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Low energy availability in male athletes: a systematic review of incidence, associations and effects

Item Type	Article
Authors	McGuire, Amy;Warrington, Giles D.;Doyle, Lorna
Citation	Translational Sports Medicine;3 (3), pp. 173-187
Publisher	John Wiley & Sons, Inc.
Download date	2026-06-17 22:53:30
Item License	https://creativecommons.org/licenses/by-nc-sa/1.0/
Link to Item	https://hdl.handle.net/10344/8812

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Article type : Review

Title: Low Energy Availability in Male Athletes: A Systematic Review of Incidence, Associations and Effects.

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Running Title: Low Energy Availability in Male Athletes

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This article has been accepted for publication and undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the [Version of Record](#). Please cite this article as [doi: 10.1002/TSM2.140](https://doi.org/10.1002/TSM2.140)

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34 The Female Athlete Triad has recently been suggested to be a threat to male athletes. This review aims to examine the
35 evidence, and associated effects, of low energy availability (LEA) in male athletes. A comprehensive search of
36 PubMed and SportsDiscus was performed. Three RCT and seven CS studies were included that measured energy
37 availability and included well trained males. Clinical LEA (<30 kcal/kg LBM/ day) or subclinical LEA (36 ± 6
38 kcal/kg LBM/day) was evident within all CS studies, documenting 25% of middle and long distance runners and race
39 walkers and 70% of cyclists with LEA. Two out of three RCTs and three out of seven CS studies reported disrupted
40 endocrine functioning, particularly reduced testosterone levels, in association with LEA. One CS study reported that
41 up to 40% of cyclists with LEA had low BMD. One CS study assessed metabolic health, reporting those with
42 suppressed levels spent more time in a severe energy deficit. This review highlights that LEA appears prevalent across
43 male athletic populations, in particular endurance and weight class athletes, and is a potentially serious threat to bone,
44 endocrine and metabolic health. Future larger scale longitudinal studies, using appropriate study designs, should be
45 undertaken to confirm these threats.

46 **Keywords:** exercise physiology; sports nutrition; bone health; endocrine function; metabolic health, energy
47 availability

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1. Introduction

62 Adequate nutritional intake, balanced with appropriate training stress and recovery, is critical to optimising sport
63 performance.^{1,2} Over the past number of decades a substantial body of scientific research has been published on
64 female athletes, investigating the Female Athlete Triad.³⁻⁶ The Female Athlete Triad is a condition typically observed
65 in exercising females, to include low energy availability with or without disordered eating, menstrual dysfunction and
66 low bone mineral density.⁷ Female athletes identified with this condition often present with one or more of the three
67 components, and immediate intervention is necessary to prevent the onset of more serious conditions such as clinical

68 eating disorders, amenorrhoea and osteoporosis.⁶ Although research has primarily focused on female athletes, there is
69 reason to believe that male athletes may present with similar symptoms⁸ as a result of reduced energy availability. In
70 2014 the International Olympic Committee (IOC) published a consensus statement on relative energy deficiency in
71 sport, communicating that low energy availability was no longer limited to female athletes, but also included male
72 athletes.⁹ Additionally, this position stand expanded the Triad conditions to include more than reproductive
73 functioning and bone health, by including aspects such as cardiovascular and metabolic health, where energy
74 availability is still the determinant for optimal functioning. This new spectrum was coined “Relative Energy
75 Deficiency in Sport” (RED-S).⁹ Furthermore, a more recent update of this position stand has reported additional
76 potential areas of concern for athletes such as haematological, gastrointestinal and exercise performance.¹⁰ The
77 suggestion to replace the Female Athlete Triad with the newly termed RED-S was refuted by the Female Athlete Triad
78 Coalition, who felt evidence supporting the RED-S model is still in its infancy. It has, however, been agreed that
79 further research is warranted in these additional areas and expanded to include male athletes.¹¹

80 Energy availability (EA) is defined as the amount of dietary energy remaining for all other metabolic processes after
81 the energy expended in exercise (EEE) has been subtracted from energy intake (EI) ¹² and is calculated as:

$$82 \quad EA = EI - EEE \text{ (adjusted for RMR)/kg LBM/day}$$

83 Optimal levels of EA are difficult to determine in the athletic population, however, according to Loucks and Thuma¹³,
84 for young and healthy sedentary females, energy balance equals 0 kcal/day when EA = 45 kcal/kg FFM/d (FFM = free
85 fat mass). Research has shown subclinical EA of 44 – 30 kcal/kg FFM/d poses no serious threat to physiological
86 responses in female athletes.¹⁴ At present there is no definitive number to determine LEA and contrary to what has
87 previously been accepted, recent research from Lieberman et al¹⁵ has shown that EA <30kcal/ kg LBM/ day does not
88 predict menstrual disturbances such as luteal phase defects, oligomenorrhea, and anovulation. This study also reported
89 that suppression of estrone -1-glucuronide (E1G) and pregnanediol glucuronide (PdG), proxy indicators of estradiol
90 and progesterone, occurred independent of EA status. Moreover, a review from Burke et al¹⁶ clearly highlights the
91 issues surrounding the current methods and calculations used to determine EA. Firstly, the initial establishment of the
92 LEA threshold of <30kcal/ kg LBM/ day was identified without considering inter-individual differences between the
93 subjects such as gynaecological age.¹³ Secondly, this review suggests that various systems within the body respond
94 differently to EA reductions. For example normal pulse frequency of luteinising hormone is apparent until EA
95 decreases below 30kcal/ kg LBM/ day, whereas insulin, leptin, and procollagen I C-terminal propeptide are
96 immediately reduced as EA starts to decrease.¹⁶ Finally the linear scaling of EA relative to LBM has been questioned
97 and it was concluded that a threshold of 30 kcal/ kg LBM/ day is not appropriate across different body sizes, due
98 individual differences in metabolic rates of skeletal tissue and vital organs. However there is evidence to suggest a
99 linear relationship between menstrual disturbances and EA <30kcal/ kg LBM/ day, with subjects being 50% more
100 likely to experience disruptions.¹⁵ Furthermore, when energy availability is less than 30 kcal/kg FFM/d reproductive
101 functioning, bone health¹³ and metabolic health¹⁷ were observed to be negatively affected. 40 kcal/kg FFM/d for
102 recreationally active males¹⁸ appears to be a threshold for physiological function. Recently, EA classifications for both
103 male and female athletes have been proposed as guidelines, rather than diagnostic tools (Table 1).¹⁴

104 Hormonal disturbances, including reduced testosterone,^{19,20} appetite suppressing hormone leptin, satiety hormones
105 ghrelin,²¹ and impaired bone health are amongst some of the consequences of LEA in male athletes.²²⁻²⁴ The majority
106 of studies which examined the associated consequences of reduced EA have involved female athletes.²⁵⁻²⁷
107 Nevertheless, there is a growing body of evidence which suggests male athletes are also at risk of the negative
108 sequelae of LEA, in particular those involved in high volume training,^{28,29} aesthetic based competition,³⁰ or weight
109 category sports.³¹ Specifically, research in male athletes has documented an increased disruption of hormone
110 levels^{19,32} with endurance trained male athletes significantly affected by lower testosterone levels across various sports
111 such as skiing,³³ cycling,³⁴ and triathlons.³⁵ There is evidence to support the threat that LEA poses for bone mineral
112 density (BMD) in male athletes,²²⁻²⁴ but significant gaps still remain in the scientific literature.

113
114 Therefore, the primary objectives of this systematic literature review were to investigate prevalence of LEA in male
115 athletes from cross-sectional studies, in addition to evaluating the associations and consequences of LEA in male
116 athletes from both cross-sectional and randomised control studies. Furthermore, the review sought to outline and
117 critically appraise the methods used to assess the prevalence of LEA and its effects.

118 The primary outcomes included the effect of LEA on bone health, reproductive health, metabolic health and endocrine
119 functioning in male athletes.

120

2. Methods

121 2.1 Search strategy

122 The Cochrane Collaboration Prisma protocol³⁶ was used to complete this review. A specific research question was
123 established as part of the search strategy, this supported the identification of key search terms. The online databases
124 PubMed, Sports Discus and individual manual searches were used to search for appropriate articles relating to the key
125 terms until the final search in November 2018. Medical Subject Headings (MeSH) terms were primarily used in the
126 PubMed database, followed by other relevant key terms where appropriate. Sports Discus was searched using
127 individual search terms and a search history of generated records for each key term from each database was created.
128 The Boolean logic was employed to search and filter through relevant results, terms including “OR” which identified
129 any articles containing one or more of said terms, “AND” which combined different terms. The search terms used to
130 generate articles included ‘athlete’ OR ‘male athletes’ AND ‘exercise’ OR ‘endurance sports’ OR ‘physical exertion’
131 OR ‘running’ OR ‘cycling’ OR ‘swimming’ OR ‘jockeys’ OR ‘boxing’ OR ‘rowers’ AND ‘bone health’ OR ‘bone
132 metabolism’ OR ‘metabolic health’ OR ‘endocrine health’ AND ‘energy availability’ OR ‘energy intake’ OR ‘energy
133 metabolism’. Only studies published in English and in human participants were included in the review.

134 2.2 Eligibility Criteria

135 Eligibility criteria for subject inclusion were: male athletes who had trained in their respective sports for more than
136 one year;³⁷ were free from injury or illness; measured EA, as opposed to energy balance;¹² suitable study design to
137 assess prevalence and/or effects of EA (cross – sectional, cohort or randomised control trials).

138 **2.3 Data extraction**

139 Articles generated as a result of the search in each online database were exported into an Excel spreadsheet. The titles
140 and abstracts of each retrieved article that resulted from the search strategy were screened based on the inclusion
141 criteria. Manual searching of the references within relevant articles were also screened to ensure no critical articles
142 were omitted. Journal article data was extracted to include: author and date, experimental design, study duration,
143 population, outcome measures and significant findings.

144 **2.4 Quality Assessment**

145 Articles using cohort and cross-sectional study designs were quality assessed using the NIH Quality Assessment Tool
146 for Observational Cohort and Cross-Sectional Studies Randomised control trials were evaluated for quality using the
147 Physiotherapy Evidence Database (PEDro) scale.^{38,39} The cross sectional and cohort studies extracted were evaluated
148 and categorised into three categories based on the eligibility criteria: low, medium or high.⁴⁰ All studies were
149 included, with limitations discussed for those that rated low in quality. The PEDro scale rated the randomised
150 controlled trials from 0 to 10, with 6 representing the cut-off score for high-quality studies.³⁹

152

153

3. Results

154 A total of 2626 articles were identified from the online databases PubMed ($n = 724$), and Sports Discus ($n = 1902$)
155 (Figure 1). After the removal of duplicates, 2580 articles remained. Titles and abstracts were screened resulting in a
156 total of 45 RCTs and 49 CS studies considered eligible. No cohort studies were deemed appropriate for review. After
157 further evaluation, a total of 9 studies (RCT = 3; CS = 6) were included. Both RCTs and CSs were quality assessed
158 and included in the review. Manual screening of the bibliography of the 9 chosen studies was also performed to ensure
159 no relevant articles were excluded ($n = 280$). One CS study was extracted from these references. A final total of 10
160 studies (RCT = 3; CS = 7) were included in this review.

161 3.1 Characteristics of the Studies

162 The studies selected included a total of 275 adult participants ($n = 200$ males; $n = 75$ females) from various sporting
163 backgrounds ($n = 27$ jockeys; $n = 208$ endurance athletes; $n = 12$ wrestlers; $n = 28$ trained individuals). The sample
164 size of studies ranged from 6 – 59; with the RCTs having between 6 and 22 participants and the CSs having between 9
165 and 59 participants.

166 3.2 Study Quality

167 Quality assessment procedures were applied to both the cross-sectional and randomised control trials respectively. All
168 of the 7 cross-sectional studies achieved >7 out of 13, categorising them as medium quality.⁴⁰ None of the cross-
169 sectional studies justified the choice in sample size or measured exposures of interest prior to outcome measure. One
170 of the cross-sectional studies assessed the different levels of exposure i.e. throughout the competition season. None of
171 the outcome measures or assessors were blinded. Two out of 3 RCT studies achieved a total of 7, with the remaining
172 study scoring 6. Concealment of allocation is not entirely relevant in studies of this nature because, given the nature
173 of energy intake and expenditure methods and sample selection used; it is difficult for researchers to limit
174 transparency for both themselves and participants. Blinding of subjects and researchers was also not applicable. Six
175 out of the seven CS studies tested participants for >3 days, one CS study assessed participants for up to 1 day.²⁶

177 3.3 Methods of Assessing Energy Availability

178 Table 2 summarises the methods of assessing energy availability used across the cross sectional studies. Energy
179 intake was measured via food frequency questionnaire⁴¹ or 3 – 7 day food diaries using household measures⁴² or
180 weighed records.^{26,43–45} One study measured energy intake 3 days per month.⁴⁶ Exercise energy expenditure was
181 assessed by training logs,^{41,43,44,46} heart rate monitoring^{26,45} or through use of the Sensewear armband.⁴² Four out of
182 seven studies (57%) derived resting metabolic rate (RMR) from predictive equations,^{42–44,46} two studies (29%)
183 measured RMR using a ventilated canopy hood system^{26,45} and one study did not report any measurement of RMR.⁴¹
184 All studies used dual energy x-ray absorptiometry (DEXA) to determine lean body mass (LBM).

185 3.4 Prevalence of Low Energy Availability

186 From the studies examined, 3 out of 7 CS studies and 1 out of 3 RCT studies included female participants, but the
187 results of male only athletes are considered within this review. Clinical LEA (<30 kcal/kg LBM/ day) or subclinical
188 LEA (36 ± 6 kcal/kg LBM/day)¹⁴ was prevalent in all of the cross-sectional studies (Table 3). Participants included
189 jockeys,⁴² wrestlers,⁴⁷ cyclists,⁴⁶ endurance athletes.^{43–45} Dolan et al⁴² investigated the nutritional and lifestyle factors
190 in 27 jockeys, 21 of these submitted written food diaries and results indicated that all of those who recorded their
191 energy intake were in a state of LEA (12 kcal/kg LBM/day). McMurray et al⁴⁷ explored the effects of calorie
192 restriction on 12 male wrestlers. The habitual energy intake of these athletes was 21 kcal/kg LBM/day, indicating the
193 presence of LEA prior to partaking in any exercise or training. Similarly, Viner et al⁴⁶ reported the mean EA of male
194 cyclists (n = 6) across the competition season was 20 kcal/kg LBM/day. More recently, Heikura et al⁴³ noted that 25%
195 of male distance runners had LEA status (<30 kcal/kg LBM/day). Furthermore, it has recently been documented that
196 male endurance athletes presenting with higher exercise dependency scores demonstrate positive associations with
197 subclinical EA and higher cortisol levels.⁴⁵

198

199 3.5 Associations and Consequences of Low Energy Availability

200 Two of the RCTs induced low energy availability (<30 kcal/kg LBM) in study participants.^{18,46} The remaining RCT
201 assessed the effect of high versus normal carbohydrate diets on wrestlers who were already in a state of low energy
202 availability⁴⁷ (Table 4).

203

204 3.5.1 Endocrine Function

205 All 3 RCTs assessed the effect of low energy availability on endocrine function. Additionally, 3 CSs assessed the
206 association of LEA with hormonal status. Koehler et al¹⁸ manipulated energy intake and expenditure such that each of
207 the six participants completed two conditions of low energy availability (15 kcal/kg LBM/day) and two conditions of
208 normal energy availability (40 kcal/kg LBM/day) for 4 days. Leptin, insulin, ghrelin, triiodothyronine (T₃),
209 testosterone and IGF-1 alterations were assessed. It was reported that LEA induced through dietary restrictions

210 resulted in significant decreases in leptin and insulin, but no changes in IGF-1, ghrelin, testosterone or free T₃ in
211 recreationally active participants. Contrary to these results, evidence from cross-sectional studies by Heikura et al⁴⁴
212 and Hooper et al⁴¹ suggest significantly lower testosterone levels is associated with LEA (21 kcal/kg LBM/day) in
213 both middle and long distance runners and race walkers. Additionally, McMurray et al⁴⁷ reported elevations in human
214 growth hormone (HGH) and significant decreases in IGF-1 in wrestlers under conditions of LEA (<30 kcal/kg LBM/
215 day). Papageorgiou et al⁴⁸ implemented two randomised, crossover studies to investigate the effects of low EA, (15
216 kcal/kg LBM/day) for 9 days, through combined energy restriction and exercise, on IGF-1, leptin, insulin, T₃ and
217 ghrelin in recreationally trained males and females. Similar to Koehler et al¹⁸, a significant decrease in leptin levels
218 was observed, however unlike Koehler et al¹⁸, Papageorgiou et al⁴⁸ also reported increases in ghrelin.

219 3.5.2 Bone Health

220 One RCT⁴⁸ investigated the effects of LEA on bone health. In addition to this, 2 CSs^{43,46} also assessed the correlation
221 between LEA and bone health. A recent RCT by Papageorgiou et al⁴⁸ examined bone mineral density (BMD) via
222 dual energy x-ray absorptiometry (DEXA) and bone turnover markers, both in terms of resorption using β -carboxyl-
223 terminal cross-linked telopeptide of type I collagen (β -CTX) and formation using amino-terminal propeptide of type 1
224 procollagen (P1NP), in recreationally trained males and females when LEA (<15kcal/kg LBM) was induced for a
225 period of 9 days. No significant difference in BMD or bone marker concentrations were seen in the male participants
226 when in LEA.

227 In contrast, a longitudinal, cross sectional study by Viner et al⁴⁶ investigated the prevalence of LEA across a
228 competition season in cyclists and the potential impact on bone mineral density (BMD) via DEXA scanning at 0
229 months, 5 months and 10 months. Over 70% of cyclists reported LEA (<30 kcal/kg LBM/day) across the season, with
230 40% of participants having low BMD at the lumbar spine and 10% with low BMD at the femoral neck (Z score <-1).
231 Similarly, Heikura et al⁴³ examined the incidence of LEA (<30 kcal/kg LBM/day) and its effects on BMD in
232 Olympian athletes (middle and long distance runners, racewalkers) during a 3 – 4 week training camp using DEXA.
233 However, no significant correlations were seen between incidence of LEA and low BMD in male participants.

234 3.5.3 Metabolic Health

235 Torstveit et al²⁶, to the author's best knowledge, is the only study to investigate the effects of within-day energy
236 balance (WDEB) in male athletes with suppressed and normal resting metabolic rate (RMR). Sixty five % of the
237 participants (n = 46) were found to have suppressed RMR. However, whilst there was no correlation between the
238 presence of suppressed RMR and prevalence of EA or energy balance, it was clear subjects with lowered RMR were
239 more likely to have spent large parts of the day in energy deficit. Additionally, Koehler et al¹⁸ assessed the effects of
240 induced LEA on metabolic substrates glucose, glycerol, free fatty acids (FFA) and also fat mass (FM) and fat free
241 mass (FFM). When in a state of LEA (15 kcal/kg LBM/ day), athletes presented with reduced insulin, glucose and FM
242 and elevated levels of glycerol and FFA, FFM remained unchanged.

243

244

4. Discussion

245 This systematic literature review investigates the methods used to determine the prevalence of LEA in male athlete
246 and examines its association with and effects on various health markers. This review is the first of its kind to
247 systematically review and critically appraise the current scientific evidence pertaining to this condition in the male
248 athletic population. The findings of the 10 studies included in the review suggest that LEA exists within males in
249 variety of sports, and the presence of LEA is associated with suboptimal bone health, endocrine functioning and
250 metabolic health. However, currently, no study exists which examines the effect of LEA on cardiovascular health in
251 male athletes. It should however be noted that there is currently no gold standard method established for measuring
252 EA, particularly in free living athletes. The scientific literature was rigorously reviewed using the PEDro scale. The
253 moderate scores (6–7), although undesirable, were unavoidable due to the nature of the studies i.e. blinding of the
254 subjects or testers. Therefore the quality of the studies should not be devalued. Whilst the scope of this review
255 spanned across all athletic groups, the only studies that have been conducted to date are in endurance and weight class
256 athletes, therefore results obtained may only be applied to these groups..

257

258 4.1 Methods of Assessing Energy Availability

259 4.1.1 Energy Intake

260 An assortment of EI measures have be used including food frequency questionnaires,⁴¹ weighed food records,⁴³ and
261 food diaries⁴⁶ were implemented across each study, making dietary intake difficult to evaluate comparatively. Energy
262 intake via conventional methods has been shown to exhibit large discrepancies^{49,50} for a number of reasons, for
263 instance, food frequency questionnaires and nutrient databases often fail to provide adequate information regarding
264 sports nutrition supplements and ergogenic aids. Additionally, an athletes' busy lifestyle renders it difficult to recall or
265 record and weigh seven day food records, as would be considered the optimal time frame⁵¹. However two of the CS
266 studies were successful in retrieving food diaries for seven days during a training camp^{43,44} or race week⁴². For those
267 athletes whom such long training camps or racing weeks are not feasible, a three day weighted food intake monitoring
268 period is considered to provide reasonably accurate estimations of habitual macronutrient consumption⁵².
269 Surprisingly, Viner et al⁴⁶ chose to instruct participating cyclists to only submit a one day food diary per month on
270 alternating days. Irrespective of the time frame, inaccurate reporting of food intake, particularly under reporting, has
271 been shown to reduce the accuracy of dietary records as a means of measuring energy intake^{53,54}. This under-reporting
272 could be due to a number of factors including: inappropriate portion size estimation⁵³ or increased frequency of eating
273 occasions in endurance athletes⁵⁵ and in particular males⁵⁶. Alternative methods of assessing energy intake, such as
274 digital photography⁵⁷, have been used to increase the validity and accuracy of energy intake estimation. However,
275 athletes have been known to change their habitual food intake or omit certain foods that they feel may be considered
276 “unhealthy” or that are difficult to measure^{55,58}.

277 4.1.2 Energy Expenditure

278 4.1.2.1 Exercise Energy Expenditure

279 Total daily energy expenditure (TDEE) is not needed in the calculation of EA, therefore expensive techniques such as
280 doubly labelled water are not warranted. Exercise energy expenditure (EEE), adjusted for resting metabolic rate
281 (RMR) is however required. Four of the studies (57%) included in this review recorded EEE by means of training
282 logs^{41,43,44,46} and estimated the calories expended based on metabolic equivalents (METs)⁵⁹. A MET is defined as
283 approximately 3.5 ml O₂/kg/min for a 70kg individual⁵⁹. It should be noted however, that it is not possible to locate
284 the origin of the data on which the 1 MET constant is based, therefore it has been suggested that this may be an
285 arbitrary figure and not generalisable across other populations⁶⁰. Objective proxy measures of estimating energy
286 expenditure in the form of heart rate (HR) monitoring was used in both studies by Torstveit et al^{26,45}. Monitoring HR
287 can provide objective feedback regarding frequency, intensity, and duration of physical activity in free-living
288 situations, whereby energy expenditure is based on the assumption of a linear relationship between HR and oxygen
289 consumption throughout the majority of the aerobic work range⁶¹. The HR at various exercise intensities can be used
290 to estimate oxygen consumption so energy expenditure during free-living activities can be computed⁶². Furthermore,
291 in certain populations, such as lactating women, HR monitoring has been proven to be more reliable than the DLW
292 technique in estimating TDEE. The increased water turnover during lactation resulted in the slope of the isotope
293 disappearance appearing more similar, which inferred an error in estimates of energy expenditure, therefore indicating
294 the DLW method may not be appropriate for use in certain cohorts⁶³. A recent meta analysis by Capling et al⁵⁸ also
295 suggested that a similar error may occur in athletes when using DLW to measure TDEE due to the high water turnover
296 associated with physical activity. It is important to note, however, that the linear relationship between EE and HR is
297 unreliable at resting and at lower aerobic intensity workloads⁶⁴. Therefore combined methods such as HR with
298 accelerometry have been suggested to more accurately estimate free living exercise energy expenditure^{65,66}. Dolan et
299 al⁴² estimated exercise energy expenditure through the use of the SenseWear armband. This combined method of
300 measuring energy expenditure is worn over the tricep and combines accelerometry with heat production and skin
301 conductivity⁶⁷. Although it has been validated in different population groups at low and moderate intensities⁶⁸⁻⁷⁰, it
302 has shown to be underestimate high intensity energy expenditure in athletes^{67,71} and in particular male endurance
303 athletes⁷².

304 4.1.2.1 Resting Metabolic Rate

305 Resting metabolic rate (RMR) constitutes the largest proportion, 65 - 80% of TDEE and is defined as the rate of
306 energy expenditure at rest and reflects the minimum amount of energy required to carry out essential physiological
307 functions.⁷³ It is most accurately measured by indirect calorimetry immediately after a minimum of 8 hours of sleep
308 and 12 hours of fasting, with the individual lying supine.⁷⁴ Both of the studies conducted by Torstveit et al^{26,45}
309 included in this review used a ventilated hood canopy method to measure RMR via indirect calorimetry. Due to the
310 stringent conditions surrounding the indirect calorimetry method and the period of time needed to conduct the
311 procedure, predictive equations have been developed in its place⁷⁵⁻⁷⁸ and were chosen method used in the other cross

312 sectional studies included in this review^{42-44,46}. The Harris Benedict formula⁷⁷ has been widely used in research,
313 however lean body mass (LBM) was not included in the development of the equation. Cunningham later updated the
314 formula to include LBM, although it was predicted and not measured, as this was considered the greatest contributor
315 to RMR.⁷⁶ A recent review by Scholfield et al⁷⁹ investigated the validity of using predictive equations of RMR in
316 determining EA status in athletes. It was reported that both the Harris Benedict and the Cunningham equations were
317 found to overestimate RMR by 5% and 14-15%, respectively, therefore it is difficult to accept either of these
318 equations in the estimation of EA. Mifflin⁷⁸ later developed a new predictive equation to distinguish between males
319 and females. Both the Cunningham and Mifflin equations were used to estimate RMR in the remainder of the studies
320 in this review. Although there is evidence to suggest that these equations are the most accurate in predicting RMR in
321 athletes, both formulas are reported to over and underestimate RMR when compared to indirect calorimetry⁸⁰

322

323 4.2 Prevalence of LEA

324 Clinical LEA or subclinical EA were reported in all CS studies (Table 3), with 25% of middle and long distance
325 runners and race walkers and 70% of cyclists reporting with clinical LEA. However, accurate comparisons of
326 prevalence between studies is difficult due to the variability in measurement techniques and small sample sizes.
327 Furthermore, as stated earlier, there is currently no gold standard in assessing each of the elements of EA in free
328 living athletes. Moreover, the duration of data collection varied across all studies. Interestingly, only one study
329 measured EA longitudinally, across an entire season.⁴⁶ Viner et al⁴⁶ measured EA in male (n = 6) and female (n = 4)
330 competitive cyclists once per month across the cycling and training season. Although results indicated that 70% of
331 cyclists were in a state of LEA across the entire season, with male cyclists reporting a mean EA of 20 +/- 9.9 kcal/ kg
332 LBM/ day, these results only represent one day per month as opposed to the recommended 3-7 days.. The remainder
333 of the studies included assessed EA over one week,^{26,42-45} or one day,⁴¹ rendering it increasingly difficult to make
334 explicit comparisons across the studies. Due to the cross sectional nature of the studies, it is unclear whether the
335 causes of LEA are intentional (disordered eating) or inadvertent (high volume training, lack of education).^{5,9} Either
336 way, LEA can contribute to nutrient deficiencies, in particular carbohydrates,⁴⁶ resulting in possible profound effects
337 on health and sports performance. Eating disorders (ED) and/or disordered eating (DE) behaviours are prevalent
338 across sports emphasising leanness such as endurance or weight class sports.⁸¹ Although EDs are more common in
339 female athletes, it was reported that twenty nine percent of male athletes competing in sports concerned with leanness
340 exhibit DE habits associated with distorted and dissatisfied body image.⁸¹ Although sparse, there is evidence to
341 suggest ED/DE behaviours may be associated with LEA. Recent research has indicated that male athletes presenting
342 with higher exercise dependency scores had more pronounced negative energy balance coupled with higher cortisol
343 levels, suggesting the body is in a catabolic state. This was further evident with reports of lower blood glucose, lower
344 testosterone:cortisol ratio and higher cortisol:insulin ratio, indicating protein breakdown.⁴⁵ This further highlights the
345 detrimental effects that LEA may pose to health, body composition and sports performance. The lack of
346 standardisation and consistency in measurement techniques and duration poses a challenge in determining the true

347 actuality of LEA in the male athletic population. Albeit a controlled laboratory study measuring energy intake and
348 expenditure would be desirable, it would not be indicative of real life settings.¹⁶

349 4.2 Associations of LEA

350 The possible relationship between energy availability and endocrine health was investigated in all three RCTs and
351 three of the seven CSs (Table 4). The RCTs assessed this association in athletes who were in a state of LEA, either
352 artificially (through diet/exercise manipulation)^{18,48} or naturally (as part of their training and diet regime).⁴⁷ Koehler et
353 al¹⁸ examined the effects of LEA through diet alone or diet and exercise on hormones and metabolic substrates. A
354 significant reduction in leptin, insulin and increase in FFA was seen in those with LEA through diet alone or through
355 diet and exercise combined. No significant difference was seen in IGF-1, testosterone, T₃ or ghrelin which is in
356 agreement with the findings reported by Papageorgiou et al⁴⁸. It was suggested however by Koehler et al¹⁸ that the
357 insignificant findings for testosterone may not be a true reflection of what is really at play. Circulating concentrations
358 of binding hormones, such as sex hormone binding globulin may have reduced the bioavailability of testosterone and
359 therefore blunted the endocrine suppression during these studies. As these binding hormones were not measured in
360 either of the studies, it is difficult to speculate the true status of testosterone. It should also be noted that both of these
361 studies^{18,48} were conducted over a short period of time (4-9 days) which may not represent an accurate time frame to
362 see robust changes in all areas of endocrine function. In contrast to these findings, a cross-sectional study by Heikura
363 et al⁴⁴ reported that 40% of world class middle and long distance male athletes had low testosterone levels and
364 furthermore, outlined that those with LEA had significantly lower total testosterone levels ($P < 0.05$; ES 1.40) than
365 those with moderate EA (30 – 45 kcal/ kg LBM/ day), in this population. In a similar population, Hooper et al⁴¹ found
366 that male long distance runners exhibiting exercise – hypo gonadal conditions had testosterone levels below the
367 normal threshold of 12 mmol/litre.⁸² Moreover, these athletes' energy availability ranged from moderate to low
368 (27.2±/– 12.7 kcal/kg LBM/day). Interestingly, the findings from both studies also documented a stronger correlation
369 between using qualitative measures such as questionnaires in identifying high risk participants, than using objective
370 measures, such as food diaries and activity logs, which have been seen to both over and underreport energy intake and
371 expenditure. Sample size was further highlighted as a potential confounding factor as high individual variation in
372 follicle stimulating hormone and luteinising hormone results, suggesting possible hypogonadism for two out of the
373 nine athletes in the study by Hooper et al⁴¹. It should be noted when reviewing the literature on endocrine function that
374 these outcomes are not entirely comparable with results derived from studies in females, particularly in reproductive
375 hormone levels due to female menstruation. A dose response relationship has been documented between energy
376 availability markers of hypothalamic pituitary gonadal function, such as reductions in luteinizing hormone pulse
377 frequency in females.^{13,83} However, no similar studies have investigated this effect in males. It has been proposed that
378 male endurance athletes may experience changes in the hypothalamic-pituitary-testicular axis that result in lower
379 testosterone levels⁸⁴ However, Hackney et al⁸⁴ suggested that a peripheral mechanism is the root of these changes in
380 males, in contrast to the central dysfunction that occurs in females.⁸⁵ Nevertheless, our current understanding of the
381 precise mechanisms of disruptions in male athletes is limited, as research in males have been through cross-sectional
382 studies or short term RCTs. Furthermore, unlike the seminal studies by Loucks et al⁸⁶ that investigated the effects of
383 EA on hormone pulsatility, studies conducted in males have assessed fasting hormone concentrations,^{41,43–45} thus

384 rendering it impossible to suggest that studies in male athletes have seen the same level of perturbation because they
385 have not investigated the same outcomes.

386 Three (2 CS and 1 RCT) studies in total examined the association and effect of LEA on bone health. Of these, one
387 cross-sectional study⁴⁶ concluded that those in conditions of LEA had low BMD. In contrast, the other CS⁴⁴ and the
388 only RCT investigating bone health⁴⁸ reported no association or negative effects of LEA on BMD or bone
389 metabolism. It should be highlighted that due to the dissimilarities between the duration of each of the above studies,
390 direct or definitive contrasts are difficult to make. In particular, changes in BMD have been suggested to take greater
391 than 10 months to be detected via DEXA scanning,^{46,87} therefore short term CS studies or RCT do not represent a
392 sufficient time frame for change. Furthermore, the methodologies used to assess bone health in these studies may be
393 questioned. Both Heikura et al⁴⁴ and Viner et al⁴⁶ assessed bone health using only DXA scanning to measure BMD.
394 DXA has been widely accepted as a valid measure of BMD and bone mineral content (BMC) in athletes, accepting
395 certain limitations⁸⁸⁻⁹⁰. However, according to Bolotin,⁹¹ DXA is an unreliable method of measuring BMD and in turn
396 bone health, because when it measures a site that contains any other tissue in addition to bone e.g. lean mass, fat mass,
397 the accuracy diminishes. It has been suggested that quantitative computed tomography (QCT) may be more sensitive
398 in assessing BMD.⁹² QCT measures pure trabecular bone volume (not cortical bone), where most of osteoporotic bone
399 loss occurs, whereas DXA measures both cortical and trabecular bone rendering a possibility for overestimation of
400 results.⁹² Additionally, peripheral QCT also provides an indication of bone strength through the use of calculated
401 “Strength Strain Index”^{93,94} and has been shown to predict fracture risk in non osteoporotic patients⁹⁵. Furthermore,
402 BMD alone provides insight into the quantity of bone tissue, which is insufficient as a measure of overall bone
403 quality,⁹⁶ therefore markers of bone metabolism such as bone formation markers, osteocalcin (OC) and PINP, and
404 bone resorption markers, CTX and NTX should be included in analysis of bone health. Papageorgiou et al⁴⁸
405 investigated the relationship between deliberately induced acute LEA on markers of bone metabolism. Similar to the
406 findings of Heikura et al⁴⁴, no associations were reported. Nevertheless, further research, perhaps longitudinal,
407 assessing both BMD and bone metabolism is warranted in this area.

408 One CS²⁶ and one RCT¹⁸ study examined the possible relationship between energy availability and metabolism.
409 Torstveit et al²⁶ assessed the within day energy deficiency in thirty one male cyclists, triathletes and long distance
410 runners. Sixty five % of the participants presented with suppressed resting metabolic rate (RMR) and these subjects
411 also spent more time in energy deficits exceeding 400 kcal. It was suggested by the authors these deficits could pose a
412 threat to both brain glucose availability and increase catabolic processes. This was further supported by the increased
413 cortisol and lower testosterone:cortisol ratio in this cohort, which could further lead to decrements in training
414 adaptations such increases in strength and lean body mass.⁹⁷ However, this study only spanned 24 hours, therefore
415 investigations of a longer duration would be required to validate these findings. Although RMR was not directly
416 assessed, Koehler et al¹⁸ indicated that LEA offers extreme interruptions to metabolic substrates as illustrated by the
417 increased levels of FFA and glycerol and reduced insulin, glucose and body fat.

418 Although there is no evidence to establish the effects of LEA on cardiovascular health in athletes, it is well established
419 that significant structural, conduction, repolarisation and peripheral vascular changes to the cardiovascular system

420 occur in patients with more severe LEA states such as anorexia nervosa.⁹⁸ Furthermore, gastrointestinal complications
421 arising from this extreme energy deficiency such as delayed gastric emptying, interrupted intestinal transit and liver
422 abnormalities are also apparent.⁹⁹ In athletic populations, immune function has been flagged as a high risk concern,
423 with recent evidence reporting LEA as a leading factor associated with impaired immune function in Olympic
424 athletes,¹⁰⁰ however more stringent measures and distinctions between male and female athletes should implemented
425 to further support this claim.

426 **4.3 Limitations**

427 One limitation of this review is the small number ($n = 10$) of studies included. This is due in part to the very recent
428 expansion of the Female Athlete Triad to include male athletes and additional associations with LEA,⁹ thus to date
429 evidence in this area is sparse. Furthermore, due to the stringent inclusion criteria, other studies were excluded as they
430 measured energy balance as opposed to energy availability, rendering the results inappropriate for the current review.
431 Although the studies included provide evidence that LEA exists in male athletic populations (Table 3), the sample
432 sizes remain relatively small throughout. Moreover, the studies included have used a varied range of protocols in
433 assessing the prevalence of LEA and establishing its associations. There is currently no gold standard measurement in
434 the assessment of EA, with a variety of techniques and methods used to measure each of the components that
435 constitute EA.¹⁶ Furthermore, EEE was assessed using a range of different measures such as training logs^{41,44} or heart
436 rate monitoring, each with their own limitations.⁴⁵ Moreover, one study failed to adjust for RMR when assessing
437 EEE,⁴¹ and those that did include RMR modifications used a variety of methods such as ventilated hood²⁶ or
438 predictive equations⁴⁶ rendering the adjustment inconsistent and possibly inaccurate. Due to the differences in
439 sampling techniques used to collect and measure data, the associations of LEA reported also lack consistency, with
440 some studies reporting references ranges of endocrine function to identify participants with “reduced” or “low”
441 testosterone⁴¹ and others stating the lowest quartile of results.⁴⁴

442 **4.4 Perspective**

443 DE coupled with high training volumes places athletes at high risk of LEA and its effects, yet there is little evidence
444 pertaining to the severity of this problem. Despite the term RED-s being coined over 5 years ago, a dearth of
445 conclusive evidence exists, indicating that this is a priority research area. This review highlights that LEA may be
446 evident across various male sporting populations and poses potentially serious risks to bone, endocrine and metabolic
447 health in male athletes.^{26,45,101} Furthermore, although little or no substantial evidence exists at present, it has been
448 suggested that male athletes may be at risk of cardiovascular dysfunction,¹⁰¹ impaired mental health,¹⁰ and immune
449 system impairment.¹⁰⁰ Due to a limited number of studies conducted in this area at present, conclusions of this review
450 are limited to male athletes involved in endurance and weight class sports. Future larger scale studies in male sporting
451 populations should be undertaken, using appropriate study designs and for longer duration to confirm these potential.
452 Methods of measurements of both energy intake and expenditure should also be evaluated and perhaps the inclusion
453 of both qualitative and quantitative methods of data collection should be considered in future research. The limitations

454 of each method of assessing EA should be kept in mind when implementing each protocol. This review has presented
455 the 'tip of the ice berg' in terms of potential areas for expansion.

Accepted Article

456 **Funding sources and potential conflicts of interest**

457 There are no conflicts of interest or external funding sources applicable to this submission.

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Figures & Tables

Table 1. Modified Classification of EA Levels

Classification	Males	Females
High EA	> 40 kcal/kg LBM/day	> 45 kcal/kg LBM/day
Optimal EA	\geq 40 kcal/kg LBM/day	\geq 45 kcal/kg LBM/day
Subclinical LEA	30 – 40 kcal/kg LBM/day	30 – 45 kcal/kg LBM/day
Clinical LEA	< 30 kcal/kg LBM/day	< 30 kcal/kg LBM/day

Adapted from Melin et al¹⁴

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Table 2: Methods Used in Assessing Energy Availability Components

Component	Method	Problem
Energy Intake	Food Frequency Questionnaire ⁴¹	Significant amount of under-reporting or changing habitual eating habits with all of these methods
	3 – 7 day Weighed Food Record ^{26,45,43,}	
	7 day Food Diary Using Household Measures ⁴²	
	3 day/month Food Diary Using Household Measures or Scales ⁴⁶	
Exercise Energy Expenditure	Training Logs ^{41,43,44,46}	METs data not be specific for athletes
	Heart Rate Monitoring based on regression equation derived from VO_{2max} ^{26,45}	Unreliable at rest and at lower aerobic intensity workloads
	Sensewear Armband ⁴²	Overestimates EE at high intensities
Resting Metabolic Rate	Predictive Equations ^{42–44,46}	Shown to under and over predict when compared to indirect calorimetry
	Ventilated Hood ^{26,45}	Gold Standard
Lean Body Mass	Dual Energy X-Ray Absorptiometry ^{26,41–46}	Gold Standard

Table 3. Summary of the Incidence and Association of LEA Reported in Male Athletes

Reference	Study Type	Participants	Study Design	Duration	Outcome Measures	Key Findings
Heikura et al ⁴³	Cross-sectional	n = 27 females, age 26 ± 3.2 years n = 21 males, age 27.2 ± 4.1 years Highly trained middle, long distance runners and race walkers	Prevalence of EA during 3 - 4 wk training camp at altitude and assess the association of EA on Hb mass and iron status	7 days	Hb mass, sex hormones, BMD, injury/illness frequency, EA, body composition	EA 36 ± 6 kcal/kg LBM/ day in male athletes, but no effect on Hb mass at altitude
Heikura et al ⁴⁴	Cross-sectional	n = 35 females, age 25 ± 3.6 years (n = 22 eumenorrhoeic; n = 13 amenorrhoeic) n = 24 males, age 27.1 ± 3.9 years (n = 10 low testosterone; n = 14 normal testosterone) Highly trained/world class middle and long distance runners and race walkers	Examine EA via food records and EEE via training records and investigate metabolic and reproductive function, injury illness and body composition, during pre-competition training camp	7 days	EI, EEE, metabolic and reproductive hormone levels, injury and illness rates, body composition	25% of males (n = 6) had LEA (<30 kcal/kg LBM/ day). Males with LEA had low testosterone levels (14.8 ± 3.6 nmol/l; <i>P</i> < 0.05; ES 1.40). 63% of males categorised as being low risk, 37% moderate risk of RED-s
Hooper et al ⁴¹	Cross-sectional	n = 9 males, age 36.3 ± 9.2 years, distance runners with EHMC n = 8 male, age 30.8 ± 6.3 years, non-running, without EHMC	Establish EA in male distance runners with EHMC vs non-runners without EHMC. Examine associated effects of LEA in those with EHMC on reproductive health	1 day	Endocrine markers, psychological markers, EA	EA (EHMC 27.2 ± 12.7 vs. CONT 45.4 ± 18.2 kcal/day; <i>P</i> = 0.029) and reduced mean testosterone (EHMC 9.2 ± 2.3 nmol/L vs. CONT 16.2 ± 3.4 nmol/L; <i>P</i> < 0.001)

Torstveit et al ²⁶	Cross-sectional	n = 31 males, age 34.7 ± 8.1 years Cyclists, triathletes and long distance runners	Estimate and compare within day energy deficiency in male endurance athletes with suppressed (RMRratio < 0.90, n = 20) and normal (RMRratio > 0.90, n = 11)	7 days	RMR, EA, body composition, blood analysis	Suppressed and normal RMR groups similar 24-hour EB (-861 ± 832 kcal vs -402 ± 1056 kcal) and EA (37 ± 12 kcal/kg LBM/day vs 41 ± 11 kcal/ LBM/day). Suppressed RMR group more time in energy deficits exceeding 400 kcal (<i>P</i> = 0.023). Within day energy deficiency was associated with ↑ cortisol levels (<i>r</i> = -0.499; <i>P</i> = 0.004) and ↓ testosterone: cortisol ratio (<i>r</i> = 0.431; <i>P</i> = 0.015)
Viner et al ⁴⁶	Cross-sectional , Longitudinal	n = 6 males, age 42.0 ± 7.7 years n = 4 females, age 38.4 ± 10.3 years Competitive cyclists	Examine EA of adult male and female competitive cyclists across the cycling training and competition season. Study eating behaviours that may contribute to LEA. Compare EA of male versus female	Once per month across season (10 months)	BMD, EA	EA remained <30 kcal/ kg LBM/day across the season in 70% of cyclists. Male cyclists mean EA 20 ± 9.9 kcal/kg LBM/day. 40% low BMD at the lumbar spine, 10% low BMD at the femoral neck

			cyclists			which remained constant across the season
Dolan et al ⁴²	Cross-sectional	n = 27 males, age 27.3 ± 6.8 years National Hunt and Flat racing jockeys	Investigate diet and lifestyle practices of professional jockeys using 7 day food diary, questionnaire. Calculate EA	7 days	EI, EE, body comp, weight loss practices	Mean race day EA = 12 ± 0.8 kcal /kg LBM/ day
Torstveit et al ⁴⁵	Cross-sectional	n = 53 male, age 35.3 ± 8.3 years Cyclists, triathletes and long distance runners	Investigate associations between exercise dependency, EA and eating disorders	7 days	EI, EE, Body composition, exercise dependency, blood analysis	30% of athletes presenting with higher exercises dependency scores reported LEA (<30 kcal /kg LBM/ day). Significantly higher cortisol levels in those with higher exercise dependency scores (549±120mmol/L) vs lower exercise dependency scores (455±109mmol/L) (p=0.005)

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LEA = Low Energy Availability; EI = Energy Intake; EEE = Exercise Energy Expenditure; EA= Energy Availability; RMR = Resting Metabolic Rate; FFA = Free Fatty Acids; BMD = Bone Mineral Density; EHMC = Exercise-Hypo gonadal Male Condition; Hb mass = Haemoglobin mass; CONT = Control; EB = Energy Balance; vs = versus; ↑ = increase; ↓ = decrease; RED-s = Relative Energy Deficiency in Sport

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806**Table 4. Summary of the Effects of LEA in Male Athletes**

Reference	Study Type	Participants	Study Design	Duration	Outcome Measures	Key Findings
Papageorgiou et al ⁴⁸	RCT	n = 11 males, age 26 ± 5 years n = 11 females, age 26 ± 5 years Recreationally active Moderate – vigorous physical activity > 3 hrs per week	Crossover RCT 2 x 9 day periods of either balanced EA (45 kcal/kg LBM/day) or restricted EA (15 kcal/kg LBM/day) achieved through diet and exercise to assess effects of restricted EA on bone turnover	2 x 9 days	Body comp, B-CTX, P1NP, parathyroid hormone, IGF-1, leptin, insulin, T ₃ , GLP-2, minerals	Restricted EA (15 kcal/kg LBM/day) = ↓ body mass (-2.5 ± 0.8%; <i>P</i> < 0.001) in males. Specific values for hormones and blood markers not reported in the results

Koehler et al ¹⁸	RCT	n = 6 males, age 25.2 ± 1 year Recreationally active	Crossover RCT 4 conditions: LEA (15 kcal/kg LBM/day) + exercise (equating to 15 kcal/kg LBM/day) LEA + no exercise Normal EA (40 kcal/kg LBM/day) + exercise Normal EA + no exercise Aim 1: Impact of controlled LEA on metabolic hormones Aim 2: Will effect differ if LEA achieved through diet alone vs diet + exercise	4 days per condition	Serum testosterone, free T ₃ , insulin, IGF-1, leptin, ghrelin, glucose, glycerol, FFA	Aim 1: Both LEA groups = ↓ in body mass (2.1 ± 0.35kg; <i>P</i> = 0.016; 95% CI), ↓ leptin (-53 to 56%; <i>P</i> = 0.0181), insulin (-34 to -38%; <i>P</i> = 0.031) and fasting glucose (-8 to -12%; <i>P</i> = 0.046; <i>P</i> = 0.031) and ↑ glycerol (+88 to +167%) and FFA (+70 to +112%) concentrations. T ₃ , IGF-1, ghrelin and testosterone not affected. Aim 2: Effects not different when LEA was achieved through diet alone or diet + exercise
McMurray et al ⁴⁷	RCT	n = 12 males wrestlers, age 20 ± 1 year Wrestlers	Parallel RCT LEA (21 kcal/kg LBM/day) with normal (NC) /high carbohydrate (HC) Aim: To assess the effect of energy deficiency, with normal (50%) or high (75%) carbohydrate composition, on aerobic, anaerobic performance, human growth hormone and IGF-1	7 days	Aerobic and anaerobic performance, IGF-1, growth hormone	LEA with HC or NC intake ↓ in body mass (2.43 ± 0.20 kg; <i>P</i> < 0.05); ↓ body fat % from 9.7 ± 0.84 to 7.9 ± 0.75% (<i>P</i> < 0.05); ↓ post 8min run lactate levels (HC = 1.3 ± 0.2 to 4.5 ± 1.0 mM/l; NC = 1.2 ± 0.1 ± to 3.2 ± 0.2 mM/l); resting hGH ↑ (HC = 2.85 ± 1.46 ng/ml; NC = 2.85 ± 1.47 ng/ml; <i>P</i> < 0.05), resting IGF – 1 levels ↓ (HC = 1.44 ± 0.76 U/ml; NC = 1.36 ± 88 U/ml; <i>P</i> < 0.05)

808 RCT = Randomised Controlled Trial; LEA = Low Energy Availability; EI = Energy Intake; P1NP = total procollagen type 1 N-terminal propeptide; T₃ = Triiodothyronine; EEE = Exercise Energy
809 Expenditure; GLP-2 = Glucagon-like peptide-2; IGF-1 = Insulin-like growth factor 1; EA= Energy Availability; FFA = Free Fatty Acids; B-CTX = Beta Cross Laps X;; Kcal = kilocalorie; LBM =
810 Lean body mass; Kg = Kilograms; ↑ = increase; ↓ = decrease; hGH = human growth hormone; HC = high carbohydrate; NC = normal carbohydrate
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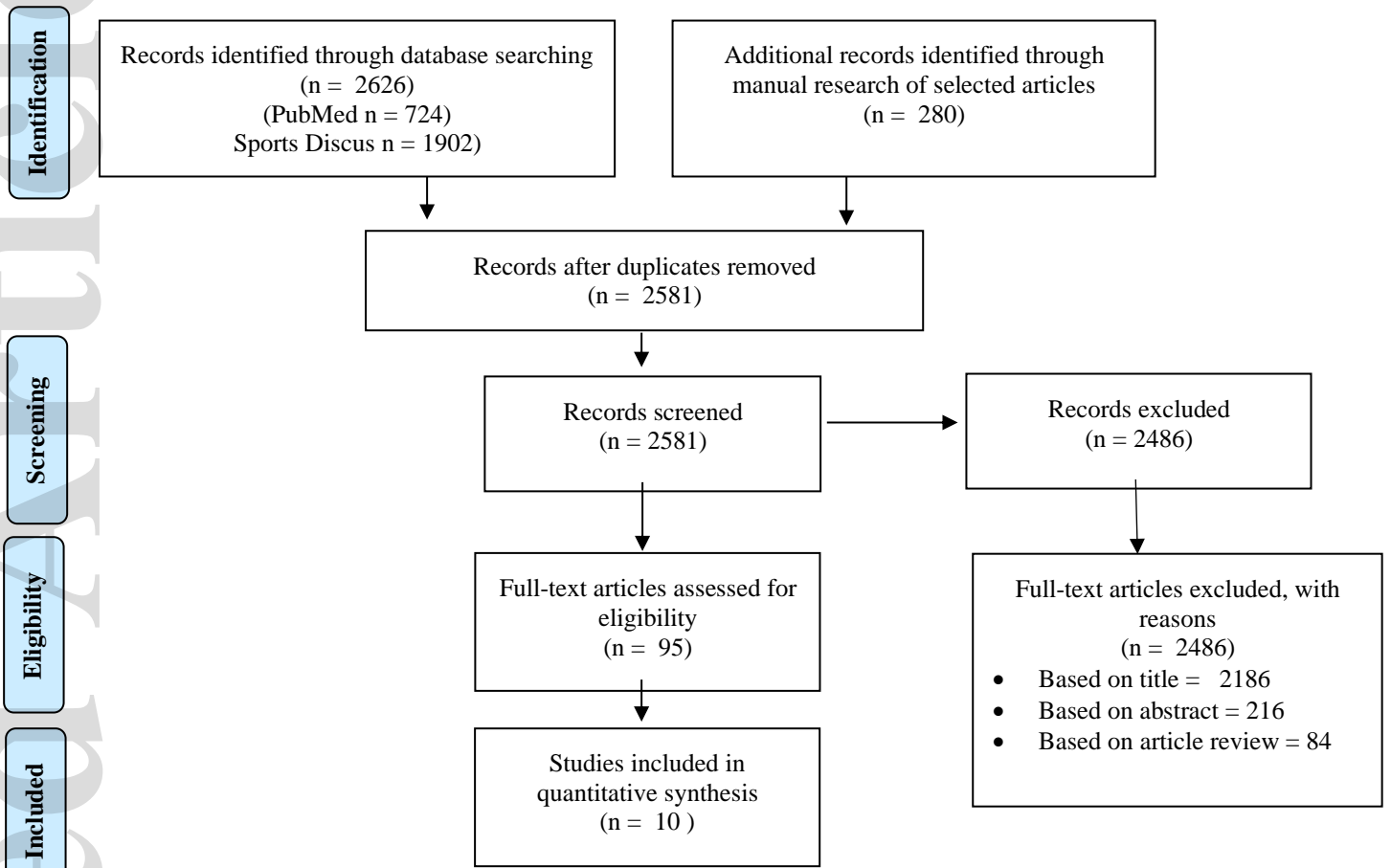


Fig 1

Prisma Flow Diagram