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Shirley, Meghan K, Longman, Daniel P, Elliott-Sale, Kirsty J, Hackney, Anthony C, Sale, Craig ORCID logoORCID: <https://orcid.org/0000-0002-5816-4169> and Dolan, Eimear (2022) A life history perspective on athletes with low energy availability. *Sports Medicine*, 52 (6). pp. 1223-1234. ISSN 0112-1642

Downloaded from: <https://e-space.mmu.ac.uk/630595/>

Version: Accepted Version

Publisher: Springer Verlag

DOI: <https://doi.org/10.1007/s40279-022-01643-w>

Please cite the published version

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A life history perspective on athletes with low energy availability

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17 **Abstract**

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

33

34 **Key Points:**

35 ☐ Life history theory is a branch of evolutionary theory that describes how finite energy resources are
36 distributed among competing biological processes. “Trade-offs” are predicted to arise because energy
37 used to support one process cannot be used for others. Energy resources are allocated to tissues and
38 functions in a hierarchical manner, with priority afforded to those that offer the greatest immediate
39 survival value.

40 ☐ Because energetic trade-offs among biological processes are predicted to be heightened under conditions
41 of scarcity, a life history lens is particularly relevant for athletes who may have low energy availability
42 due to high exercise training energy expenditure, inadequate energy intake (resulting from conscious or
43 inadvertent restriction), or a combination of both.

44 ☐ Energetic trade-offs are context specific and may be impacted by a range of factors, including sex, life-
45 stage, training and health status, dietary composition and the length and severity of exposure to low
46 energy availability.

47

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

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 (<i>e.g.</i> , intentional restricted intake to achieve a desired body composition), or inadvertent (<i>e.g.</i> , 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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54 **1. Introduction**

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

62 *Evolutionary biologists* recognize that the biological response to LEA is an evolved trait, and have long examined
63 how finite energy is distributed among competing physiological processes in humans and other species within a
64 theoretical framework called *life history theory* [4,5,12–15]. Classically used to understand species-level
65 differences in the pattern and timing of important biological events (*e.g.*, maturation, reproduction, and death),
66 life history theory's conceptual framework has increased understanding of which physiological functions are
67 prioritized or sacrificed in humans under varying environmental circumstances, including energetic stressors such
68 as LEA. This has broad implications for health in human populations and may hold specific relevance for athletes,
69 many of whom struggle to achieve adequate energy availability due to high training energy expenditure and/or
70 *inadequate energy intake* (which may occur via conscious restriction, reaching alimentary limits [16], or due to
71 inadvertent under-eating [8,17]). Among athletes, those with particularly strenuous training schedules may incur
72 extreme energetic costs that can increase their susceptibility to LEA. Many evolutionary anthropologists use
73 exercise, sport and physical activity to investigate and inform theories of human evolution and adaptation [18–
74 22]. Similarly, sport and exercise scientists may find the overarching conceptual framework of life history theory
75 useful to consider the biological basis and variability of the human body's response to LEA. The aim of this
76 review, therefore, is 1) to describe the core tenets of life history theory; 2) consider trade-offs that might occur in
77 athletes with LEA in the context of four broad biological areas that are commonly considered within the life
78 history literature, *i.e.*, reproduction, somatic maintenance, growth and immunity; and 3) use this evolutionary
79 perspective to consider potential directions for future research.

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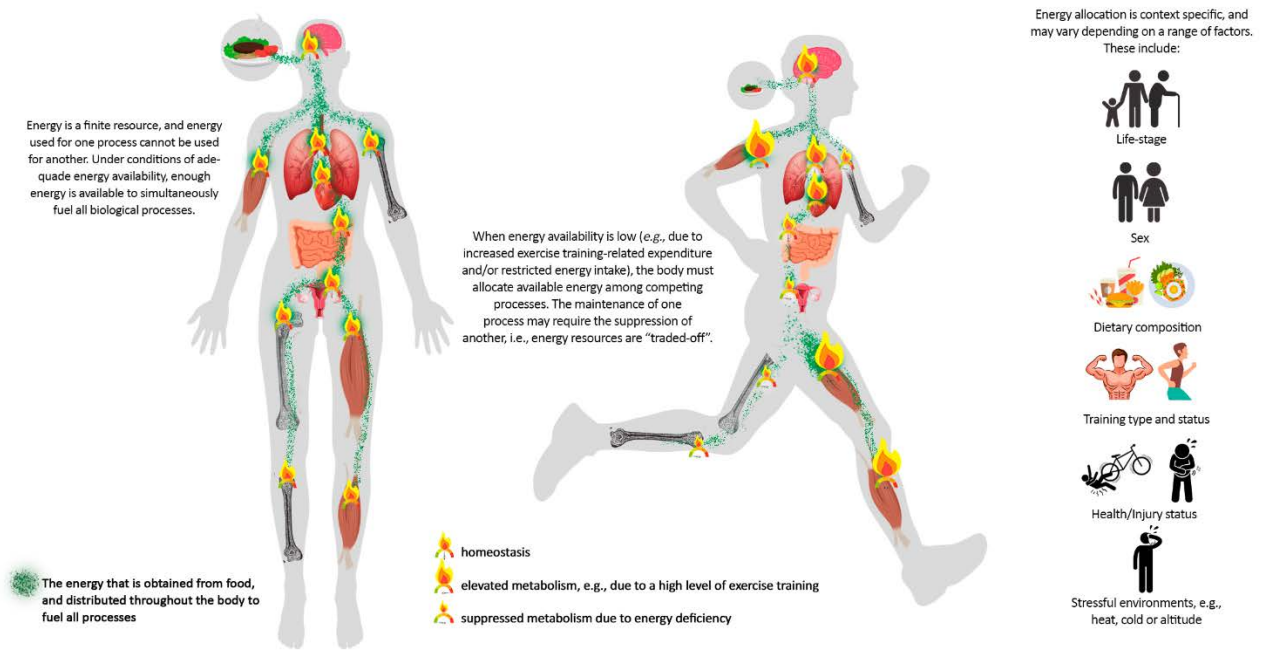
81 **2. Brief Background to Evolutionary Life History Theory:**

82 The notion of 'competition' features prominently in Charles Darwin's theory of evolution by natural selection
83 [23]. Darwin and his contemporary, Alfred Russel Wallace, described competition amongst individuals for the
84 resources needed to successfully pass genetic material to subsequent generations - this constitutes '*fitness*' in
85 evolutionary terms [24]. Life history theory, a branch of evolutionary theory, also predicts competition at an intra-
86 individual level. This theory considers energy to be a finite resource that is competitively allocated between four
87 principal areas: reproduction, growth, somatic maintenance, and immune defence [25–31]. Energy can also be
88 used for physical activity, or stored in adipose tissue, or as liver or muscle glycogen or triglycerides. A key tenet
89 of life history theory is that energy used for one function cannot be used for others, *i.e.*, energy resources are
90 "*traded off*" among competing functions. Notably, competition among functions will be heightened during
91 conditions of scarcity [30,32], as may be the case for athletes with LEA.

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

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

122



123

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

127

128 3. Reproduction

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

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

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

162 Although much of the sport and exercise science literature focuses on the influence of LEA on the reproductive
163 status of female athletes, it is important to highlight that men are also affected [9,43,59,60]. In addition to gamete
164 production (which has minimal energetic costs), male reproduction also incurs costs related to child-care,
165 attracting a mate and fending off male competitors, and reproductive ecologists have pointed to the modulation
166 of physiological correlates of male mating effort (*e.g.*, testosterone, muscle mass) during times of energetic stress
167 [39]. Evidence indicates that LEA can lead to downregulation of the hypothalamic-pituitary-testicular axis in
168 exercising men [9,61]. Sexual dimorphism does, however, seem to exist in relation to trade-offs made under
169 conditions of LEA [39,43,48,62,63]. This is unsurprising, given that the cost of reproduction is substantially
170 greater for women than men [39,49,50], meaning that women in a state of LEA have a stronger biological
171 imperative to weigh the benefits of reproductive investment against its energetic cost.

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

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

191

192 4. Somatic Maintenance

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

203

204 a. Bone

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

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

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

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

236

237 *b. Muscle and Fat Mass:*

238 Muscle is the primary tissue driving human movement. It is therefore a critical determinant of athletic
239 performance, in addition to human health [82]. Given this, one might expect muscle to maintain its energy supply
240 during conditions of LEA. Muscle is, however, a metabolically expensive tissue accounting for approximately
241 20% of resting energy expenditure in adult humans [83], and it incurs an even higher metabolic cost during
242 physical activity. Substantial energy savings could, therefore, be made by reducing muscle mass when resources
243 are scarce [9,84,85]. In addition, muscle provides an important reservoir of endogenous amino acids that can be
244 liberated during periods of scarcity and used elsewhere, for example, to support enzyme and immune factor
245 synthesis and maintenance of plasma protein concentrations [86]. This diversity in essential physiological
246 functions renders muscle tissue responsive to a wide range of stimuli. The response of muscle to LEA may depend
247 on which of its functions (*e.g.*, mechanical, regulatory or storage) proves to be most immediately essential. This
248 in turn may depend on a host of factors, including sex, life-stage, health and training status, body composition
249 phenotype, the length and severity of LEA, and the macro and micronutrient composition of available energy, as
250 discussed elsewhere [87]. For example, biological sex is thought to influence the muscle response to periods of
251 LEA, with men potentially more susceptible than women to muscle-loss during times of scarcity [88–90]. The
252 proportion of muscle mass loss increases exponentially with decreasing body fat content [91]. Women have, on
253 average, a larger “energy buffer” in the form of greater body fat stores than men [88,89,92]. This likely explains,
254 at least in part, the sexually dimorphic response of muscle and fat to LEA. To the best of our knowledge, however,
255 the relative loss of muscle and fat has not been directly tested in male or female athletes with LEA, some of whom
256 may already be at, or close to, their lower biological adiposity limits. It is feasible that the high training status and
257 baseline body composition of athletes may influence muscle-related trade-offs under conditions of LEA, but this
258 requires further investigation.

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

274

275 *c. Physical performance:*

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

293 Available intervention data directly investigating how LEA may impact exercise performance are conflicting,
294 with both positive, neutral and negative effects shown. For example, Oliver et al. [102] reported that 48 hours of
295 energy restriction reduced 30-minute treadmill time-trial performance [102], whereas Kimojo et al. [103] reported
296 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

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

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

362

363 **6. Perspectives for Future Research**

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

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

376 Currently, limited information is available regarding the extent or duration of LEA that will provoke deleterious
377 outcomes under different circumstances and this is an important avenue for ongoing research. Additionally, it is
378 important to consider that LEA is extremely challenging to accurately measure [125], while varying perspectives
379 exist related to factors such as the timeframe within which potential deficits should be considered. For example,
380 some suggest that 3- or 7-day rolling averages are more useful, while others indicate that greater within-day
381 imbalances may have increased consequences over similar intakes consumed more frequently throughout the day
382 [126]. Validation of differing approaches to outcome assessment and consideration of how varying characteristics
383 of LEA may impact health and related outcomes is an important direction for future research. This could include,
384 for example, investigations of larger deficits across shorter time-periods versus smaller deficits across longer time
385 periods, as well as imbalances arising primarily from higher training related expenditures versus more restricted
386 energy intakes. It is also important to consider that a life history perspective considers the transient suppression
387 of non-immediately essential physiological processes to be a normal, and necessary, adaptive response to
388 conditions of energetic stress. As such, conclusions about the influence of LEA on health or performance related
389 outcomes should only be made based on outcomes that directly relate to health or performance. Unsubstantiated
390 assumptions that all trade-offs are negative should be avoided, as should over-extrapolation of mechanistic
391 findings. Indeed, there may be situations where transient exposure to LEA within a periodized nutritional cycle
392 may be desirable to drive certain phenotypic outcomes [101], while more moderate energy restriction throughout
393 the life-course is hypothesized to increase longevity and health-span [127]. The topic of LEA in athletes is both
394 complex and nuanced, and embracing these characteristics of the research topic may be key to the development
395 of appropriate guidelines to support at-risk athletes.

396

397 **7. Conclusions and Practical Applications**

398 In summary, life history theory holds that finite energy must be competitively distributed among competing
399 processes, and that this competition will be heightened when available energy is low. This theory has many
400 practical applications for athletes and their support-teams. For example, life history theory considers trade-offs
401 provoked by LEA to be context-specific, with their direction and magnitude impacted by factors including
402 age/life-stage, sex, dietary composition, and health and training status. As such, each athlete may respond
403 differently to a given situation, and their individual characteristics and requirements should be considered. A “one
404 size fits all” approach is unlikely to suit the majority, and so individualization of strategies is recommended.
405 Although there is little doubt that prolonged exposure to very LEA (*e.g.*, across months or years) may result in
406 adverse effects on health, life history theory would not consider energetic trade-offs and their effects to always be
407 pathological *per se*. As such, athletes and their support staff should not be overly concerned by brief periods of
408 LEA (*e.g.*, as may occur during days or weeks of intensified training or competition) in otherwise healthy athletes,
409 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

414 **Acknowledgements**

415 The authors thank Rafaela Silvério Pinto for her assistance with the development of Fig. 1.

416

417 **Declarations**

418

419 **Funding**

420 Eimear Dolan is financially supported by the Fundação de Ampara a Pesquisa do Estado do São Paulo
421 (FAPESP: 2019/05616-6 and 2019/26899-6). No other sources of funding were used to assist in the preparation
422 of this article.

423

424 **Conflicts of Interest**

425 Meghan Shirley, Daniel Longman, Kirsty Elliott-Sale, Anthony Hackney, Craig Sale and Eimear Dolan declare
426 that they have no conflicts of interest relevant to the content of this review.

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.

431

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