities between primary RED-S/Triad or training-overload studies (with or without an OTS/NFOR/FOR diagnosis). Each primary paper was designated as *either* a training-overload study (with or without an OTS/NFOR/FOR diagnosis) or RED-S according to the main study topical focus. Papers with numerous outcomes have been cited across multiple performance and health outcome categories. The table is meant to showcase extensive symptom overlaps between RED-S versus training-overload studies, but not be an exhaustive collective of every study published in the respective OTS and RED-S fields

Group	Parameter	Training-overload (with or without an OTS/NFOR/FOR diagnosis) papers	RED-S/triad papers
Decreased performance outcomes	Endurance	[20–22, 78, 82, 85, 93, 96, 107, 155–164]	[63, 165, 166]
	Strength/speed/power	[21, 96, 155, 167–170]	[63, 166, 171–173]
	Injury/illness (risk or outcome)	[84, 139, 174–183]	[45, 46, 48, 65, 148, 184–203]
	Training adaptation (e.g. lactate (peak or curve), HR/HRV, training indicators etc.)	[21, 78, 80, 82, 85, 95, 107, 157, 168, 204]	[165]
	Glycogen/protein synthesis	[80, 94, 96, 156]	[205–207]
	Judgement/coordination/reaction time	[208–210]	[166, 171, 211, 212]
Health decrements	Sex hormones (e.g. estrogen, progesterone, testosterone, etc.)	[22, 159, 167, 168, 213, 214]	[43–45, 47, 51, 56–58, 63, 65, 75, 173, 192, 193, 200, 215–228]
	Other hormones (e.g. LH, ACTH, prolactin, HPA and HPG axis; cortisol; leptin;T3; IGF-1)	[19, 21, 78, 82, 84, 90, 92, 107, 156, 161, 169, 210, 213, 214, 229–231]	[45, 63, 75, 165, 172, 217, 222, 228, 232–242]
	Hematological/biochemistry (e.g. CBC, ferritin, lactate, hepcidin, CK, glutamine, other blood markers)	[84, 90, 107, 160, 162, 167, 208, 243]	[10, 45, 60, 63, 75, 148, 173, 206, 244–246]
	Immunological (including glutamine studies)	[79, 84, 90, 139, 160, 167, 174–176, 178–180, 204, 247–250]	[186, 187, 251]
	Metabolic/cardiovascular (e.g. RMR; HRV, etc.)	[19–21, 82, 91, 92, 96, 107, 158, 163, 164, 252]	[63, 102, 150, 165, 173, 192, 226, 228, 241, 242, 244, 253–255]
	Bone (e.g. BMD or bone biomarker outcomes)		[43–48, 51, 65, 150, 184, 185, 188–192, 194, 195, 197–200, 223, 225, 227, 253, 255–259]
	Psycho-social-emotional factors (e.g. DALDA, mood, fatigue, appetite, sleep, etc.)	[19–22, 80, 85, 91, 93, 95, 107, 139, 155–157, 159, 160, 162, 168, 208, 210, 230, 260]	[75, 193, 202, 203, 211, 226, 227, 242, 244, 261–265]

ACTH, adrenocorticotrophic hormone; BMD, bone mineral density; CBC, complete blood count; CK, creatine kinase; DALDA, Daily Analysis of Life Demands for Athletes questionnaire; FOR, Functional Overreaching; HPA, hypothalamic-pituitary-adrenal; HPG, hypothalamic-pituitary-gonadal; HR, heart rate; HRV, heart rate variability; IGF-1, insulin growth factor-1; LH, luteinizing hormone; NFOR, Non-Functional Overreaching; OTS, Overtraining Syndrome; RED-S, Relative Energy Deficiency in Sport; RMR, resting metabolic rate; Triad, Female Athlete Triad; T3, triiodothyronine

a two-bout maximal exercise protocol (with 4 h of rest between) in athletes with NFOR/OTS to detect attenuated changes in the typical exercise-induced increases to the hypothalamic–pituitary–adrenal (HPA) axis-related hormones (cortisol, adrenocorticotrophic hormone (ACTH), prolactin and human growth hormone [37, 38]. Building upon this, the Meeusen group has recently undertaken s tatistical discriminate analysis of 100 athletes reporting excessive fatigue and/or poor performances and found attenuated ACTH and prolactin responses in athletes with OTS to a second exercise test, coupled with a psychological test, resulted in correct diagnosis of NFOR and OTS with 98% sensitivity [39]. Additional data are needed to further validate this two-bout test across various athletic populations as well as explore its potential utility, or differential diagnosis, in RED-S situations.

It is also worth noting that some sports feature significant mechanical forces (e.g. running or contact based sports) or excessive mechanical overuse repetition (e.g. elite rowers take > 30,000–40,000 strokes/week). Therefore, diagnostic aspects of 'mechanical' over-training [5, 40] and overuse injuries can occur irrespective of an athlete's EA due to over-riding tissue strain/damage (e.g. muscle, connective tissue, bone) and biomechanical inefficiencies [40–42]. It is beyond the scope of this review to expand further on the diagnosis of mechanical over-training, but important to note the extensive body of evidence demonstrating chronic LEA can accelerate poor bone mineral density (BMD) that is certainly an important moderating factor of the mechanical training load-injury etiology [16, 17, 43–48].

2.2 Diagnosis Complexities Related to Relative Energy Deficiency in Sport (RED-S)

To aid with the screening, diagnosis and return to play, the IOC published a RED-S clinical assessment tool (RED-S CAT) to assist clinicians [32]. The RED-S CAT provides a framework for assessment of athletes by accumulated clinical parameters of risk (e.g. blood work, injury history, menstrual status, bone mineral density, etc.), many of which are highlighted in Table 1. These accumulating risk factors result in categorizing athletes as low (green; full training and competing), moderate (yellow; continued training, with supervision, some modifications, and regular assessments) or high risk (red; athlete removed from sport for health and safety reasons but clinically supported and reassessed regularly). The tool also provides a RED-S specific return-to-play framework to facilitate these decisions [32]. The Low Energy Availability in Females Questionnaire (LEAF-Q) is a diagnostic screening tool, validated in female endurance athletes and dancers, to facilitate the identification of athletes at risk for RED-S [49]. A LEAM-Q screening tool for male athletes is under development. The Brief Eating-Disorder in Athletes Questionnaire (BEDA-Q) is another validated tool to aid in the diagnosis of eating disorders in athletes, which may be an underlying cause of RED-S [50].

It has long been recognized that LEA is the underlying etiology of the Triad, and by extension the RED-S syndrome [44, 51]. However, it is important to note that despite a mathematically simple definition (Fig. 1), the *accurate* calculation of EA is challenged by methodological considerations that introduce risk for significant under- or overestimation of EI [52] and/or EEE [53] and confound accurate estimations of daily dietary CHO intake [45, 54]. Furthermore, the "low" EA threshold of ~ 30 kcal/kg FFM/day which was established by assessing luteinizing hormone (LH) pulsatility every 10–15 min for 24 h under strictly controlled laboratory conditions, along with other LEA indicators such as bone turnover markers and triiodothyronine (T3), in healthy sedentary females [55, 56], may not translate to menstrual disturbances in free-living female athletes and/or other athlete cohorts, obviously including males. Indeed, experimental evidence has not always shown a relationship between measured EA and menstrual disturbances [45, 54, 57, 58]. Furthermore, an EA threshold in males remains to be established, but is thought to be lower [45, 59–63], while EA thresholds in various classifications of para-athletes continue to be speculative [64–66]. These methodological or diagnostic challenges associated with the precise assessment of EA underpin the complexity associated with accurate RED-S diagnoses [54], and by extension many situations of LEA may go undetected and lead to a potential misdiagnosis of NFOR/OTS, instead of RED-S.

2.3 Extensive Symptom Commonalities Between OTS and RED-S Resulting In Challenging Diagnoses

Given that many NFOR/OTS outcomes are based on the HPA axis (primarily cortisol/testosterone imbalance with regard to OTS [6, 28, 29, 33, 67, 68]), while many of the RED-S outcomes are based on the HPG axis [15, 16, 69–71], and thus both initiating from a hypothalamic-pituitary origin, it should not be surprising that NFOR/OTS and RED-S have significant commonalities of symptoms. Table 1 provides a primary original study comparison of symptom similarities (as reported in each individual study) clustering around six performance and seven health-based groupings (n = 57 OTS studies and n = 88 RED-S/Triad studies found). It is beyond the scope of this review to extensively elaborate on the impact and/or directionality of every marker, but we direct the reader to several OTS [31, 33, 72] and RED-S/Triad [17, 69, 73] reviews on this subject. These aforementioned reviews highlight many overlapping symptoms of OTS and RED-S, but our understanding of a definitive set of symptoms to diagnose either syndrome remains poor.

Indeed, across these 13 general performance and health symptom outcome groupings, RED-S/Triad and OTS share reported symptom similarities for all but one of them (bone outcomes). The predominant RED-S diagnostic criteria centers around endocrinology (sex and metabolic hormones) and bone health outcomes leading to injury (Table 1), due to the strongly developed research spanning nearly 40 years on the Triad [17], which triangulates on EA, reproductive and bone health. Conversely, OTS studies have centered primarily on associated hormone and blood work markers which have been shown to be associated with endurance performance and training adaptation decrements. Furthermore, both OTS and RED-S can also be significantly impacted by psychogenic factors and lifestyle stress. It is also interesting to note that the 88 identified RED-S/Triad studies feature primarily female athletes/subjects [female n = 7400 (78%); Males n = 2105 (22%)]. Conversely, the opposite sex breakdown is found across the 57 various training-overload/OTS studies [females n = 210 (19%); males n = 880 (81%); data only reported for studies given subject and sex breakdown numbers].

Several previous OTS reviews have potentially misdiagnosed OTS by highlighting a potential caloric mismatch (LEA) or low CHO availability as a confounding factor involved in the development of OTS [5, 28, 29, 31, 33, 35, 67, 68], instead of the development of RED-S. As emphasized above, OTS and RED-S identification are both based on a diagnosis of exclusion, as they mutually lack a single validated universal identifier for diagnosis. However, as highlighted within the OTS consensus statement, negative energy balance and/or insufficient CHO should be excluded

from the OTS diagnosis [6]. Nevertheless, both OTS and RED-S feature accumulating quantitative and qualitative symptoms resulting in more certainty of accurate identification [6, 32, 74]. However, excluding LEA/RED-S from an OTS diagnosis is incredibly challenging, given the significant challenges with accurate EI and EEE measurement leading to RED-S identification [54]. Furthermore, both OTS and RED-S are syndromes of "under-recovery" resulting in a constellation of symptom overlap between OTS and RED-S (Table 1 and Section 3 below), which again makes accurate diagnosis challenging. Importantly, the current OTS consensus statement, published in 2013, pre-dates the 2014 introduction of the RED-S syndrome and its recognition of a wider sequalae of outcomes of chronic LEA [15]. In addition, because the focus of LEA within the Triad may have prevented consideration of its presence in male athletes [62, 75, 76], the overlap with the significant prevalence of males (81%) in training-overload/OTS studies may have gone unnoticed. The wide-ranging and over-lapping OTS and RED-S/Triad symptoms (Table 1) span across nearly all sport science and medicine disciplines. Therefore, to adequately prevent, diagnose and/or treat the multi-factorial underpinnings of under-recovery (OTS/RED-S), the collaborative expertise within medical, nutrition, physiology and psychology are all particularly essential (Fig. 2) [77]. Unfortunately, many athletes/coaches do not have access to such an extensive multi-disciplinary expertise.

Undeniably, the close parallels of OTS and RED-S symptoms are remarkable, resulting in significant chance for misdiagnoses; thus, the pursuit of more clear and separate diagnostic criteria for both syndromes needs more scientific consideration. However, the further development of accurate diagnostics and validated assessment tools is also scientifically challenging, because they are typically reliant on the collection of the prevalence of a range of cohort symptoms in cross-sectional observational studies rather than the more scientifically robust intervention-based randomized clinical trial (RCT) designs. Generally, long-term interventional RCTs are less ethically feasible since one would need to induce full-blown OTS or RED-S for an accurate and comprehensive diagnoses. Accordingly, as outlined below, most of the intervention-based OTS literature are actually short-term studies inducing FOR/NFOR, rather than actual OTS.

3 Does Unappreciated Under-Fueling (LEA) and/or Low CHO Availability Confound Much of the Training-Overload/OTS Literature?

In this section, we hope to further demonstrate the hypothesis that many of the negative outcomes of training-overload (with, or without an OTS, NFOR or FOR diagnosis) may primarily be due to misdiagnosed under-recovery from under-fueling (LEA leading to RED-S). Evidence for this hypothesis is derived from published studies implementing training-overloads (many of which resulted in an OTS or NFOR diagnosis) that have also collected and analyzed for EI and potentially EEE, energy balance (EB) and/or EA during the training-overload period to enable analysis and comparison of EA between or within cohorts. Herein EB = EI – total daily energy expenditure (TDEE = Basal Metabolic Rate (BMR) + EEE + Non-Exercise Activity Thermogenesis (NEAT) + Thermic Effect of Food (TEF)). With these criteria, we identified 21 investigations summarized in Table 2 (Note: we have highlighted the extensive series of Endocrine and Metabolic Responses on Overtraining Syndrome (EROS) publications as a single analyzed investigation). Here, we made within- ($n = 9$ studies) and between-group ($n = 12$ studies) comparisons to demonstrate potential LEA where EEE has been reported or where training loads have otherwise been characterized (as increase/decrease compared to control condition). Two papers reported actual EA data [19, 22], while another four studies [78–81] reported EEE (along with EI and FFM; thus EA could be directly calculated) and two papers reported total daily EE [82, 83]. In four studies [84–87], we were able to estimate EEE using a metabolic equivalent of task (MET) approach [88] or for running data, utilizing the conversion factor 1 kcal/kg BM/km of running [89]. In addition to the two studies reporting EA data [19, 22], there were enough data to enable EA and/or CHO availability estimations in 9 of 21 studies [20, 78–81, 83–85, 87], but not in the remainder [21, 82, 90–97], so we examined the *relative* (increase/decrease/no change) differences and overall associated direction in EA and/or CHO availability.

3.1 Energy Availability Analysis

Fourteen of the 21 studies [19–22, 78–81, 83–85, 90–92, 97] showed indications of an EA decrease across time or difference between two cohorts within a given study during the training-overload/OTS period compared to control conditions (Table 2). Within these 14 studies, ten studies (71%; [19, 21, 22, 78, 83–86, 90, 91, 97, 98]) reported OTS or RED-S symptoms likely linked to lower energy or CHO availability (across time or compared to the control group); meanwhile, two investigations [79, 80] reported those symptoms in both groups (i.e. no between-group difference) while no REDS or OTS symptoms were shown in the last two studies [81, 92]. Only eight studies reported performance outcomes, with a performance impairment (relative to baseline or control group) evident in five studies [21, 22, 78, 86, 97], with the rest [79, 80, 85] showing either no change or no difference between groups.

The impact of significantly increased EEE has on EA outcomes in OTS was first reported more than three decades ago, as Costill et al. [80] observed lowered EA in 12 male collegiate swimmers during a 10-day intensive training block. Based on subjective feelings of fatigue, the authors divided the athletes retrospectively into OTS and non-OTS (paper identified OTS distinction) and discovered an ad libitum EI of 4682 kcal/day in the non-OTS swimmers versus ~ 20% lower EI (3631 kcal/day) in the OTS group despite the same increase in EEE. We estimate EA to have been around 20 and 36 kcal/kg FFM/d for OTS and non-OTS, respectively. Accordingly, the authors suggest that OTS swimmers may have been in a ~ 1000 kcal/day energy deficit, which was accompanied by several training overload/OTS or RED-S related symptoms while noting that because this time period is likely too brief to induce a full-blown OTS case (nor is that ethically advisable in a study), subjects were probably NFOR. The only cross-sectional investigation included in this analysis, albeit one that resulted in a total of 13 publications (of which 3 were relevant for the purpose of our summary in Table 2, [90, 91, 98]), implemented an evaluation of 67 health parameters in athletes with OTS (n = 14), healthy athletes (n = 25), and healthy non-athlete controls (n = 12). Here, food diaries [91] indicated drastically lower EI in OTS vs healthy athletes (estimated at 30 vs 58 kcal/kg/day). Although no training data were provided, the nearly twofold greater EI of healthy versus OTS athletes is likely to translate into a significant difference in EA between groups. Several publications on various aspects of this single study support our hypothesis of the potentially confounding role that low energy and/or CHO intake may play in the misdiagnosis of OTS [90, 99]. We concede, however, that since the collection of dietary intake data in this study was implemented only after athletes had already presented with symptoms of OTS, the energy and CHO intakes reported in food diaries may not represent habitual intake, but rather a compensation to reduced training volumes. As no information is provided on training in any of the papers by the Cadegiani group, and as we point out in table 2, it is impossible to estimate EA for these athlete groups. Taken together, our analysis suggests between- and within-group differences in EA ranging from 15–100% and 18–28%, respectively (Table 2). Although a difference of 15–18% in EA between intervention groups may appear small, one study has demonstrated that this translates into a 450 kcal daily negative deviation from EB [83], with all other studies in our analysis demonstrating much greater energetic mismatches than ~ 15 to 18%. Indeed, even small within-day energy deficits (only 300–400 kcal) have been associated with clinically meaningful RED-S symptom outcomes in

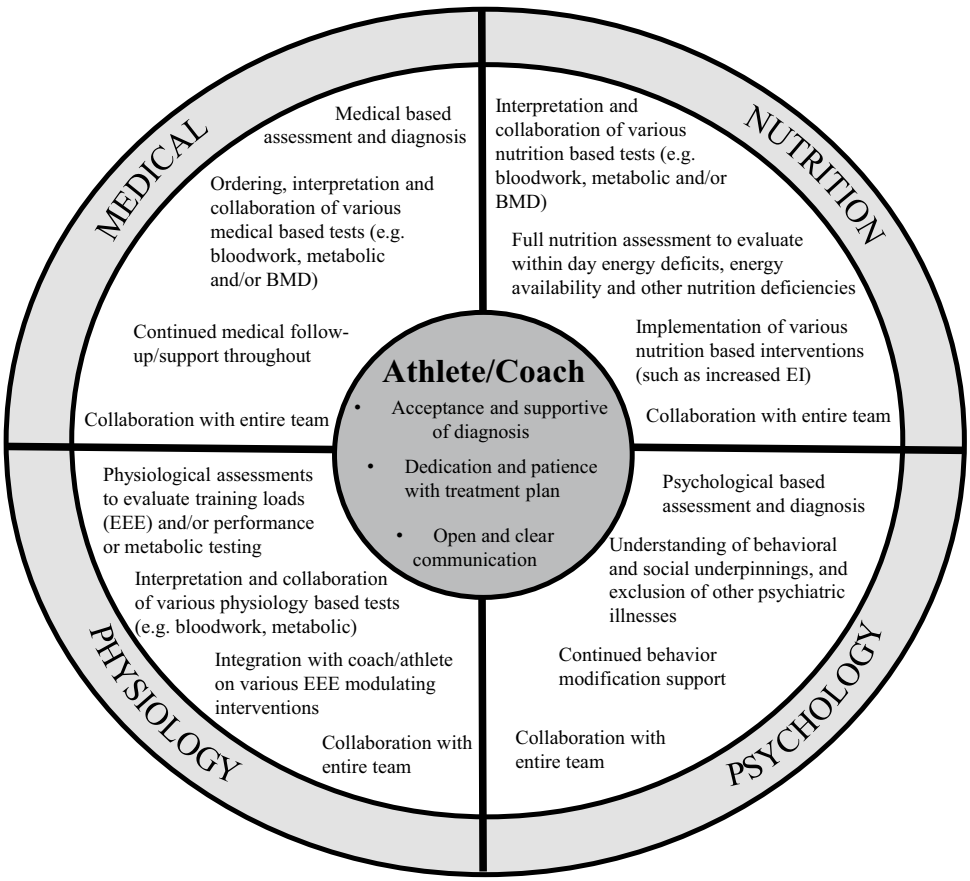

Fig. 2 Theoretical responsibilities and accountabilities model of the multi-discipline practitioner approach required to monitor/prevent, diagnose and treat OTS or RED-S in an athlete, with an associated coach.

Table 2 Caloric and dietary analysis of primary training-overload studies (with or without an OTS/NFOR/FOR diagnosis) highlighting where low energy and/or carbohydrate availability may be an underlying factor. It is important to note that only studies that characterize a significant and sustained decline in performance outcomes can be termed, by definition, OTS, NFOR or FOR (see section 2.1).

Study	Participants	Study design	Training and dietary intervention	Energy intake (EI), expenditure (EEE), and energy availability (EA)	CHO intake	OTS and RED-S outcomes
Training-overload studies (with or without an OTS/NFOR/FOR diagnosis) that report or estimate differences EI, EEE and/or EA (n = 14)						
Barr et al. 1992 [81]	$n = 24$ male collegiate swimmers; VO_{2max} ~ 52 and ~ 56 mL/kg/min for Short and Long, respectively	Parallel study design over 6 weeks (increased [long: $n = 12$] or habitual [Short: $n = 12$] training volume)	**Training intervention**: ↑ + 200% (long: 44 km/week vs short: 22 km/week swimming) compared to habitual training volume (supervised field training) **Dietary intervention:** Ad libitum. 2 days food diaries on week 4 of the 6-week block	**EI:** From baseline to intense training: ↑ + 13%; long: 3728–4230 kcal/day; ↓ − 11% Short: 3609–3227 kcal/day; Intense training block: ↑ + 24%; Long vs Short **EEE:** ↑ 200%; Long: 1793 kcal/day vs Short: 884 kcal/day **EA (estimated):** ↓ ~ − 14 to 15% in both; long: 44–38 kcal/kg FFM/day; Short: 39–33 kcal/kg FFM/day	**CHO intake:** from baseline to intense training: ↑ + 20%; long: ~ 7.0 g/kg/day to ~ 8.3 g/kg/day; ↓ − 5% Short: ~ 6.3 g/kg/day to ~ 6.0 g/kg/day	**BM:** ↔ in both groups **BF%:** ↓15% in both groups
Cadegiani and Kater 2019 [90]; Cadegiani and Kater 2018 [91]; Anderson et al. 2021 [98]	OTS-affected athletes (OTS, $n = 14$), healthy athletes (ATL, $n = 25$), and healthy non-physically active controls (NCS, $n = 12$); Athletes: weekly exercise at least four times and >300 min of moderate-vigorous training intensity	A cross-sectional evaluation of 67 parameters and their differences between OTS, ATL and NCS	**Training assessment:** not reported **Dietary assessment:** self-report for 1 week	**EI:** ↓ 48% OTS: 30 kcal/kg/day vs ATL 58 kcal/kg/day vs NCS 58 kcal/kg/day **EEE**: not reported **EA:** not reported—but likely ↓ OTS vs ATL *(Although EEE data is not reported, clearly significant difference in EI between groups and, thus, likely lower EA in OTS group)*	**CHO intake:** ↓ 48% OTS: 3.8 g/kg/day vs. ATL 7.3 g/kg/day	**Basal hormones**: Testosterone ↓ − 22% OTS (422.6 ng/dL) vs ATL (540.3 ng/dL) Estradiol ↑ + 26% OTS (40.1 pg/mL) vs ATL (29.8 pg/mL) Testosterone:estradiol ratio ↓ − 48% OTS (10.84) vs ATL (20.85) ↔ IGF1, T3, T:cortisol ratio, CRP, ferritin in OTS vs ATL Salivary cortisol response upon awakening ↓ in the OTS vs ATL **Mood:** ↓ OTS vs ATL POMS Fatigue subscale 18.9 OTS vs 1.7 ATL

Table 2 (continued)

Study	Participants	Study design	Training and dietary intervention	Energy intake (EI), expenditure (EEE), and energy availability (EA)	CHO intake	OTS and RED-S outcomes
Costa et al. 2005 [84]	$n = 32$ male triathletes; VO_{2max} 69.9 ± 6.5 mL/kg/min; typical training loads of 20.9 ± 5.8 h/week	Parallel group design (CON [$n = 16$] or HCHO [$n = 16$]) for 6 days	**Training intervention:** Habitual training (3 h/day) with additional 1 h/day running at 70% VO_{2max} (total load ↑ + 133%) **Dietary intervention:** Athletes allocated into control (CON; self-selected) or high CHO (HCHO; 12 g/kg/day CHO) groups (food diary assessed post-study).	**EI:** ↓ 34% CON: 2818 kcal/day vs. HCHO: 4246 kcal/day **EEE (estimated):** not reported, but equal for each group and monitored. Estimated (via METS) at ~ 2250 kcal habitual and up to ~ 2800 kcal during 6 days intervention **EA (estimated):** ↓ 50–100% for CON and ↑ 43–100% for HCHO CON: baseline ~ 11 to 18 kcal/kg FFM/day CON: 6 days intervention: ~ 0 to 9 kcal/kg FFM/day HCHO: baseline ~ 11 to 18 kcal/kg FFM/day HCHO: 6 days intervention: ~ 23 to 32 kcal/kg FFM/day	**CHO intake:** ↓ 100% CON: ~ 5.9 g/kg/day (419 g/day) vs. HCHO: ~ 11.9 g/kg/day (838 g/day)	**Hormones**: ↑ + 43% cortisol response to exercise with CON
Costill et al. 1988 [80]	$n = 12$; male collegiate swimmers; VO_{2max} ~ 62 mL/kg/min	Longitudinal design for 10 days	**Training Intervention:** Two-week training phase + 10 days intensified training phase ↑ + 200% compared to baseline. Post training an OTS and non-OTS groups identified **Dietary intervention:** Self-report on days 1, 2, 9 and 10	**EI:** ↓ 23% OTS: 48 kcal/kg/day (3631 kcal/day) vs. non-OTS 63 kcal/kg/day (4682 kcal/day) **EEE**: ↑ + 200% vs. habitual (Assume identical ~ 2300 kcal/day EEE for both OTS and non-OTS groups) **EA (estimated):** ↓43% OTS: ~ 20 kcal/kg FFM vs. non-OTS: ~ 36 kcal/kg FFM	**CHO intake:** ↓35% OTS: 5.3 g/kg/day vs. non-OTS: 8.2 g/kg/day	**Muscle glycogen:** ↓ 24% OTS vs. non-OTS **Submax HR, lactate and max HR**: ↓ Both groups **Performance**: ↔ Both groups **Training distance covered**: ↓ 10% in OTS vs. non-OTS

Table 2 (continued)

Study	Participants	Study design	Training and dietary intervention	Energy intake (EI), expenditure (EEE), and energy availability (EA)	CHO intake	OTS and RED-S outcomes
Killer et al 2017 [85]	$n = 13$ highly-trained cyclists; VO_{2max} 72.2 ± 4.9 mL/kg/min	Cross-over design for 9 days with a 10 days washout (LCHO vs HCHO)	**Training intervention:** Intensified training (↑ + 250% compared to baseline) with (HCHO) or without (LCHO) CHO support before, during and after training. Training equal across groups and training monitored throughout **Dietary intervention:** 2 × 3 days self-report habitual intakes before and after the training block. Controlled CHO intakes before, during and after training: HCHO: pre training: 24 g CHO; during training: 60 g/h CHO; post training: 44 g CHO + 17g protein LCHO: pre training: 2 g CHO; during training: 20 g/h CHO; post training: 11 g CHO	**EI:** ↓ − 16% LCHO: 3501 kcal/day vs HCHO: 4148 kcal/day **EEE (estimated)**: not reported, but equal across groups and training monitored throughout. Estimated (via METS) as ~ 2400 kcal/day **EA (estimated):** ↓ − 36% LCHO: ~ 19 kcal/kg FFM/day vs. HCHO: ~ 29 kcal/kg FFM/day	**CHO intake:** ↓ − 25% LCHO: 7.4 g/kg/day vs. HCHO: 9.9 g/kg/day	**Performance:** ↓ − 4.1% both groups (Wmax) **Sleep**: ↓ − 6.7% both groups (sleep efficiency) **Mood**: ↓ LCHO vs HCHO
Noland et al. 2001 [92]	$n = 21$ (12M, 9F) intercollegiate swimmers	Longitudinal design including pre-season testing and 9 weeks of gradual overload (testing at 4 and 9 weeks)	**Training intervention**: ↑ + 147% (~ 40 km/week vs 27 km/week compared to habitual training volume). Training supervised field training. **Dietary intervention:** ad libitum. 3 days food diaries during pre-season week and at 4-week and 9-week points.	**EI:** ↔ in women; ↓ in men (pre-season to week 4 and 9) **EEE:** ↑ in both women and men (pre-season to week 4 and 9; but kcal not reported) **EA:** not reported—but likely significant ↓ *(cannot estimate due to lack of specific data. But assume significant ↓ due to changes reported in EI and ↑ EEE components)*	**CHO intake**: not reported	**BM:** ↔ in both **BF%:** ↓ ~ 17% in women (14.3–11.8%); ↔ in men **Hormones**: ↔ leptin in both

Table 2 (continued)

Study	Participants	Study design	Training and dietary intervention	Energy intake (EI), expenditure (EEE), and energy availability (EA)	CHO intake	OTS and RED-S outcomes
Poffe et al. 2019 [78]	$n = 18$ healthy active males; 4.8 ± 0.4 h/week activity; VO_{2max} ~ 56 mL/kg/min	Longitudinal 3-week intensive cycling program (Ketone ester (KE) supplementation, $n = 9$; Control (CON), $n = 9$)	**Training intervention**: ↑ + 120%, + 165%, and + 300%/week on weeks 1, 2 and 3 compared to habitual training volume (supervised lab training). Training load 15% higher in the KE vs. CON group on week 3. **Dietary intervention:** Ad libitum dietary intakes (recording for 2 days at the beginning, middle and end). Controlled post-exercise recovery drinks for both groups (500ml CHO-protein drink). Immediately post-exercise and 30 min pre-sleep a ketone ester drink for KE group.	**EI:** KE**:** ↑ + 20%; 3513 kcal to 4207 kcal/day CON**:** ↔ Slight decrease of 92 kcal/day at week 2 and 38 kcal/day at week 3 **EEE:** ↑ 1100–1530–2294 kcal/day on weeks 1, 2 and 3 *Weeks 1 and 2 identical between KE and CON/ week 3*: KE: 2454 kcal/ day; CON: 2142 kcal/ day **EA (estimated):** KE: ↔ ↓ − 21%; ~ 47, 48 and 37 kcal/kg FFM/day on weeks 1, 2 and 3 CON: ↓↓ − 35%; ~ 51 to 42 to 33 kcal/kg FFM/ day from weeks 1–2–3	**CHO intake:** KE: ↑ + 9% (6.6, 8 and 8.3 g/ kg/day on weeks 1, 2, and 3) CON: ↔ (6.7, 7.0 and 6.9 g/kg/day on weeks 1, 2 and 3) ↑ *KE vs CON on weeks 2 and 3.*	**Performance:** KE: ↑ 4.9% MPO in the 30 min cycling TT immediately post-intervention; MPO on days + 3 and + 7 post-intervention 7.5% and 8.3% higher than pre-test CON: ↔ MPO in the 30 min TT immediately after intervention; On day + 7 post-intervention,: MPO ↑ 7.0%; KE vs CON: ↑ ~ 4.9% immediately and ↑ ~ 7.5% on day + 3 post-intervention; ↔ on day + 7 post-intervention **Body fat%:** ↓ − 1.4% in both groups **Resting HR:** KE: ↔/ CON: ↓− 20% (from 66 to 55 bpm)
Rämson et al. 2008 [83]	$n = 8$ trained collegiate male rowers; training experience 7.5 ± 2.2 years	Longitudinal design: 1 week baseline training followed by 2 weeks of intensified and 1 week of recovery training	**Training Intervention**: ↑ + 150–165% on weeks 1 and 2 of the 2-week intensified block compared to habitual training volume (supervised field training) **Dietary intervention:** Ad libitum. 3 × 3 days food diaries at the end of baseline, second week of intensified training, and recovery week	**EI:** ↑ + 8.7%; Baseline: 4488 kcal/day vs. intensified training: 4880 kcal/day **Total daily EE:** ↑ + 16.4%; baseline 4576 kcal/day vs. intensified training: 5329 kcal/day **EA (estimated):** ↓ ~ − 18% *(energy balance (EB) equations utilized: baseline EB ~ − 88 kcal/day to large negative EB of ~ 449 kcal/ day during intensified training)*	**CHO intake:** not reported	**Recovery state**: ↓ RESTQ profile with increased Social Stress and Fatigue scores as well as a decrease in RESTQ-Index **Hormones**: ↓ − 29% fasting leptin

Table 2 (continued)

Study	Participants	Study design	Training and dietary intervention	Energy intake (EI), expenditure (EEE), and energy availability (EA)	CHO intake	OTS and RED-S outcomes
Schaal et al. 2021 [22]	$n = 16$ female runners divided retrospectively into well adapted (WA; $n = 9$) and NFOR ($n = 7$); VO_{2peak}: WA 52.0 ± 1.7 mL/kg/min; NFOR: 51.8 ± 1.3 mL/kg/min	Longitudinal study design over 10 weeks (4-week baseline; 4-week intervention; 2-week recovery)	**Training Intervention:** 4 weeks of training overload ↑ + 131% followed by a 2-week recovery **Dietary intervention:** Ad libitum, 7-day food logs during each phase	**EI:** WA ↑ + 4.2% (46.1–50.4) vs NFOR ↔ + 0.6% (53.4–53.9 kcal/kg LBM) baseline to intervention **EEE**: WA ↑ + 4.0% (22.4–24.8) vs NFOR ↑ + 5.5% (22.9–29.1 kcal/kg LBM) baseline to intervention **EA:** WA ↔ + 2.5% (24.4–26.3) vs NFOR ↓ − 5.6% (30.4–24.8 kcal/kg LBM) baseline to intervention	**CHO intake**: WA ↑ + 17.8% (213–251 g/day or ~ 3.7 to 4.4 g/kg/day); NFOR ↔ + 6.8% (292–312 g/day or ~ 4.9 to 5.3 g/kg/day) baseline to intervention	**Running performance:** WA ↑ + 4.4% vs. NFOR ↓ − 9.2% **Recovery**: WA ↔ (REST-Q fatigue subscale from 1.6±0.2 to 2.0±0.3 points) vs. NFOR ↓ (REST-Q fatigue subscale + 78% ; from 1.5±0.2 to 2.6±0.4 points) **REE**: WA ↑ + 8.4 % (27.8–29.8 kcal/kg LBM/day) vs. NFOR ↔ (28.6–28.8 kcal/kg LBM/day) **BM**: WA ↓ − 1.0% (57.6–57.0 kg) vs. NFOR ↔ (59.1–58.7 kg) **Hormones**: **leptin**: NFOR ↓ − 17% (3625–2994 ng/mL) vs. WA ↔ (4311–4142 ng/mL) **Estradiol**: NFOR ↓ at the mid-cycle point (− 27%) and in the first 5 days of the luteal phase (− 47%); as well as decreased luteal phase length vs. WA ↔ **Oral temperature**: NFOR ↓ (36.4–36.1 °C) vs. WA ↔ (36.4–36.3 °C)

Table 2 (continued)

Study	Participants	Study design	Training and dietary intervention	Energy intake (EI), expenditure (EEE), and energy availability (EA)	CHO intake	OTS and RED-S outcomes
Schaal et al. 2017 [19]	$n = 11$ elite female synchronized swimmers; VO_{2max} 62.1 ± 0.3 mL/kg/min	Longitudinal study design over 4 weeks	**Training intervention:** increased volume of ↑ + 119% in training impulse compared to baseline **Dietary Intervention:** 4-days self-report at baseline and the use of photography on weeks 2 and 4	**EI:** ↔ (44.4–47.1–42.1 kcal/kg LBM) **EEE**: ↑ + 125% (19.3–24.9–24.1 kcal/kg LBM/day) **EA:** ↓ − 28% (25–22.3–18 kcal/kg LBM)	**CHO intake**: ↔ (~ 4.7 to ~ 5.1 to ~ 4.5 g/kg/day; or 275–302–262 g/day) *While ↔ in absolute intake, likely ↓ in CHO availability due to ↑ EEE*	**BM and LBM:** ↔ **Fat mass:** ↓ − 5.6%/**BF%:** from 17.3% to 16.4% **Hormones**:↓ − 38% leptin, ↑ + 7% ghrelin; compared to baseline; ↔ cortisol
Snyder et al. 1995 [97]	$n = 8$ male competitive cyclists	Longitudinal study design	**Training intervention:** increased volume of ↑ + 146%; Normal training (moderate intensity, long duration, ~ 12.5 h/week; 7 days, NORM); overtraining (high intensity training, ~ 18 h/week; 15 days, OVER); recovery (minimal training, 6 days, REC) **Dietary intervention:** a liquid CHO supplement (160 g CHO) daily within 2 h after training; ad libitum intakes recorded over 4 days at the end of each phase	**EI:** ↔ (3274–3370 kcal/day NORM to OVER) **EEE:** not reported **EA:** not reported *(cannot estimate due to lack of specific data. But assume significant ↓ due to no change in EI combined with a + 146% increase in training volume (EEE likely even further increased due to focus on higher intensity during OVER)*	**CHO intake**: ↔ (~ 512 to 548 g/day or ~ 6.9 to 7.4 g/kg/day) *While ↔ in absolute intake, likely ↓ in CHO availability due to ↑ EEE*	**Resting muscle glycogen:** ↔ (NORM = 530.9 +/- 42.5, OVER = 571.2 +/- 27.5 µmol/g^{-1} dry weight) **BM, LBM and BF:** ↔ **Resting HR and sleep:** ↔ **Wmax** ↓ − 3% for 7/8 subjects NORM to OVER **Maximal HR**: ↓ NORM to OVER **Plasma cortisol**: ↓ − 26% (515–382 nmol/L NORM to OVER)

Table 2 (continued)

Study	Participants	Study design	Training and dietary intervention	Energy intake (EI), expenditure (EEE), and energy availability (EA)	CHO intake	OTS and RED-S outcomes
Svendsen et al. 2016 [79]	$n = 13$ male cyclists; VO_{2max} 72 ± 5 mL/kg/min	Cross-over design for 8 days with a 15-days washout (LCHO vs HCHO)	**Training intervention**: Intensified training ↑ + 200% compared to baseline (individual prescribed) **Dietary intervention:** Self-record food diaries throughout interventions. LCHO: 2% CHO solution before, during and after training sessions or HCHO: 6% CHO before, during and after training sessions + 20 g protein after training	**EI:** ↓ 18% LCHO: 3513 kcal/day vs. HCHO: 4159 kcal/day **EEE:** ~ 5807 kcal over 8 days so ~ 725 kcal/day (↑ compared to baseline; ↔ between groups) **EA (estimated):** ↓ 20% LCHO: ~ 46 kcal/kg FFM/day vs. HCHO: ~ 57 kcal/kg FFM/day	**CHO intake:** ↓ 26% LCHO: 7.2 g/kg/day vs. HCHO: 9.7 g/kg/day	**PPO:** ↓ − 4.1% both groups **BM:** ↔ both groups **Cortisol:** ↑ ~ 44% both groups vs baseline **Inflammatory markers:** ↑ + 112% LCHO vs + 55% HCHO
Woods et al. 2017 [20]	$n = 10$ national team rowers	Longitudinal design over 4 weeks	**Training intervention:** intensified training (↑ + 125% compared to baseline) **Dietary intervention:** 2 × 3 days self-report before and after the training block	**EI:** ↔ 3585 kcal/day (no change pre-post) **EEE (estimated)**: not reported, but estimated EEE using METS to be ~ 2740 kcal/day **EA (estimated):** ~ 12 kcal/kg FFM/day	**CHO intake:** ↔ 480 g/day or 6 g/kg/day (no change pre-post)	**Performance:** ↓ − 13.3% power output (on-water 5 km rowing) **RMR**: ↓ − 5.7%; from 33.2 to 31.3 kcal/kg FFM/day **BM**: ↓ − 2% (− 1.6 kg)/**BF%:** ↓ − 8.3% (− 2.2 kg) **Fatigue** ↑ **Mood and sleep**: ↓

Table 2 (continued)

Study	Participants	Study design	Training and dietary intervention	Energy intake (EI), expenditure (EEE), and energy availability (EA)	CHO intake	OTS and RED-S outcomes
Woods et al. 2018 [21]	$n = 13$ male cyclists; VO_{2max} 61.1 ± 6.2 mL/kg/min (> 200 km/week, >4 years)	Longitudinal design with 3-week intensified training	**Training intervention:** Intervention: 1 week baseline training; 3 weeks of intensified training (↑ + 120% to + 150% compared to baseline); 2weeks of recovery (80% compared to baseline). Weekly training prescribed individually using Training Stress Score (TSS); all sessions monitored and adjusted if required **Dietary intervention:** self-report throughout the study	**EI** ↔ Baseline 3707 kcal/day Intensified training: 3896–4140 kcal/day (↑ ~ 5% to ~ 12% compared to Baseline) Recovery 3585 kcal/day **EEE:** not reported **EA:** not reported *(cannot estimate due to lack of specific data. But assume significant ↓ due to changes reported in EI and ↑ EEE components)*	**CHO intake:** not reported	**Performance:** ↓ 21.1% from baseline to loading (PPO for the 15s sprint) **RMR**: ↓ 12% from baseline to loading **BM and FFM**: ↓ ~ 1.4% from baseline to loading for both **Fat mass:** ↓ 14.7% from baseline to loading **Appetite** ↓ ~ 5.6% from baseline to loading **Hormones**: ↔ in leptin or T3 **Mood**: ↓ from baseline to loading
Training-overload studies (with or without an OTS/NFOR/FOR diagnosis) that report or estimate differences in CHO intake (n = 4)						
Achten et al. 2004 [93]	$n = 7$ trained runners; >50 km/week running; VO_{2max} 64.7 ± 2.6 mL/kg/min	Cross-over design for 11 days with a 10-days washout (CON vs HCHO)	**Training intervention:** ↔ equivalent overload (training prescribed, but no details given) **Dietary intervention:** Isocaloric diets controlled and given, providing either high (HCHO: 65E%; ~ 8.5 g CHO/kg/day) or low (CON: 40E%; ~ 5.4 g CHO/kg/day) CHO availability	**EI:** ↔ (52.3 and 53.8 kcal/kg/day) **EEE**: not reported **EA:** not reported *(cannot estimate due to lack of specific data. But assume identical between conditions due to matched EI and training)*	**CHO intake:** ↓ 36%; HCHO: ~ 8.5 g/kg/day vs. CON: ~ 5.4 g/kg/day	**Performance: (8km running TT)** CON ↓↓ − 8.2% vs. HCHO ↓ − 3.2% **Mood:** (POMS-65 scores): CON ↓ + 17.8% vs. HCHO ↔ **BM:** ↔ for CON and HCHO

Table 2 (continued)

Study	Participants	Study design	Training and dietary intervention	Energy intake (EI), expenditure (EEE), and energy availability (EA)	CHO intake	OTS and RED-S outcomes
Kirwan et al 1988 [94]	$n = 10$ highly trained male distance runners; VO_{2max} 67.6 ± 1.0 mL/kg/min	Cross-over design for 5 days (LCHO vs HCHO)	**Training intervention:** 3 days baseline diet and training (80% habitual distance @70% VO_{2max}) followed by 5 days of either low or high CHO (isocaloric) diet and intensified training (↑ + 150% habitual distance @80% VO_{2max}). Training outdoors, HR monitors used for recording; duration recorded **Dietary intervention:** Self-report throughout the study; Habitual: 6.2 g CHO/kg CHO	**EI:** ↔ (but data not shown) **EEE**: not reported but ↑ compared to baseline **EA**: not reported *(cannot estimate due to lack of specific data. But assume ↓ due to no change in EI and ↑ EEE)*	**CHO intake:** ↓ LCHO: 3.9 g/kg/day vs HCHO: 8 g/kg/day	**Muscle glycogen:** ↓ − 33% LCHO vs. HCHO **Performance:** ↓ − 3.4% LCHO vs. HCHO (running economy)
Mujika 2019 [95]	$n = 1$ world class long distance triathlete; case study	Longitudinal case study over 32 weeks, with races on weeks 21, 24 and 32	**Training intervention:** ↔ (self-report) **Dietary intervention:** 32-week LCHF diet in a habitual HCHO vegetarian athlete, with acute restoration of high CHO availability on the pre-race meals (food logs, self-report)	**EI:** ↔ 3965 kcal/day on HCHO and 3984 kcal/day on LCHF **EEE**: not reported **EA:** not reported *(cannot estimate due to lack of specific data. But assume ↔ across time (identical EI and EEE))*	**CHO intake:** ↓↓↓ General preparation (HCHO) ~ 8.5 g/kg/day (~ 638 g/day); intervention (LCHF) ~ 0.7 g/kg/day (~ 52 g/day)	**Triathlon race performance:** ↓ Weeks 21, 24 and 32 of the LCHF diet (with DNF for the final race). Recovery of performance after 5 weeks of habitual HCHO diet post-LCHF intervention **Mood:** ↓ **Resting and submaximal HR:** ↑ Resting HR from ~ 40 to ~ 48–50 bpm; submaximal HR ~ 7 bpm higher **BM:** ↓ ~ 76kg to 71.6 kg (− 5.8%) but then restored back to 76kg while on LCHF diet

Table 2 (continued)

Study	Participants	Study design	Training and dietary intervention	Energy intake (EI), expenditure (EEE), and energy availability (EA)	CHO intake	OTS and RED-S outcomes
Simonsen et al. 1991 [96]	$n = 22$ collegiate rowers (12M, 10F); VO_{2peak} ~ 55 and ~ 42 mL/kg/min for M and F, respectively	Parallel group design (LCHO vs HCHO) over 4 weeks	**Training Intervention**: Twice-a-day training (6 days/week); no information provided on change compared to habitual training volume (supervised lab and habitual field training) **Dietary intervention:** controlled, isocaloric: HCHO 10 g CHO/kg/day vs. LCHO 5 g CHO/kg/day	**EI:** not reported, but ↔ between groups **EEE:** not reported, but ↔ between groups **EA:** not reported *(cannot estimate due to lack of specific data. But assume ↔ between groups (identical EI and EEE))*	**CHO intake:** ↓ 50%; LCHO: 5 g/kg/day vs HCHO: 10 g/kg/day	**Performance:** ↑ + 10.7% HCHO vs. ↑ + 1.6% LCHO compared to baseline (TT MPO) **BM:** ↓ ~ − 1.3% for all subjects **Muscle glycogen**: ↑ 65% HCHO vs. ↔ LCHO (compared to baseline)
Training-overload studies (with or without an OTS/NFOR/FOR diagnosis) reporting no difference in EA or CHO availability (n = 3)						
Bellinger et al. 2020 [87]	n = 24 highly trained middle-distance runners (16M, 8F); VO_{2max} 73.3 ± 4.3 ml/kg/min (M) and 63.2 ± 3.4 ml/kg/min (F)	Longitudinal design with 3 weeks of increased training volume; retrospective division into functionally overreached (FOR; $n = 12$) and asymptomatic (AF; $n = 12$) groups	**Training intervention:** ↑ + 10%, + 20% and + 30% on weeks 1, 2 and 3, respectively, compared to baseline **Dietary intervention:** self-report food logs for 3 days at the end of the training block	**EI:** ↑ + 25% for FOR and ↑+ 7% for AF; FOR: ~ 2616 kcal/day vs AF: ~ 2577 kcal/day during intervention **EEE (estimated)**: not reported, but equal across groups and training monitored throughout. Estimated (via km*BM) as ~ 873 and ~ 832 kcal/day for FOR and AF, respectively **EA (estimated):** *assume ↔ between groups;* FOR: ~ 33 kcal/kg FFM/day vs. AF: ~ 30 kcal/kg FFM/day	**CHO intake:** ↔ *between groups;* FOR: 4.3 g/kg/day vs AF: 4.5 g/kg/day	**Performance:** ↓ TTE in FOR **BM:** ↔ for either group **FFM or FM:** ↔ for either group **Hormones:** ↔ for either group for cortisol, T3, T, IL-6, iron, DHEA, GH **Wellness:** ↓ FOR vs AF

Table 2 (continued)

Study	Participants	Study design	Training and dietary intervention	Energy intake (EI), expenditure (EEE), and energy availability (EA)	CHO intake	OTS and RED-S outcomes
Lehmann et al. 1992 [107]	$n = 17$ middle- and long-distance runners; VO_{2max} ~ 66 mL/kg/min	Parallel group design with 3 weeks of increased volume (IVT; $n = 8$) or intensity (ITI; $n = 9$)	**Training intervention:** IVT; ↑ + 200% compared to baseline; ITI; ↑ interval runs from 0.5% to 4.7% of all runs; speed runs from 5.9 to 10.7%. Note: ↓~ 50% volume in ITI vs IVT **Dietary intervention:** self-report throughout the study	**EI:** ↓ − 14% ITI: 3515 kcal/day or 48 kcal/kg/day vs. IVT::4110 kcal/day or 58 kcal/kg/day **EEE**: not reported, but likely ↓ ITI vs IVT (difference in training volume) **EA:** not reported *(cannot estimate due to lack of EEE data)*	**CHO intake:** ↓ − 21% ITI: 5.4 g/kg/day vs. IVT: 7.1 g/kg/day	**Performance:** ↑ ITI vs. ↔ IVT **BM:** ↓ − 1.4% in ITI vs. ↔ IVT **Mood**: ↓ in IVT vs. ↔ IVT **Catecholamines:** ↑ ~ + 200% urinary levels for dopamine, noradrenaline and adrenaline in IVT vs. ITI **Hormones:** ↑ cortisol and aldosterone in IVT; ↔ in ITI
Rämson et al. 2012 [82]	$n = 12$ highly trained international level male rowers; training experience 7.5 ± 2.2 years	Longitudinal design including a 2-week overload period	**Training intervention**: ↑ + 150–200% at weeks 1 and 2 of overload compared to habitual training volume (~ 10 h/week) **Dietary intervention:** Ad libitum. 3 days food diaries at the end of baseline and overload periods	**EI:** ↑ + 15%; 3981 vs. 4473 kcal/day baseline vs. overload **Total daily EE:** ↑ + 18%; 4136 vs. 4881 kcal/day baseline vs. overload **EA:** not reported *(cannot estimate due to lack of specific data, but assume ↔ due to near-identical ↑ in EI and TDEE)*	**CHO intake:** not reported	**Performance:** ↓ − 2.4% (2 h rowing ergo test) **BM and BF%:** ↔ **Hormones**: Leptin ↓ − 38%

cross-sectional study designs [100–102]. Although a one-off small 300 kcal deficit (either within day, or over an entire day) is most certainly not clinically impactful, when multiplied over months, these small mismatches in energy can become significant (e.g. energy deficit of 300 kcal/day over 1 year = 100,000^{+} kcal deficit).

3.2 CHO Availability Analysis

Four of the 21 studies [93–96] demonstrated an *independent* effect of CHO on OTS during isoenergetic conditions, and performance impairments were evident in all studies (relative to baseline or control condition). Here, CHO intake reached up to a 2-fold difference between groups (e.g. 4 vs 8 g/kg/day), which for a 65-kg athlete would translate into a difference of 260–325 g CHO/day. Further, all but one paper reporting a decrease in EI or EA in the previous section also reported a concomitant decrease in CHO (ranging from a difference of 1.4–6.0 g/kg/day, corresponding to a caloric difference of 364–1560 kcal/day for a 65-kg athlete). When considering the studies reporting a reduction in EA (Sect. 3.1), along with studies in this section, it appears that the reduction in EA from increased EEE is primarily driven via deficits in CHO intake, as observed in 14 out of 21 studies (Table 2) [19, 78–81, 84, 85, 90, 91, 93–96].

Indeed, poor chronic CHO availability, beyond or instead of LEA, is emerging as a potential mechanism also associated with some RED-S related outcomes (Table 1); although according to the current CAT would not strictly be diagnosed as RED-S. For example, it was recently shown that 3.5 weeks of extreme CHO restriction in a group of elite race walkers (a ketogenic LCHF diet; <50g CHO/day) impaired markers of bone remodeling despite adequate EA. Indeed, a control group implementing an isocaloric high CHO diet for the same time period experienced no change in bone markers [103]. Another recent investigation assessed the acute (< 24 h) effects of energy versus CHO availability on markers of bone remodeling in male participants [104]. In this study, it was shown that low CHO intake (3 g/kg/day) with either low (20 kcal/kg FFM/day) or adequate (60 kcal/kg FFM/day) EA led to a similar magnitude of increase in the marker of bone resorption as opposed to no change with a high energy, high CHO (60 kcal/kg FFM/day and 12 g/kg/day CHO) diet. Interestingly, the influence and impact of CHO availability has been shown in earlier research demonstrating that hormones associated with Triad outcomes, such as luteinizing hormone [56], T3 [105] and leptin [106], appear to be especially sensitive to changes in CHO availability. This energy-independent hormonal signaling role of CHO in the development of poor health outcomes is an intriguing finding and one that may exacerbate the LEA outcomes traditionally been linked to RED-S/Triad, but requires further research.

It is important to note that not all situations of training increases/overload result in decreased EA or CHO availability accompanied by RED-S related symptoms. Accordingly, three training-overload/OTS studies reporting impaired performance outcomes [82, 87, 107] and symptoms of OTS/REDS [82, 107] failed to support our hypothesis that increased EEE results in LEA and associated RED-S symptoms. Lehmann et al. [107] had 17 middle- and long-distance runners complete a 3-week overload protocol characterized by either increased training volume (ITV) or increased training intensity (ITI). Habitual dietary energy and CHO intakes were higher in ITV compared to ITI (58 vs 48 kcal/kg/day and 7.1 vs 5.4 g/kg/day, respectively). However, as training volume differed between groups (115–175 km for IVT and 63–85 km for ITI), we estimate EEE to have been lower in ITI and thus, EA at around 43 kcal/kg FFM/day for both groups. Rämson et al. [82] showed near-parallel increases in EI and TDEE during an intense training block (thereby assume unchanged EA), which was accompanied by impaired performance metrics. Both these studies failed to demonstrate clear differences in symptom outcomes between the study groups. Finally, a recent investigation has shown evidence for the role of muscle fiber typology (type I vs II fibers) in the development of FOR, without any indications of LEA [87]. In this 3-week investigation, the researchers showed that athletes with a higher gastrocnemius carnosine z-score (suggesting a higher proportion of type II fibers) were more likely to develop symptoms of FOR which were unrelated to indices of EA such as RMR, changes in body mass or composition, or blood hormone concentrations. No difference was shown in dietary intakes nor our estimations of EA between the two groups.

3.3 Methodological Challenges Associated with EA and CHO Availability Calculations

Collectively, we have many years of experience in the collection of EI and EA data, both in the laboratory and in the field, and while we acknowledge the difficulty in deriving accurate estimations of EA (Sect. 2.2), we also note that the main purpose of our analysis is to provide evidence for a notable difference in EA between two time points or groups (e.g. baseline vs fatigued; or OTS vs control athletes). Accordingly, within our analysis (Table 2) the potential confounding issues with EA and/or CHO availability calculations apply equally and randomly to all groups and time points. Nevertheless, 14 out of 21 studies demonstrated greater than 15% decrease in estimated EA between treatment groups or pre to post (with some studies showing as much as 100% difference). Taken together, 86% of the training-overload/OTS studies reporting dietary outcomes (18 out of 21) showed reduced EA ($n = 14$ studies) and/or CHO availability ($n = 4$) between treatment groups or between

pre and post concurrent with symptoms consistent with both OTS and RED-S (Table 1). Indeed, in absolute terms, the magnitudes of change or difference in EA between treatment groups in our analysis (Table 2) [19, 22, 78–81, 84, 85] averaged ~ 10 kcal/kg FFM/day (range 6–18 kcal/kg FFM/day difference) which is in line with significant symptom differences demonstrated with an EA difference of just ~ 7 kcal/kg FFM/day shown by Schaal et al. [19].

Another key real-world elite athlete consideration is an appreciation of the absolute EEEs implemented by elite/professional endurance athletes compared to typical study designs, as highlighted in Table 2. For example, training volumes can approach ~ 30 h/week in endurance sports such as swimming, cycling, and rowing [1–4]. This volume of training can easily project (depending on body mass, exercise intensity and mode of exercise) to at least ~ 20,000–35,000 kcal/week for just EEE (~ 2500–5000 kcal/day of EEE at ~ 500–1000 kcal/h). Supporting this, studies in elite athletes have shown TDEE of ~ 6000 kcal/day in Tour de France cyclists [108], ~ 9000 kcal/day in a 10h Ironman [109] and ~ 11,000 kcal/day in a 5 day ultra-running event [110]. Our lab has recently assessed EA in professional cyclists over 8 days (including several race days) where we reported a range from ~ 4000 to 6000 kcal/day for just the EEE portion of TDEE [111]. Conversely, several of the studies included in our analysis (Table 2) implemented an increase in training load protocol resulting in a daily EEE of only 725 kcal [79] to ~ 1212 kcal/day [19]. Overall, the mean (± SD) reported or estimated daily EEE of investigations summarized in Table 2 (across groups and time points) was just 1639 ± 714 kcal/day (range 725–2800 kcal/day). We propose that if the moderate increases in training loads/EEE reported in the current literature result in a mismatch between EEE and EI and various subsequent OTS/RED-S symptoms outcomes, the likelihood for these mismatches to manifest in the real-life elite sporting training environment is significantly greater.

4 Evidence of Inadequate EI with Large Increases in EEE: What Limits Sustained Extreme EEE's?

Most certainly, deliberate restriction from eating disorders (ED)/disordered eating can be an underlying cause of inadequate EI to sustain healthy EA, across a range of training loads. The reader interested in sport-related eating disorders is directed to these recent position statements [112, 113]. Nevertheless, we are unaware of studies examining NFOR/OTS prevalence rates, let alone the prevalence of eating disorders that would be diagnostically excluded in cases of NFOR/OTS. There is a higher prevalence of EDs in athletes than non-athletes [114] and a particularly high prevalence in aesthetic and weight class sports (i.e. sports favoring a high power to weight ratio [115]). Therefore, endurance sports might provide a risk for both symptoms of NFOR/OTS and prevalence of EDs; a hypothesis that requires scientific validation. Deliberate restriction or reduction of EI and/or CHO availability may be involved when manipulation of body composition or training adaptation (e.g. low CHO availability training [116, 117]) are desired outcomes of a training block. However, the phenomenon of an "inadvertent" mismatch between ad libitum EI and EEE is of most interest in situations of increased training loads/OTS.

Without a mismatch between EI and EEE, our hypothesis that many situations of training-overload resulting in symptoms of OTS/NFOR are a result of LEA would not be supported. Therefore, it is important to include an examination of the EI, satiety and exercise literature within our review. Overall, systematic reviews of studies of EI across populations with different physical activity levels, or changes in EI in response to the initiation of an exercise program, have found only a loose relationship between EI and physical activity, and little evidence of consistent increases in EI to compensate for increased EEE [118, 119]. Across these two systematic reviews, ~ 75% of the studies (n = 173 studies) demonstrate a lack of a compensatory increase in EI with an acute increase in EEE; this aligns with our analysis of 84% studies demonstrating either a clear decrease in EA (n = 14 studies) or CHO availability decrease (n = 4 studies) during the training-overload/OTS period (Sect. 3/Table 2). However, studies focused on the role that exercise has in determining appetite and EI suggest a lag of days or even weeks between an increase in EEE and adjustments to EI [120, 121]. Indeed, short-term appetite regulation following an acute bout of exercise appears to reduce appetite in an intensity-dependent manner, with increasing intensity leading to a greater suppression of orexigenic signals and greater stimulation of anorexigenic signals [122, 123]. There is also great variability in individual responses to changes in EI with exercise [118–123] and this is complicated in highly active individuals due to the effect of spontaneous physical activity or NEAT [124] as well as recent evidence demonstrating the excretion of energy in feces from non-digested food components [125]. To add further complication, emerging evidence from gut microbiota research has demonstrated dysbiosis (imbalances between pathogenic and symbiotic species; lack of diversity) potentially contributing to dysfunction between the gut–brain axis and the pathophysiological development of EDs [126]. This complexity if further compounded, as EI on its own may not represent fully bioavailable energy as healthy gut microbiota diversity appears required to fully assimilate and digest food (energy). It is also important to recognize that an individual's eating behaviour and EI is determined by a complex interaction of characteristics, including factors of non-biological origin.

For example, the Eating Motivation Survey [127] identifies 78 main motives which can be divided in 15 themes (such as: Habit, Need and Hunger, Convenience, Pleasure, etc., etc.) [128]. This illustrates the complexity of eating behaviours and encourages identification of sport- and/or individual-specific environmental and social/cultural factors that occur specific to the athlete's lifestyle. Here, the availability of food/drinks and the opportunity to consume them, coupled with a sport or training group's culture around eating, are key considerations in determining EI, particularly during the substantial proportion of an athlete's "waking hours" which involve preparing for, undertaking, or recovering from large volumes of training. Nevertheless, the topics present here are rife for further scientific investigation, as there is a paucity of similar data (impact of increased EEE on subsequent EI) and potential mechanisms in elite athletes, especially athletes with > 20 h week of training volume. A full account of the neurohumoral control of appetite and energy homeostasis is beyond the scope of the current paper; readers are referred to excellent summaries of various models and information on the complexity of orexigenic (appetite-stimulating) and anorexigenic (appetite-inhibiting) hormones [129–131].

The logistics of eating or drinking during exercise vary according to the exercise mode (e.g. rowing or swimming versus running or cycling), as some are more permissive for EI during training. The availability and palatability of nutrition is clearly important, while rules and sport-specific cultural traditions that promote eating/drinking within some sports assist the athlete to match their EI to high energy demands. By way of illustration, professional road cycling involves sports scientists/nutritionists and chefs within the team support personnel, team busses equipped with mobile kitchens, a "coms-linked" network of feed zones and team cars during races to supply riders with drink bottles and *musette* bags individually composed for riders with diverse tastes and texture to reduce "flavor fatigue". Cyclists even wear jerseys with pockets to carry foods, gels, and bottles and have bottle cages on their bikes. Furthermore, peloton traditions such as allowing periods within a race in which riders can eat without their competitors "attacking" and designating *domestique* riders to ferry food/drink supplies to the team leader also promote energy intake. Such features might explain the consistency of reports from stage races in which professional cyclists are observed to consume high mean daily intakes of energy [5600–6000 kcal (23–25 MJ)] and CHO [12–13 g/kg BM] to achieve a remarkably close match of TDEE, with impressive proportions of daily EA consumed while racing [108, 132, 133]. Obviously, whether these reports of EB found during racing can be matched during daily high-volume training situations in cycling and other sports, where athletes tend to be much less professionally supported, remain to be fully elucidated. However, despite this nutrition support, both training-overload/OTS and RED-S do occur in professional cycling, and without staff support, training-overload/OTS and RED-S is even more likely in recreational/national class athletes.

Food access and eating convenience vary for different sport settings. Professional sports teams are congregated many hours per day and supported via catered "training tables". In contrast, availability of suitable foods may limit EI in other sports or individual athletes, especially during solo-training situations. Indeed, food insecurity and limited finances are a challenge for many athletes, while traditional eating patterns, which include features such as reliance on food choices with low energy density or a limited number of eating occasions in a day, may also restrict total EI. Finally, there is speculation that there is a finite capacity of the gastrointestinal system to absorb and process foods, which sets an upper limit to sustained EI, and, as a by-product, sustainable EEE (from a health perspective). Although observations of sporting and recreational activities involving high rates of EEE in humans first suggested an upper ceiling equivalent to 4–5 × BMR over moderate time courses of several days to weeks [134, 135], the appreciation of a curvilinear relationship with event duration now suggests a chronic limit of approximately 2.5 × BMR [136]. It should be noted, surpassing and sustaining beyond 2.5 × BMR is theoretically routine in some elite endurance athletes, in that many accumulate 25–30h/week of training over extended periods [– 4, 8].

5 Conclusions and Future Directions

We have presented evidence to suggest that decreased EA and CHO availability may actually be a confounding factor in a significant number of training-overload/OTS studies (Table 2), resulting in misdiagnoses of training-overload/OTS, instead of LEA leading to RED-S. Fortunately, not all athletes who increase EEE manifest with OTS/NFOR and/or RED-S. Consequently, many athletes seem to, (un) consciously, be able to enhance the multi-factorial approach of recovery during sustained and intense periods of training/competition, including aspects of sleep [137–139], hydrotherapy [140], psycho-social-emotional factors [141–143], along with optimization of nutrition/hydration [144–147]. Many athletes appear to be able to periodize EI: increasing EI to meet large and sustained EEE during training phases and decreasing EI to meet body composition and performance goals at competition phases [12, 13]. Indeed, recent data from a single study suggest that full-blown OTS diagnoses are rare, as only 15% of an athletic endurance-based population who were already reporting under-performance and fatigue ($n = 100$) were diagnosed with OTS [39]. However, the overall prevalence rates (either seasonally or over a career and across many different types of sports and between

sexes) of OTS, NFOR or FOR in athletes are unknown and can also impact training and performance outcomes. Nevertheless, the prevalence rates of at least one RED-S component range from ~ 30 to 90% (depending on the sport, type and level of athlete, sex of the athlete, and diagnostic tool [148–151]). Our analysis showed that 84% (n = 18 studies; Table 2) of training-overload/OTS studies show indications of either LEA and/or low CHO availability with subsequent OTS/RED-S symptoms (n = 14 or 67% of papers; [19, 21, 22, 78, 83–86, 90, 91, 93, 95, 97, 98]) or impaired sports performance (n = 9 or 43% of papers; [21, 22, 78, 86, 93–97]). These findings are not surprising as they are in line with the RED-S prevalence rates in the literature. Indeed, we have highlighted the challenges of achieving accurate diagnoses of LEA/RED-S increases the potential for a failure to exclude RED-S within an NFOR/OTS diagnosis.

Accordingly, Fig. 3 is a hypothetical, but clinically relevant (*authors' observations*) OTS/RED-S framework that captures many of the concepts highlighted in this review. Indeed, and especially during the junior development phases, increases in training load (EEE) [1–4, 8] tends to initially result in improvements in performance, potentially through a combination of increased training adaptations and/or initial changes in body composition outcomes [9–11] (Fig. 3). This can, in some athletes and coaches, "feed-forward" the misconception that even more training and/or more decreases in extreme body composition metrics might further drive positive performance outcomes. This combination of pursuits (long-term large EEE and/or EI manipulation) creates a perfect storm for potential LEA, which can result in an ever increasing risk of adverse health and performance outcomes [148–151].

The prevention of either overtraining/under-fueling (OTS/RED-S) should be primarily based on awareness and monitoring. RED-S prognosis has been shown to be linked to duration of illness/dysfunction, and thus early recognition (awareness) and early intervention, when symptoms are still minor, is desired [77]. This is probably true of OTS as well. Nevertheless, RED-S/Triad awareness remains poor, as less than 50% of physicians, coaches, physiotherapists and athletic trainers were able to identify the three components of the Triad [152–154]. We are unaware of studies examining OTS awareness among athletes, coaches, and sport-practitioners. Nonetheless, as authors with decades of experience in the field, we observe that the conceptual awareness and appreciation of nutritional under-recovery/under-fueling (LEA) is low, many times resulting in the potential for misdiagnosis of OTS/NFOR, instead of RED-S. This poor awareness of RED-S is especially concerning given the data presented in this review demonstrating that the insufficient

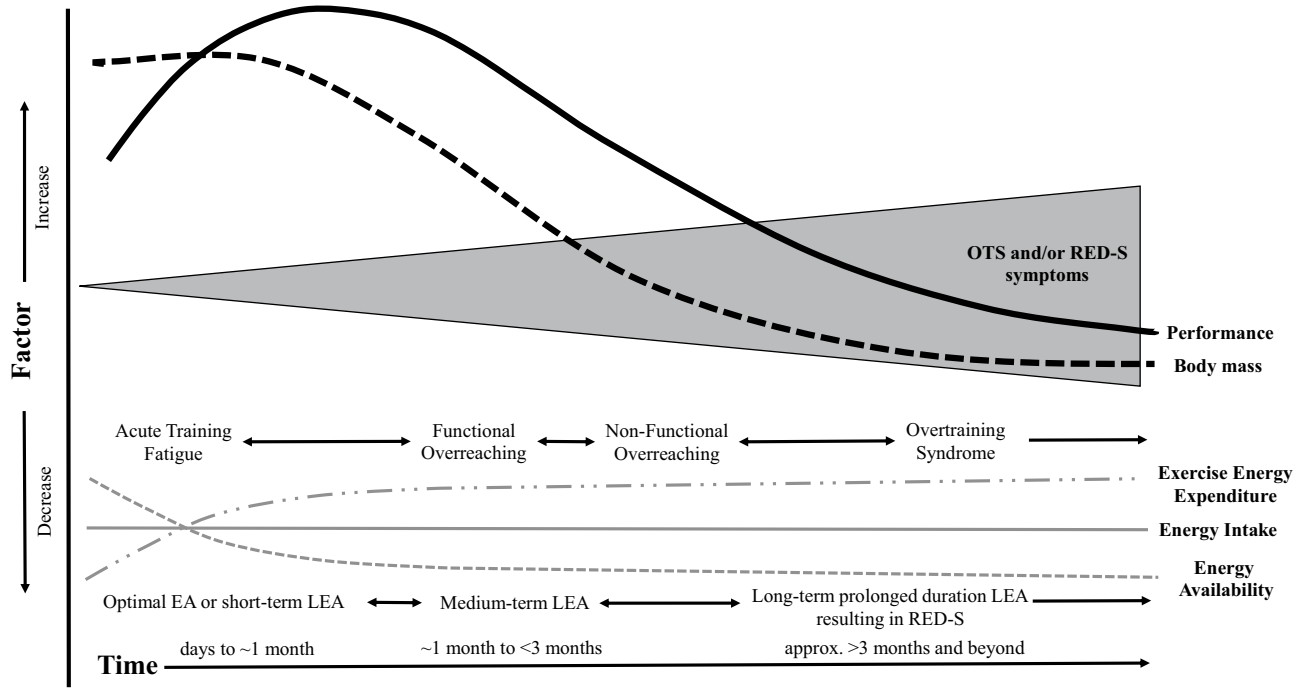

Fig. 3 An OTS/RED-S framework demonstrating the approximate time course of performance outcomes to OTS/RED-S symptoms linked to increased EEE with no appreciable change in EI resulting in low(er) EA, and possible drops in body composition/body mass metrics. LEA may contribute to many, but not all, FOR/NFOR/OTS symptoms. EEE, exercise energy expenditure; LEA, low energy availability; OTS, overtraining syndrome; RED-S, relative energy deficiency in sport

recovery underpinning many OTS/NFOR studies and clinical diagnoses may actually be primarily due to LEA and/or low CHO availability, and instead actually be RED-S. We hope this review creates more awareness of the close symptomatic parallels and diagnostic challenges for both OTS and RED-S. We have highlighted that in many instances the negative outcomes of training-overload (with, or without an OTS diagnosis) may primarily be due to under-fueling and thus actually RED-S, which could be avoided by emphasizing adequate nutrition support for the increased metabolic demands of exercise during periods of training overload.

Declarations

Funding No funding was received for this study.

Conflict of interest All authors declare that they have no conflict of interest as it relates to the material associated with this review.

Author contributions Development and formulation of concept and primary authors: TS and IH; Original draft authors: TS, IH, MM and LB; Critical feedback and revisions: RM, SB, SS. All authors have read and approved the final version.

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