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INVITED COMMENTARY

Low Energy Availability, Carbohydrate Intake, and Relative Energy Deficiency in Sport: The Low Triiodothyronine Hypothesis

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Background: Recent research findings suggest that a daily diet containing low carbohydrate (LCHO) consumption coupled with low energy availability (LEA, specifically what is termed problematic LEA [P-LEA]) exacerbates the risk of developing relative energy deficiency in sport (REDs). Regrettably, research evidence also indicates that dietary carbohydrates are likely underconsumed by many athletes in their daily diets. How these factors, P-LEA and LCHO intake, interact to precipitate the amplification of REDs risk is currently not entirely clear and is the source of much speculation. ***Purpose:*** As such, we present herein a hypothetical model of how LCHO dietary intake and P-LEA exposure can interact to create an amplification of the endocrine disruptions associated with REDs, specifically via the development of a low triiodothyronine (T_3) state, clinically referred to as low T_3 syndrome. The hypothesis presented postulates that P-LEA + LCHO interact to promote reductions in T_3 levels in part by inducing a greater cortisol response (at rest or exercise), which in turn inhibits the endocrine function involving the production of T_3, as well as the conversion of thyroxine to T_3. The resultant low T_3 state in turn amplifies the negative hormonal consequences associated with REDs (eg, reduced reproductive, anabolic, and metabolic hormone levels). ***Conclusions:*** Practically speaking, athletes and their coaches must recognize the importance of carbohydrates in the diet, specifically the amounts and the timing of their consumption; strive to avoid P-LEA exposure; and monitor for REDs indicators (eg, T_3). Researchers are encouraged to pursue investigations to challenge and evaluate our proposed hypothesis concerning how low T_3 is the critical factor in the negative hormonal consequences of REDs and the role cortisol plays in these outcomes.

Keywords*:* athlete health, sex hormones, energy expenditure, diet, carb shaming

Evidence supports that a state of persistent or severe low energy availability (LEA), which is termed *problematic* LEA, puts an athlete at significant risk for developing relative energy deficiency in sport (REDs) syndrome. This is a concern as the existence of REDs compromises both the health and the training/competitive performance capacity of an athlete.[1,2] Problematic LEA was introduced as a new concept in the 2023 International Olympic Committee (IOC) Medical Commission consensus statement and is defined as follows:

> An exposure to LEA that is associated with greater and potential disruption of various body systems, often presenting with signs and/or symptoms, and represents a maladaptation response. The characteristics of problematic LEA exposure (eg, duration, magnitude, frequency) may vary according to the body system and the individual. They may be further affected by interaction with moderating factors (eg, athlete behaviors, lifestyle, environment) that can amplify the disruption to health, well-being, and performance.[1]

Research indicates that problematic LEA (P-LEA) and REDs are associated with hormonal profile disturbances in both male and female athletes.[1] These disturbances are especially noted in the reproductive sex steroid hormones (reduced levels of testosterone [T], oestradiol-β-17 [E_2], and progesterone [P_4]) as well as several metabolic-anabolic hormones (eg, reduced insulin, leptin, triiodothyronine [T_3]; growth hormone; insulin-like growth factor-1; elevated cortisol).[1,3,4] The time course for these hormonal disruptions to manifest is highly variable but can occur within a matter of a few days (eg, T_3, T) or take up to several weeks (eg, E_2, P_4) and seems dependent in part on the severity/duration of P-LEA exposure as well as the sex of the athlete.[1,5] To a degree, these hormonal changes are proposed to be both indicators and causative factors in the detrimental consequences of REDs.[1,I]

Evidence suggests a daily diet involving low carbohydrate (LCHO) consumption and/or a short-term LEA state that reduces muscle glycogen availability, exacerbates the likelihood of P-LEA occurrence, and REDs risk development.[6,7] Researchers vary in how they define what constitutes LCHO consumption in athletes; however, herein, values of <3 g/kg/d or <30% daily caloric intake are utilized as an operational definition based on the literature.[8–10] Interestingly and regrettably, evidence supports that dietary carbohydrates (CHO) are commonly underconsumed by many athletes, especially females, both chronically and especially when experiencing a LEA state, increasing the risk for P-LEA/REDs.[1,11,12]

How these factors, P-LEA and LCHO consumption, interact to precipitate the amplification of the risk for REDs are currently not entirely clear and are a source of much scientific speculation. To this end, we present herein a physiological model to address this issue, proposing that a key mechanistic element for the increased REDs risk is the development of a persistent low T_3 hormone state (clinically referred to as the *Low T_3 Syndrome*). We hypothesize that the etiology for the low T_3 state centers on cortisol-induced thyroid hormone disruptions, resulting in a cascade of negative hormonal consequences found to be associated with REDs.

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Triiodothyronine (T_3) Role

Thyroid hormones play critical roles in the human body due to their direct actions on tissues as well as indirectly via their permissive actions in facilitating the functionality of other hormones. For example, the major hormones released by the thyroid gland, thyroxine (T_4) and T_3, can enhance the endocrine actions of growth hormone and epinephrine.[13] These latter hormones, along with T_4 to T_3, are critical for regulating aspects of the physiological response to exercise and in facilitating training adaptations.[13,14] T_3 is the more biologically active of the hormonal pair, and many endocrinologists actually consider T_4 a prohormone, as it can be converted to T_3 in peripheral tissues of the body.[13]

It is well established that caloric underconsumption leads to reduced thyroid hormone levels, with T_3 more severely affected.[15] Hence, LEA or P-LEA exposure can be a factor driving aspects of thyroid hormone disturbances found in athletes with REDs. Furthermore, typically with reduced caloric intake, the adrenal hormone cortisol can become elevated and, as a consequence, induce gluconeogenesis and proteolysis to provide additional energy substrates, for example, CHO.[16] Notably, cortisol, through inhibition of the mechanistic target of rapamycin system, also mitigates aspects of protein synthesis, allowing further proteolysis but, as a result, potentially compromising training adaptations.[17]

Additionally, relative to the thyroid hormones, elevated cortisol levels can attenuate the activity of the 5'deiodinase enzyme, resulting in a reduction in the conversion of T_4 to T_3 in peripheral tissues.[18] As a consequence of this inhibition, T_4 is converted to inactive metabolites, principally 3,3',5'-triiodothyronine (reverse T_3 [rT_3]), which as a hormone is not biologically active compared to T_3.[13] Furthermore, elevated cortisol can inhibit thyroid-stimulating hormone (TSH) from the anterior pituitary, also reducing T_4 and T_3 levels overall.[13] This type of inhibition, as well as down-regulation of T_4 to T_3 conversion, is an attempt by the body to conserve energy, which is medically referred to as the "Low T_3 Syndrome," also known as euthyroid sick syndrome (see reference for details concerning the clinical symptoms of this syndrome).[19]

Cortisol–Carbohydrate Interaction

Over 45 years ago, Professor Henrik Galbo (emeritus, University of Copenhagen) demonstrated that a low-carbohydrate diet (~10% daily caloric intake) augmented the cortisol response to a prolonged running bout.[20] More recent work found that as little as 3 days of low-carbohydrate intake (~30% of daily caloric intake [eucaloric diet]) in male endurance athletes, combined with intensive exercise training sessions, resulted in significant elevations in basal and exercise cortisol responses.[21] Such exercise cortisol responses are intensity dependent, and the more an exercise session exceeds 60% of maximal oxygen uptake, the more augmented cortisol responses become, especially in scenarios such as high-intensity interval training.[22,23] To this end, athletes at the elite, as well as nonelite levels, frequently engage in high-intensity activities, such as high-intensity interval training, as major components of their intensive periods in the training programs, making this elevation of cortisol potentially a common occurrence.[24]

Low T_3 and REDs

As noted,[1,2,4] the occurrence of P-LEA combined with LCHO consumption substantially elevates the basal and exercise cortisol responses in an athlete (*N.B.,* even within day energy deficiency [LEA], has been reported to evoke higher cortisol responses in female athletes in subsequent training sessions).[25] In our hypothesis, we postulate that these 2 factors, P-LEA + LCHO, interact to promote and amplify reductions in T_3 levels by inducing greater cortisol responses, and the subsequent effect creates a Low T_3 Syndrome-like state (see Figure 1). This state leads to further

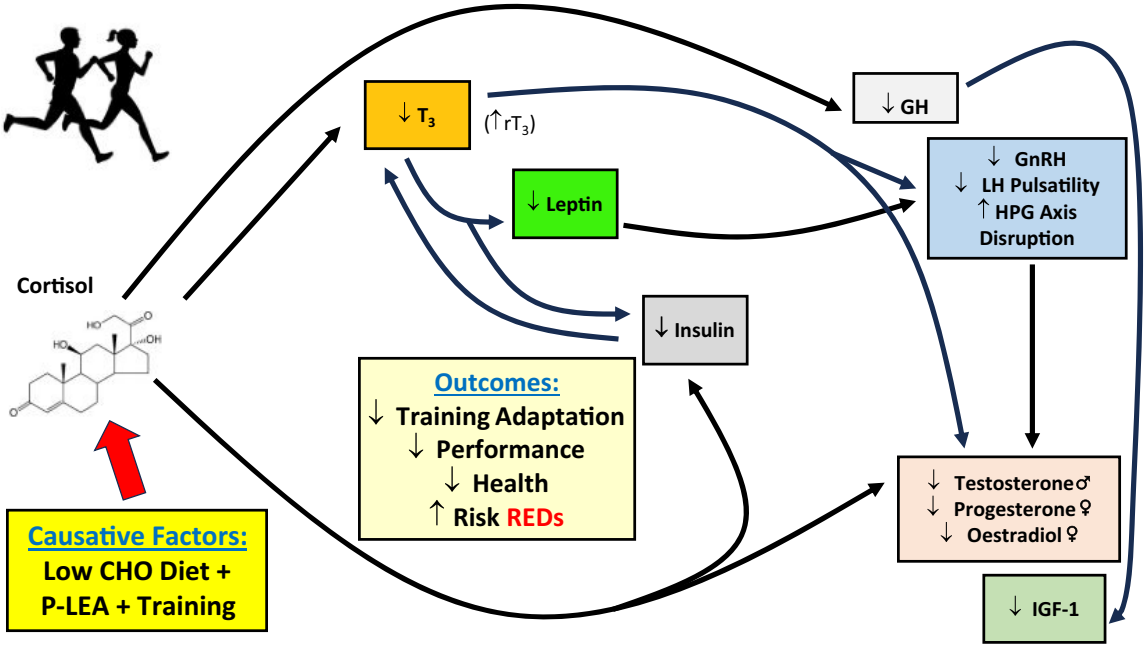

Figure 1 — Hypothetical model of how low energy availability plus low CHO intake creates an amplification effect on creating a hormonal profile associated with P-LEA and REDs. ♀ indicates female; ♂, male; CHO, carbohydrate; GH, growth hormone; GnRH, gonadotropin-releasing hormone; HPG, hypothalamic-pituitary-gonadal; IGF-1, insulin-like growth factor-1; LH, luteinizing hormone; P-LEA, problematic low energy availability; REDs, relative energy deficiency in sport; rT_3, reverse T3; T_3, triiodothyronine.

physiological-hormonal disruptions occurring in the athlete. Specifically, for example, T, E_2, and P_4 levels become compromised, as without adequate T_3, there is disruption of the hypothalamic-pituitary-gonadal regulatory axis controlling their production,[26,27] with evidence suggesting a greater disruption occurring in female athletes. Additionally, these key reproductive hormones can be further compromised via cortisol's direct inhibition of their production (ie, steroidogenesis inhibition) when elevated, as well as its similar inhibitory actions on leptin and insulin, critical hormones relative to metabolism and reproductive hormonal function/regulation (*N.B.,* the lowering of insulin further attenuates the conversion of T_4 to T_3 [resulting in greater reductions in T_3 levels][28] and lower insulin can be a function of the hormonal cascade depicted in Figure 1, as well as a LCHO or reduced caloric consumption).[4,16,29,30] Furthermore, while cortisol and growth hormone linkage are complicated, glucocorticoids such as cortisol can inhibit hypothalamic growth hormone-releasing hormone, thereby reducing circulating growth hormone levels, and such reductions subsequently lead to reduced insulin-like growth factor levels too.[31] These latter hormones are critical to the adaptation process to an athletic training program. Figure 1 is a summary illustrating our working model postulating how the development of low T_3 can have cascading ramifications and interact with a multitude of other endocrine hormones that relate to overall health, training adaptations, and sports performance.

There are additional physiological perturbations that can subsequently occur in the athlete due to these endocrine disruptions depicted in the figure. For example, the presence of low T_3 and T levels induces hematological consequences, specifically anemia. This occurs as the red bone marrow erythropoietic activity is compromised.[32] Additionally, a state of low T_3, T, E_2 can significantly affect muscle function, leading to muscle weakness/fatigue and reduced rate of protein synthesis, as well as a compromised immune system (*N.B.,* the latter is further exacerbated by reduced glucose availability in the LCHO state).[33,34] Notably, further aligned with low thyroid hormone effects are reductions in overall metabolic energy production, such as a lowered resting metabolic rate.[13] Also, an LCHO diet itself can directly lead to a lower resting metabolic rate, due to reduced energy expenditure from CHO metabolism, and can indirectly contribute to glycogen depletion and exercise fatigue development by limiting glycogen available as an energy source during physical activity, especially in high-intensity exercise situations.[2,12] Anemia, compromised muscle/immune function, and suppressed resting metabolic rate are frequent consequences found in both female and male athletes experiencing REDs.[1,2,4] Such consequences have a significant impact on an athlete's ability to train and perform or have appropriate adaptations to training (ie, leading to maladaptations).

To date, several critical review articles have summarized the endocrine profiles of female and male athletes suffering from REDs.[2–4] Our proposed hypothetical model and the discussions herein do not capture all facets of the hormonal changes as noted in these published findings (which may reflect the complex, individualized nature of REDs development).[5] But our proposed model does encapsulate the major ones frequently found in athletes experiencing P-LEA or manifesting REDs, as well as many of the physiological changes earmarked by the 2023 IOC Consensus statement as health and performance consequences of REDs.[1]

Practical Applications

Practically speaking, athletes must recognize the importance of CHO in their diets. The amount of CHO consumed is critical, as is the timing of consumption within daily fueling.[4,11,12] Regrettably, the fear of consuming too many CHO and the practice of "carb shaming" (also sometimes referred to as "carb fear"), due to concerns about body weight and body composition, exist in sports and are tangible problems for both males and females, though it is more prevalent in the latter.[35,36] Proper dietary practices to allow adequate energy intake ("fueling") involving all nutrients (especially CHO) are essential to decrease the risk of REDs developing. These and other lifestyle habits (eg, sleep, stress management) will greatly aid athletes in obtaining proper adaptations to their training and ultimately improve their performance (see reference for discussion of the influence of lifestyle factors in athletes).[37]

Conclusions

In conclusion, there are several primary take-home messages for athletes, coaches, and scientists concerned about P-LEA and REDs, and the interacting roles of dietary CHO and hormones:

- The combined impact of P-LEA and LCHO dietary intake (<3 g/kg/d or <30% daily caloric intake) can potentially create an amplification of the hormone profile disturbance associated with compromised adaptation, performance, and health in athletes and increase the risk of REDs.
- Monitoring athletes for the signs and symptoms of P-LEA–REDs development regularly (eg, during the different phases of the annual training program) is highly recommended (*N.B.*, T_3 assessment is one of the primary indicators recommended by the IOC to be examined in athletes). Specific steps should involve following the guidance of the IOC-developed clinical assessment tool (ie, REDS Cat 2)[1] to screen and assess the risk for REDs.
- Investigators and clinicians studying P-LEA and REDs should use a multidimensional approach to encompass hormonal measurements to facilitate a greater understanding of the endocrine interactions on adaptation/performance within athletes. That said, we highly recommend that any hormonal profiling involve the assessment of the thyroid hormone status of the athlete.

Finally, we acknowledge that the perspective we present here is a "working hypothesis" and, as such, we encourage researchers to pursue investigations to challenge and evaluate our proposed model concerning how low T_3 is a critical factor in the negative hormonal consequences of REDs and the role cortisol plays in these outcomes. Such work will either support or refute our hypothesis, which will ultimately allow more evidence-based insights to maintain and improve the health, well-being, and performance potential of athletes—the overarching goal for the sport sciences.

Notes

I. The reader is encouraged to review the IOC 2023 consensus statement for an extensive discussion on REDs,[1] specifically: (1) symptomology and outcomes, (2) the distinctions between LEA and P-LEA, and (3) how sport type influences the relative risk and prevalence of the syndrome.

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