# A life history perspective on athletes with low energy availability

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**Abstract**

The energy costs of athletic training can be substantial, and deficits arising from costs unmet by adequate energy intake, leading to a state of low energy availability, may adversely impact athlete health and performance. Life history theory is a branch of evolutionary theory which recognizes that the way the body uses energy - and responds to low energy availability - is an evolved trait. Energy is a finite resource that must be distributed throughout the body to simultaneously fuel all biological processes. When energy availability is low, insufficient energy may be available to equally support all processes. As energy used for one function cannot be used for others, energetic "trade-offs" will arise. Biological processes offering the greatest immediate survival value will be protected, even if this results in energy being diverted away from others, potentially leading to their downregulation. Athletes with low energy availability provide a useful model for anthropologists investigating the biological trade-offs that occur when energy is scarce, while the broader conceptual framework provided by life history theory may be useful to sport and exercise researchers who investigate the influence of low energy availability on athlete health and performance. The goals of this review are: 1) to describe the core tenets of life history theory; 2) consider trade-offs that might occur in athletes with low energy availability in the context of four broad biological areas: reproduction, somatic maintenance, growth and immunity; and 3) use this evolutionary perspective to consider potential directions for future research.

**Key Points:**

- Life history theory is a branch of evolutionary theory that describes how finite energy resources are distributed among competing biological processes. “Trade-offs” are predicted to arise because energy used to support one process cannot be used for others. Energy resources are allocated to tissues and functions in a hierarchical manner, with priority afforded to those that offer the greatest immediate survival value.
- Because energetic trade-offs among biological processes are predicted to be heightened under conditions of scarcity, a life history lens is particularly relevant for athletes who may have low energy availability due to high exercise training energy expenditure, inadequate energy intake (resulting from conscious or inadvertent restriction), or a combination of both.
- Energetic trade-offs are context specific and may be impacted by a range of factors, including sex, life-stage, training and health status, dietary composition and the length and severity of exposure to low energy availability.

48 **Table 1: Operational Definition of Terms** *(italicized in main text at first mention*)

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| Energy availability (EA) | Dietary energy intake minus energy expended in training, or the amount of energy available for biological processes after the demands of exercise training have been met [1,2]. Herein we refer to energy availability as "adequate" when sufficient to sustain usual functioning of all body systems, and "low" when energy availability cannot adequately sustain all systems. |
| Energy balance (EB) | Dietary energy intake minus total energy expenditure. An individual may be in energy balance while simultaneously experiencing low energy availability, as a downregulation of physiological systems can reduce total energy expenditure to match lower energy intakes [1,2]. |
| Inadequate energy intake | A dietary energy intake that is insufficient to meet the physiological requirements of the body. Inadequate intakes may be conscious (*e.g.,* intentional restricted intake to achieve a desired body composition), or inadvertent (*e.g.,* unintended undereating or alimentary limitation, whereby individuals cannot consume sufficient quantities of food to meet energetic demands of training). |
| Evolutionary anthropology/biology | Evolutionary anthropology refers to the study of human evolution and variation, including in relation to human physiology and behaviour [3]. Evolutionary biology investigates the biological basis of evolution across species. |
| Life history theory | A branch of evolutionary theory describing the competitive allocation of limited energy resources among physiological functions [4,5]. |
| Evolutionary fitness | The ability to successfully capture and use available resources to reproduce and pass genetic material to the next generation. |
| Phenotypic plasticity | The capacity to alter aspects of morphology, physiology, and behaviour in response to varying environmental circumstances. |
| Energy "trade-offs" | A reduction in energy allocation to one function as a result of energy being allocated to another function (considering that energy resources are finite). |
| Total daily energy expenditure (TDEE) | The total amount of energy expended in a day, which for adults will comprise basal metabolism, voluntary and involuntary activity, and the thermic effect of food. |

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## 1. Introduction

Meeting training demands with adequate energy intake can be challenging for many athletes, and evidence suggests that *low energy availability* (LEA; Table 1) is a common occurrence, with potentially negative consequences for health and performance [6–9]. These consequences have been conceptualized primarily in three models - the Female Athlete Triad [10], Relative Energy Deficiency in Sport (RED-S) [11], and the Exercise-Hypogonadal Male Condition [9] models. Despite ongoing investigation and important advances in knowledge, gaps in understanding of the physiology and pathophysiology of athletes with LEA remain, and these must be filled to progress screening, treatment, and return-to-play recommendations.

*Evolutionary biologists* recognize that the biological response to LEA is an evolved trait, and have long examined how finite energy is distributed among competing physiological processes in humans and other species within a theoretical framework called *life history theory* [4,5,12–15]. Classically used to understand species-level differences in the pattern and timing of important biological events (*e.g.,* maturation, reproduction, and death), life history theory's conceptual framework has increased understanding of which physiological functions are prioritized or sacrificed in humans under varying environmental circumstances, including energetic stressors such as LEA. This has broad implications for health in human populations and may hold specific relevance for athletes, many of whom struggle to achieve adequate energy availability due to high training energy expenditure and/or *inadequate energy intake* (which may occur via conscious restriction, reaching alimentary limits [16], or due to inadvertent under-eating [8,17]). Among athletes, those with particularly strenuous training schedules may incur extreme energetic costs that can increase their susceptibility to LEA. Many evolutionary anthropologists use exercise, sport and physical activity to investigate and inform theories of human evolution and adaptation [18–22]. Similarly, sport and exercise scientists may find the overarching conceptual framework of life history theory useful to consider the biological basis and variability of the human body's response to LEA. The aim of this review, therefore, is 1) to describe the core tenets of life history theory; 2) consider trade-offs that might occur in athletes with LEA in the context of four broad biological areas that are commonly considered within the life history literature, *i.e.,* reproduction, somatic maintenance, growth and immunity; and 3) use this evolutionary perspective to consider potential directions for future research.

## 2. Brief Background to Evolutionary Life History Theory:

The notion of 'competition' features prominently in Charles Darwin's theory of evolution by natural selection [23]. Darwin and his contemporary, Alfred Russel Wallace, described competition amongst individuals for the resources needed to successfully pass genetic material to subsequent generations - this constitutes '*fitness*' in evolutionary terms [24]. Life history theory, a branch of evolutionary theory, also predicts competition at an intra-individual level. This theory considers energy to be a finite resource that is competitively allocated between four principal areas: reproduction, growth, somatic maintenance, and immune defence [25–31]. Energy can also be used for physical activity, or stored in adipose tissue, or as liver or muscle glycogen or triglycerides. A key tenet of life history theory is that energy used for one function cannot be used for others, *i.e.*, energy resources are "*traded off*" among competing functions. Notably, competition among functions will be heightened during conditions of scarcity [30,32], as may be the case for athletes with LEA.

Like all aspects of human biology, the physiological mechanisms responsible for energy allocation among competing functions were shaped by humans' evolutionary journey. The evolutionary history of our species involved repeated cycles of colonization and dispersal, characterized by periods of food insecurity and other ecological challenges [33–35]. In the language of evolutionary biology, such challenges were 'selective pressures' that shaped the development of adaptive mechanisms to successfully survive periods of biological stress, including that caused by LEA. One such adaptive mechanism allows for an internal reallocation of energy during times of scarcity toward physiological processes providing the greatest immediate survival value. This capacity to dynamically redistribute energy resources derives from *phenotypic plasticity* [34,36,37], whereby morphological, physiological, and behavioural traits vary in response to changing environmental circumstances and stressors without alterations to the genetic code. Resultant trade-offs can occur (or be reversed if circumstances change) at several levels, and on a range of timescales. This could be considered at a behavioural level, where changes can take place immediately. It could also be considered at a cellular level with, for example, glycogen depletion resulting in a reduced capacity for performance over minutes or hours. At the organ level, deficits in, for example, bone mass or microarchitecture may accrue over months or years. Underlying all of these scenarios is the imperative to allocate available energy in the manner most likely to improve survival and reproductive success.

A useful analogy commonly employed in the life history literature considers the parallel between energy availability and financial budgets [18,38]. When an individual earns (or consumes) more than they spend (or expend), all expenses are paid and excess income is deposited in a savings account (*e.g.,* fat tissue). In situations where expenditure exceeds income, however (*e.g.,* if wages are cut, or expenses increase), funds must be allocated to the most immediately essential expenses, even if this comes at the cost of something else. Purchasing food, for example, may be traded off against purchasing clothing. Importantly, such trade-offs are context specific. If an individual already has adequate clothing, it makes little sense to use limited resources to buy more, particularly if this means going without food. In a cold climate with a well-stocked cupboard, however, the decision may be reversed. Alternatively, small savings can be made by marginally reducing expenditure in multiple areas, while short term deficits can be temporarily covered by withdrawing funds from one's savings account (although this strategy is not sustainable in the long-term). In a comparable way, athletes whose training load or dietary practices leave them with insufficient energy to cover the costs of all biological processes must allocate energy to certain processes at the expense of others (see Figure 1). We discuss this scenario in the following sections in the context of 4 broad categories commonly considered within the life history literature: reproduction, somatic maintenance, growth and immune defence [25–31].

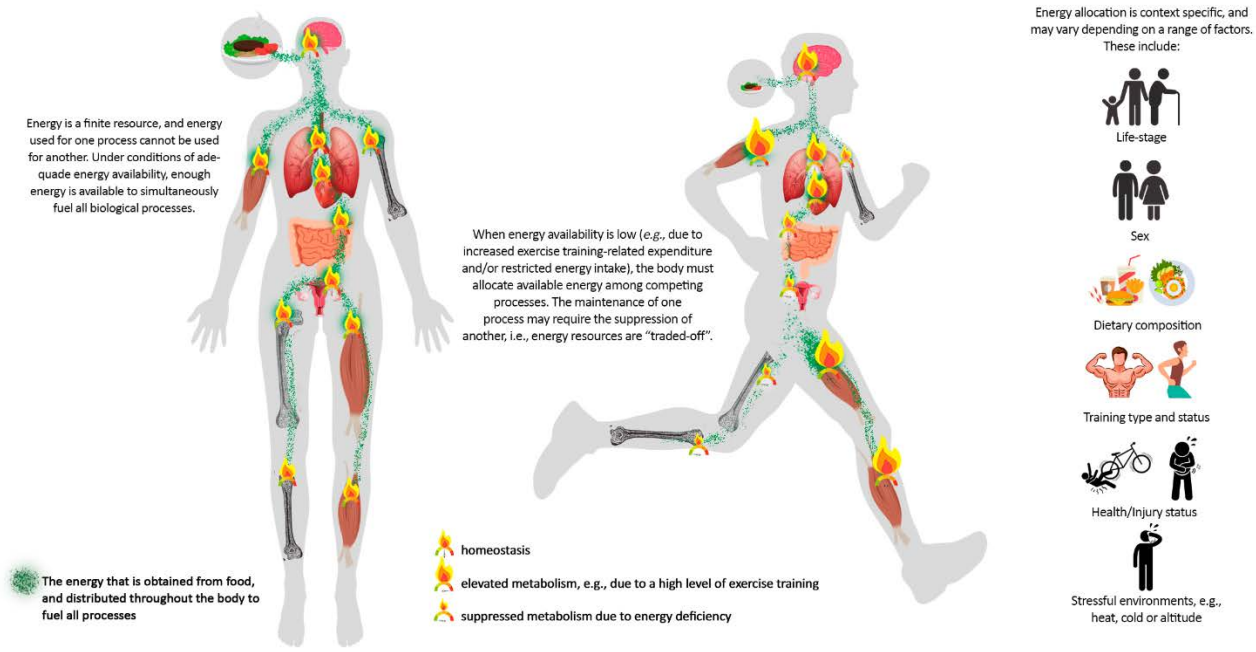


***Figure 1****. Schematic depicting how low energy availability - whether arising via increased training expenditure, inadequate intake, or both - requires the body to competitively allocate available energy among various biological processes. Some processes will maintain their energy supply at the expense of others.*

## 3. Reproduction

Life history theory posits that biological trade-offs at all stages of the life cycle ultimately serve to maximize reproductive success, a term often used synonymously with evolutionary fitness [39–41]. Producing offspring that go on to successfully reproduce themselves is the currency of evolutionary fitness. Energy allocation around reproduction largely depends upon the relative pace of a species' life history, *i.e.,* its full lifespan, from conception to death. Species with "faster" life histories generally prioritize current reproduction over somatic maintenance or future reproduction. An example is the fruit fly, which may respond to environmental threats by increasing mating effort to pass on its genetic material before death [42]. In contrast, larger, longer-lived mammals, who have a "slower" life history pace typically implement a very different strategy in the face of environmental challenges like LEA or pathogen exposure. Namely, environmental stressors that signal a short-lived fruit fly to reproduce now may signal a larger, longer-lived mammal (*e.g.,* humans) to downregulate reproduction and wait for more favourable conditions.

Reproductive downregulation is a well-established consequence of LEA in male and female athletes [9,43]. For example, downregulation of the hypothalamic-pituitary-ovarian axis in exercising women can lead to cessation of menses, or functional hypothalamic amenorrhea [10,44,45]. This represents a diversion of available energy away from reproduction towards other functions and is often discussed in pathological terms. It is true that menstrual disruption can serve to warn that energy demands of exercise training are not being met, and that prolonged exposure to LEA may have clinical consequences (*e.g.,* for cardiovascular or bone health [46,47]). However, an evolutionary perspective would not consider ovarian suppression to be a pathological condition *per se*. Instead,

interruption of the menstrual cycle can be viewed as a "*perfectly normal adaptive response to the prevailing environmental conditions when animals are challenged energetically*" [48]. Energy diverted away from current reproduction can be made available to other processes, while simultaneously conserving energy for future reproduction when energetic conditions improve [39,49]. This strategy makes sense in light of the high energetic costs of human female reproduction, which, in addition to pregnancy and lactation [50,51], also include caring for offspring [39,49,52]. Indeed, undertaking such an energetically expensive process in a state of LEA could jeopardize both mother and offspring health, and this appears to be a risk that humans have evolved to avoid. Functional hypothalamic amenorrhea in athletes appears to be a reversible phenomenon, with evidence suggesting affected athletes resume cycling once adequate energy is available [53–55]. A similar situation has been observed in a non-athletic context in response to seasonal fluctuations in workload-associated energy expenditure and food availability [49,56–58]. Little is currently known, however, about the longer-term reproductive consequences of transient periods of reproductive suppression in female athletes, nor how different lengths and extremes of LEA may impact subsequent reproductive capacity. Theoretically, the resumption of menses after a period of LEA-related amenorrhea could be hypothesized to impact subsequent fecundity positively, negatively, or not at all, but it appears this question is currently unaddressed in the literature.

Although much of the sport and exercise science literature focuses on the influence of LEA on the reproductive status of female athletes, it is important to highlight that men are also affected [9,43,59,60]. In addition to gamete production (which has minimal energetic costs), male reproduction also incurs costs related to child-care, attracting a mate and fending off male competitors, and reproductive ecologists have pointed to the modulation of physiological correlates of male mating effort (*e.g.,* testosterone, muscle mass) during times of energetic stress [39]. Evidence indicates that LEA can lead to downregulation of the hypothalamic-pituitary-testicular axis in exercising men [9,61]. Sexual dimorphism does, however, seem to exist in relation to trade-offs made under conditions of LEA [39,43,48,62,63]. This is unsurprising, given that the cost of reproduction is substantially greater for women than men [39,49,50], meaning that women in a state of LEA have a stronger biological imperative to weigh the benefits of reproductive investment against its energetic cost.

Perspectives differ on how indicators of energy status may predict or influence reproductive status. Reproductive ecologists postulate that *total daily energy expenditure* (sometimes referred to as "flux") and reproductive success have a curvilinear relationship, with both very low and very high energy turnover negatively impacting reproductive success. For example, research conducted among rural Polish and Nepali women reported reproductive downregulation during more work-intensive seasons (*e.g.,* the monsoon season), despite comparable food availability [56,58]. These observations may potentially be explained by the "constrained downregulation" hypothesis, which posits that high total daily energy expenditure may render a woman less able to downregulate her metabolism, thus constraining her ability to meet the energetic demands of reproduction and lactation. Ovarian function may, therefore, be suppressed until energetic conditions change [49,58]. This is consistent with the broader constrained energy model proposed by Herman Pontzer and colleagues, which holds that human energy expenditure is constrained within physiological limits [64,65]. This model predicts that an excess of energy spent in one area (*e.g.,* training related energy expenditure) may result in a compensatory downregulation of another (*e.g.,* reproduction, or somatic maintenance) even when energy intake is adequate. Both the constrained downregulation hypothesis and the constrained energy model build upon life history theory, but interestingly, they

do not align well with the sport and exercise science literature. In the latter, high energy expenditure is only considered to become problematic when not met by adequate energy intake [45,66]. From this perspective, energy availability – rather than energy intake or expenditure *per se* – is considered the key regulator of reproductive function [45]. Ongoing investigation of the influence of LEA on athletic reproductive health, along with the factors that may influence this, is undoubtedly warranted.

## 4. Somatic Maintenance

Somatic maintenance refers to "*preserving the body in good condition through diverse homeostatic processes, and thus promotes longevity and future opportunities for reproduction*" [29]. In conditions of LEA, certain essential maintenance processes cannot be traded off, lest survival be threatened. Other processes, however, may be temporarily downregulated without (or with fewer) negative consequences, and thus represent areas from which energy can be diverted when needed elsewhere [67]. More severe and/or prolonged periods of energy deficiency will decrease the flexibility of these energetic trade-offs, with potentially greater detriments to the body. This is particularly relevant for athletes, as undertaking, recovering from, and adapting to physical training are costly processes that may potentially result in higher somatic maintenance costs and different trade-offs than those experienced by the general population. In the following sections we consider from a life history perspective how LEA might impact the somatic maintenance of large body tissues and physical performance.

### *a. Bone*

A substantial body of research exists regarding the bone response to LEA, and this has been reviewed in detail elsewhere [68–70]. Briefly, cross-sectional and observational studies of athletes indicate that prolonged exposure to LEA manifests in lower bone mass, compromised bone micro-architecture, perturbed bone metabolism and lower estimated bone strength [71–74]. Perturbations in bone biomarkers have also been reported in individuals experimentally exposed to acute periods of LEA (*i.e.,* 3 – 5 days). This initially appears to manifest as a reduction in markers of bone formation [63,75], thus aligning with the prediction that energy will be diverted away from non-essential but energetically expensive metabolic processes during periods of LEA. Importantly, indicators of LEA are also associated with increased stress fracture risk [76–78], which has important practical and clinical implications for athlete health and performance.

When considered in the context of life history theory, diverting limited energy away from bone during periods of LEA could be considered a prudent strategy to preserve essential functions elsewhere, given that bone metabolism may be considered a temporarily "expendable" process [67]. Indeed, the "disposable soma" theory – an explanation of why we age that is closely related to life history theory - predicts that selection favours energetic investment in reproduction at the *expense* of longer-term bodily maintenance [79], which is likely to include the maintenance of bone tissue [80]. Bone mineral composition and micro-architecture are relatively stable in the short-term and measurable changes can take months or even years to develop, with bone-related disorders like osteoporosis primarily impacting older adults. Consequences of diverting energy away from bone metabolism

during periods of LEA may therefore not be felt until after the reproductive lifespan of the individual, and this could explain its apparent susceptibility to being traded off when energy resources are limited.

A recent review suggested that LEA-associated perturbations to bone metabolism could be offset by high-impact exercise [69]. The authors of this review posited that the benefits of high-impact exercise on bone may occur *despite* the negative influence of LEA, thus representing a potential countermeasure. A non-mutually exclusive, life history-oriented explanation is that the increased stress of high-impact loading on the skeleton may increase the necessity of skeletal preservation; potentially shifting bone higher up the hierarchy of preservation, thus rendering the tissue less likely to have resources diverted from it to other areas. Although there is a theoretical justification for a protective effect of high-impact exercise on bone during times of LEA, controlled trials are required to test the efficacy of this approach. Indeed, recent observational evidence indicates that the incidence of bone stress injury is higher in female distance runners with menstrual disturbances (assumed to be due to LEA) but that plyometric training did not influence this [81]. It is also pertinent to consider if mitigation strategies intended to protect against certain trade-offs might potentially lead to downstream trade-offs with unforeseen consequences.

*b. Muscle and Fat Mass:*

Muscle is the primary tissue driving human movement. It is therefore a critical determinant of athletic performance, in addition to human health [82]. Given this, one might expect muscle to maintain its energy supply during conditions of LEA. Muscle is, however, a metabolically expensive tissue accounting for approximately 20% of resting energy expenditure in adult humans [83], and it incurs an even higher metabolic cost during physical activity. Substantial energy savings could, therefore, be made by reducing muscle mass when resources are scarce [9,84,85]. In addition, muscle provides an important reservoir of endogenous amino acids that can be liberated during periods of scarcity and used elsewhere, for example, to support enzyme and immune factor synthesis and maintenance of plasma protein concentrations [86]. This diversity in essential physiological functions renders muscle tissue responsive to a wide range of stimuli. The response of muscle to LEA may depend on which of its functions (*e.g.,* mechanical, regulatory or storage) proves to be most immediately essential. This in turn may depend on a host of factors, including sex, life-stage, health and training status, body composition phenotype, the length and severity of LEA, and the macro and micronutrient composition of available energy, as discussed elsewhere [87]. For example, biological sex is thought to influence the muscle response to periods of LEA, with men potentially more susceptible than women to muscle-loss during times of scarcity [88–90]. The proportion of muscle mass loss increases exponentially with decreasing body fat content [91]. Women have, on average, a larger "energy buffer" in the form of greater body fat stores than men [88,89,92]. This likely explains, at least in part, the sexually dimorphic response of muscle and fat to LEA. To the best of our knowledge, however, the relative loss of muscle and fat has not been directly tested in male or female athletes with LEA, some of whom may already be at, or close to, their lower biological adiposity limits. It is feasible that the high training status and baseline body composition of athletes may influence muscle-related trade-offs under conditions of LEA, but this requires further investigation.

Experimental evidence suggests that the muscle response to LEA is susceptible to manipulation by factors such as training or protein availability. Muscle mass is ultimately maintained, lost, or increased based on a dynamic balance between muscle protein synthesis and breakdown. In common with data on bone biomarkers, controlled laboratory studies indicate that short-term exposure to LEA typically manifests in a reduced rate of muscle protein synthesis [93–96]. This suggests that a portion of the energy typically allocated to building or maintaining muscle may be traded-off when resources are scarce. It appears that this can be prevented, or at least attenuated, by anabolic stimuli such as resistance exercise or amino acid availability, including the muscle protein synthesis-"triggering" amino acid leucine [93–95,97]. It has also been reported that intensive resistance training in conjunction with a high protein intake can stimulate muscle mass gains with simultaneous fat loss, despite individuals being experimentally exposed to an energy deficit of approximately 40% below estimated requirements [98]. This suggests that in certain situations the negative consequences of certain biological trade-offs may be susceptible to mitigation, given the right strategy. At the same time, as noted above regarding bone, it is important to consider whether such mitigation strategies could potentially lead to downstream trade-offs with unforeseen consequences, while the potential for the energy cost of resistance training to exacerbate the extent of LEA must also be considered.

*c. Physical performance:*

From an evolutionary perspective, physical activity during periods of energetic stress would have been essential to our ancestors' survival by allowing them to hunt and gather the food required to restore adequate energy availability. Locomotion and physical activity have, however, been described as potentially reducible processes when energy availability is low [67], and substantial energy savings could be made by reducing non-essential activity-related expenditure. Convincing theoretical arguments could be made for either a positive or negative influence of LEA on exercise performance, with the actual response likely to depend upon factors including the type of activity, the training and nutritional status of the individual and the extent and duration of deficit. Intuitively, it seems likely that LEA may negatively impact exercise performance, given the importance of fuelling (both in terms of energy and individual nutrients) to optimise performance. On the other hand, and under certain circumstances, it seems plausible that performance could be maintained, or even improved, in the face of LEA. For example, a review of the influence of eating disorders on sporting performance suggested that shorter-term energy restrictions may induce transitory performance improvements [99]. This could be due to increased stress hormones like cortisol, which may have short-term stimulatory effects [100], or to increased power-mass ratios. A transitory increase in performance capacity in response to LEA could make sense from a survival perspective if it facilitates the catching or finding of needed calories. It has also been suggested that transient exposure to LEA within a periodized training and nutritional program may be desirable to drive certain phenotypic adaptations, such as body composition changes or increased efficiency of specific bioenergetic pathways [101].

Available intervention data directly investigating how LEA may impact exercise performance are conflicting, with both positive, neutral and negative effects shown. For example, Oliver et al. [102] reported that 48 hours of energy restriction reduced 30-minute treadmill time-trial performance [102], whereas Kimojo et al. [103] reported no influence of 3 days of LEA on time to exhaustion while running at 90% of $VO_2$ max, despite reduced muscle

297 glycogen availability. In contrast, 4 weeks of energy restriction with protected protein intake led to improved
298 countermovement jump height and 20-m sprint time in a group of male track and field jumpers and sprinters. At
299 the same time, rapid, or gradual, weight-cutting (2.4 hours versus 3 weeks) did not impact 30m sprint or 1-minute
300 Wingate performance or increased loaded vertical jump height [104] in a group of wrestlers and judo athletes.
301 The type of exercise test may have been influential in these outcomes, and, plausibly, more explosive
302 power/speed-type activities may be less susceptible to the influence of LEA than more prolonged endurance-type
303 activities, although this hypothesis requires empirical testing. In contrast to this hypothesis, some evidence
304 indicates that LEA may lead to an increase in exercise energy efficiency in aerobic activities [105,106]. For
305 example, an observational study by Tornberg et al. [106] reported increased energy efficiency in a group of
306 amenorrheic female runners, compared to their eumenorrheic counterparts, as evidenced by a lower caloric cost
307 of pedalling on a cycle ergometer at 50 and 100 Watts.

308 The duration of exposure to LEA is likely to be an important determinant of its influence on exercise performance,
309 particularly if it leads to consequences such as the suppression of reproductive hormones, muscle loss, or increased
310 stress fracture risk. For example, observational studies have indicated that ovarian suppression as determined by
311 states of oligo or amenorrhea may be detrimental to performance outcomes including knee muscle
312 strength/endurance [106], 400m swim velocity [107] and training volume [108]. Athletes who increased their
313 energy intake in response to increased training intensity had a superior performance on a running test than their
314 counterparts who did not increase their energy intake to match the extra energy expended in training [109].
315 Collectively, these findings suggest that chronic LEA should be avoided to protect performance, although it is
316 important to highlight that these are observational findings and that causality between LEA and exercise
317 performance is as yet unconfirmed. Substantial further investigation is required to further elucidate how varying
318 lengths and severity of LEA may impact exercise performance using a range of test protocols.

319

320 **5. Growth and Immune Defence**

321 The life history literature classically considers both growth and immune defence as important areas between which
322 finite energy must be distributed [25–31]. Relatively little evidence is available, however, regarding the influence
323 of LEA on growth in young athletes, or on how LEA impacts the ability to mount an immune defence in athletes
324 of all ages. For this reason, we offer a short, combined discussion of these parameters in the hope of stimulating
325 ideas for future research. With respect to growth, this may be threatened in young, still-growing athletes if energy
326 availability is insufficient to meet the simultaneous demands of training, in addition to other essential maintenance
327 functions, and growth. Research performed in rats and rabbits indicated negative impacts of nutritional deprivation
328 on bone epiphyseal plates [110], with implications for longitudinal bone growth. Potential adverse effects of LEA
329 on growth may be particularly relevant in sports that simultaneously emphasize early specialization and leanness
330 and/or lightness (*e.g.,* dancers, gymnastics, and horse-racing jockeys), given that athletes may be required to
331 maintain low body mass at pivotal developmental stages. Some evidence indicates that growth in young female
332 gymnasts is impacted by intensive training, with catch-up growth potentially occurring when training intensity is
333 reduced [111] (although differing perspectives on this topic do exist [112]). It seems likely that intensive training

will adversely affect the growth of young athletes only if the energetic demands of training are not met, thus leading to LEA, however this arguably requires further investigation according to age, sex and sport.

With respect to the immune system, energetic investment in maintenance and function is critical for survival, reproduction, and evolutionary fitness [26–28], but it is also energetically costly, "*particularly for those at the margins of nutritional adequacy*" [28]. This was recently demonstrated by Urlacher and colleagues, who found that among young Shuar forager-horticulturalists in Amazonian Ecuador, heightened immune activity alongside increased physical activity (compared to children from industrialized populations [113]) resulted in constraints on growth [114]. Interestingly, children with larger body fat levels were less susceptible to the growth-inhibiting effects of heightened immune activity, which was attributed to the greater energy reserve that higher body fat levels convey. Investigating the direct influence of LEA on immune function in athletes is challenging, given that high levels of training and competition may also influence the immune response, rendering it difficult to separate the effects of training and competition from those attributable to energy availability *per se* [65,115–117]. Because adequate energy from key fuel sources (*i.e.*, carbohydrates, amino acids and fatty acids) underlies the immune system's ability to resist infection by various pathogens [116], it is intuitive that LEA might adversely affect immune response. Consistent with this, several studies have reported increased illness [118–120] and/or alterations to immune biomarkers [118,121] in athletes who were believed to have LEA at the time of data collection, in comparison to their energy-replete counterparts. Importantly, these observations are based on cross-sectional investigations, implying a more chronic adaptation to LEA (although of course causality cannot be confirmed from an observational study). In contrast, a recent study investigating potential trade-offs in response to the acute energetic stress of a 160km ultramarathon reported evidence for increased investment in innate immune function (indicated by increased bacterial killing ability and haemolytic complement activity) over reproductive effort (proxied by measures of testosterone and arousal) [122]. In common with many studies in this area, however, it is difficult to differentiate between LEA, versus the stress of the exercise event itself (or some other factor) as the causative agent. It is also interesting to consider that other populations with extreme LEA, *e.g.,* individuals with anorexia, are considered to be relatively robust to infection [123,124], implying that certain aspects of immunity (*e.g.,* innate versus acquired) may be more robust to trade-offs. Substantial investigation is required in this area to better understand whether and to what extent different aspects of immune function are traded-off when available energy is low.

## 6. Perspectives for Future Research

We believe that considering LEA in athletes from a life history perspective has substantial potential to inform research on this topic, both by providing an alternative viewpoint on existing data, while also allowing for discussion and hypothesis-generation to guide future research efforts. A core tenet of life history theory is that differential energy allocation will preserve – or sacrifice – different functions in different contexts, meaning that physiological processes will not be equally or simultaneously affected. A more holistic, whole-organism perspective that considers how LEA may impact specific processes *and* mediate trade-offs among other essential functions is preferable to a reductionist approach that considers functions in isolation. Additionally, a life history perspective emphasizes that LEA may differentially influence outcomes of interest (*e.g.,* bone metabolism or

immune function) based upon a myriad of factors, such as the life-stage of the individual, their broader nutritional status (*e.g.,* macro and micronutrient adequacy) and the type and intensity of their training schedule. Although undoubtedly challenging to adequately control and standardize for all relevant variables, researchers must remain cognizant of potential mediators and confounders and consider these in study design and interpretation.

Currently, limited information is available regarding the extent or duration of LEA that will provoke deleterious outcomes under different circumstances and this is an important avenue for ongoing research. Additionally, it is important to consider that LEA is extremely challenging to accurately measure [125], while varying perspectives exist related to factors such as the timeframe within which potential deficits should be considered. For example, some suggest that 3- or 7-day rolling averages are more useful, while others indicate that greater within-day imbalances may have increased consequences over similar intakes consumed more frequently throughout the day [126]. Validation of differing approaches to outcome assessment and consideration of how varying characteristics of LEA may impact health and related outcomes is an important direction for future research. This could include, for example, investigations of larger deficits across shorter time-periods versus smaller deficits across longer time periods, as well as imbalances arising primarily from higher training related expenditures versus more restricted energy intakes. It is also important to consider that a life history perspective considers the transient suppression of non-immediately essential physiological processes to be a normal, and necessary, adaptive response to conditions of energetic stress. As such, conclusions about the influence of LEA on health or performance related outcomes should only be made based on outcomes that directly relate to health or performance. Unsubstantiated assumptions that all trade-offs are negative should be avoided, as should over-extrapolation of mechanistic findings. Indeed, there may be situations where transient exposure to LEA within a periodized nutritional cycle may be desirable to drive certain phenotypic outcomes [101], while more moderate energy restriction throughout the life-course is hypothesized to increase longevity and health-span [127]. The topic of LEA in athletes is both complex and nuanced, and embracing these characteristics of the research topic may be key to the development of appropriate guidelines to support at-risk athletes.

## 7. Conclusions and Practical Applications

In summary, life history theory holds that finite energy must be competitively distributed among competing processes, and that this competition will be heightened when available energy is low. This theory has many practical applications for athletes and their support-teams. For example, life history theory considers trade-offs provoked by LEA to be context-specific, with their direction and magnitude impacted by factors including age/life-stage, sex, dietary composition, and health and training status. As such, each athlete may respond differently to a given situation, and their individual characteristics and requirements should be considered. A "one size fits all" approach is unlikely to suit the majority, and so individualization of strategies is recommended. Although there is little doubt that prolonged exposure to very LEA (*e.g.,* across months or years) may result in adverse effects on health, life history theory would not consider energetic trade-offs and their effects to always be pathological *per se*. As such, athletes and their support staff should not be overly concerned by brief periods of LEA (*e.g.,* as may occur during days or weeks of intensified training or competition) in otherwise healthy athletes, but rather should pay attention to the warning signs the body provides (*e.g.,* menstrual cycle changes) and adapt

410 training and nutritional strategies as required. Consideration of these and other factors discussed in this review
411 may provide insight into how, and why, the body evolved to respond to periods of LEA, and ultimately serve to
412 better inform screening, identification and treatment strategies for at-risk athletes.

413

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416

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418

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423

424 **Conflicts of Interest**
425 Meghan Shirley, Daniel Longman, Kirsty Elliott-Sale, Anthony Hackney, Craig Sale and Eimear Dolan declare
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427

428 **Author Contributions**
429 MKS and ED conceived the original idea and developed the initial draft of this manuscript, with ongoing critical
430 input from DPL, ACH, KES and CS. All authors read and approved the final version.

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