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Systematic Review

Does Lighter Mean Faster? Dietary Restriction and Fasting in Athletics: A Systematic Review

Leon Bedrač [1,*] and Simon Iskra [2]

[1] University of Portsmouth, Portsmouth PO1 2UP, UK
[2] Faculty of Sport, University of Ljubljana, 1000 Ljubljana, Slovenia; simon.iskra@fsp.uni-lj.si
* Correspondence: leon.bedrac@gmail.com

Abstract

Background/Objectives: Reducing body mass is common in weight-sensitive athletics, yet whether dietary restriction improves performance remains uncertain. This systematic review examined performance, body composition, and physiological responses to prescribed energy restriction, controlled low energy availability, time-restricted eating and Ramadan fasting during continued training in athletics. Methods: PubMed, Scopus, SPORTDiscus and Web of Science were searched up to 30 September 2026, with supplementary citation searching, in accordance with PRISMA 2020. Studies of dietary restriction or fasting during continued training in trained athletics populations, with a comparator condition or baseline, were eligible. All studies were appraised with CASP checklists, and the randomised studies were additionally assessed with the Cochrane RoB 2 tool. Reporting bias was assessed, the certainty of evidence was rated with GRADE, and findings were synthesised narratively by exposure category. Results: Twenty reports representing 16 studies and approximately 234 athletes (14.5% women) were included. No exposure category demonstrated a between-condition performance benefit. Elite race walkers improved 10,000 m performance after energy restriction followed by refuelling, but not more than under higher energy availability. In the only controlled Ramadan race comparison, 5000 m performance deteriorated without statistically significant changes in body mass or reported energy intake. Body mass and fat mass commonly decreased; significant fat-free-mass reductions were confined to bioimpedance-based studies. Bone-formation markers declined within days of restriction, whereas bone-resorption, iron-regulatory, endocrine and recovery responses varied with protocol and timing. The certainty of evidence was low to very low. Conclusions: The available evidence does not demonstrate that dietary restriction improves athletics performance. Outcomes measured during restriction or fasting must be distinguished from those assessed after refuelling. Lower body mass is not evidence of benefit, and a non-significant performance change is not evidence of physiological tolerance.

Keywords: athletics; track and field; energy availability; RED-S; weight loss; time-restricted eating; Ramadan; body composition; bone turnover; athletic performance

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1. Introduction

The importance of body mass and body composition for performance in athletics varies across events. While body-mass reduction is not a typical goal for throwers, sprinting and jumping require the optimisation of force and power relative to body mass. Distance running and race walking involve the repeated energetic cost of transporting body mass [1]. In these events, the belief that "lighter is faster" reinforces the appeal of intentional weight

loss [2]. Classic experimental work supports the energetic rationale: externally added load reduced relative aerobic capacity and distance-running performance [3]. Field observations are consistent with such practice: doubly labelled-water measurements documented negative energy balance in elite Kenyan endurance runners in the weeks before major competition [4]. Unlike height, body composition is modifiable through diet and training, albeit within individual biological constraints [5]. Weight management is typically embedded in periodised training, not undertaken in isolation. Indeed, energy expenditure, intake and body composition vary across the season in endurance disciplines [6], and planned body-composition periodisation has been documented across an Olympic-level female middle-distance runner's career [7]. These observations do not, however, establish a competitive benefit. There is little prospective evidence that weight loss improves performance in highly trained athletes. Any benefit may depend on the changes in fat mass and fat-free mass (FFM), nutritional costs and potential interference with recovery and training adaptations [2]. Neither an optimal rate of weight loss nor a protein intake that preserves performance and physiological function has been established for athletics [2].

Energy availability (EA) is defined as the difference between energy intake and exercise energy expenditure relative to FFM [8], whereas energy balance compares intake with total expenditure. Neither measure is equivalent to short-term body mass change, which also reflects glycogen, water and gastrointestinal content. Indicators of low energy availability (LEA) are common in athletics, particularly in distance running and jumping events [9]. In national- and world-class distance athletes, menstrual dysfunction in women, low testosterone in men and frequent bone injuries have been reported alongside LEA markers [10]. Recent cross-sectional work continues to document this risk profile at the collegiate level, including in populations underrepresented in earlier research [11]. Laboratory dose–response experiments show that bone-turnover markers respond differently to reductions in energy availability [12]; they do not establish a single threshold for all outcomes or for trained athletes undertaking deliberate short-term restriction. Evidence from rapid weight-making in weight-category sports is not directly transferable, because dehydration-based methods, recovery windows and competition demands differ fundamentally [13].

Energy restriction commonly reduces carbohydrate availability, which complicates the interpretation of physiological responses. Comparisons incorporating an energy-adequate ketogenic diet indicate that selected bone, iron-regulatory and immune responses are not specific to LEA [14,15]. These findings require marker-specific interpretation rather than treating energy and carbohydrate restriction as interchangeable exposures.

How well athletes tolerate dietary restrictions cannot be judged from performance alone. Stress fractures accounted for approximately 20% of injuries in one prospective athletics cohort [16], and collegiate track and field stress-fracture rates are among the highest [17]. Given the link between LEA and bone injury, physiological outcomes warrant attention alongside competition results. Procollagen type I N-terminal propeptide (P1NP) and β-isomerised C-terminal telopeptide of type I collagen (β-CTX) provide short-term biomarkers of bone formation and resorption, respectively. Importantly, they are not direct measures of skeletal strength or injury risk [14]. Timing also matters: testing after refuelling may answer a different question from testing during restriction, when training quality could be affected [2].

Recently, Guisado-Cuadrado et al. [18] reviewed biochemical responses to experimentally induced LEA across sports. However, to our knowledge, no systematic review has examined dietary restriction and fasting in athletics, integrating performance and body-composition outcomes with physiological biomarkers. The present systematic review therefore asks a narrower applied question: Among trained adults in weight-sensitive athletics, how do dietary restriction and fasting during continued training affect performance,

body composition and physiological biomarkers relative to an alternative condition or baseline? The objectives were to characterise exposures, synthesise outcomes and critically appraise the quality and applicability of the evidence.

2. Materials and Methods

2.1. Design and Registration

This systematic review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement [19]. The review was not registered. All search strategies, screening records, per-item appraisal justifications and risk-of-bias assessments are openly available in an Open Science Framework repository (Supplementary Materials).

2.2. Eligibility Criteria

Eligibility criteria were structured according to the PICOS (participants, interventions, comparators, outcomes and study designs) framework. Eligible participants were trained or competitive adults aged 18 years or over in running events from 800 m to the marathon, race walking, sprints, hurdles, jumps or combined events, spanning approximately Tiers 1–5 of the participant classification framework [20]; recreational runners were required to complete at least three weekly training sessions. Throwing events were excluded a priori, as body-mass reduction is typically not a performance goal in these disciplines. Eligible interventions targeted reductions in energy intake, energy availability, body mass or fat mass for at least three consecutive days alongside continued training. Time-restricted eating targeting body composition was eligible without a prescribed deficit; Ramadan fasting was considered separately because of concurrent food and fluid abstinence. Achieved weight loss was not an inclusion criterion. These four exposure categories (prescribed energy restriction, controlled LEA, time-restricted eating and Ramadan fasting) share the defining exposure of this review, a reduction or absence of energy intake during continued training. Because their mechanisms, prescriptions and contexts differ, they were synthesised as separate categories throughout, and no finding was pooled across categories. Comparators were adequate-intake or habitual diets, alternative dietary conditions, non-fasting control groups, or baseline. Eligible studies were required to assess at least one of the outcomes specified in Section 2.5. Randomised parallel and crossover, non-randomised controlled, and uncontrolled prospective pre–post designs were eligible. For feasibility, eligibility was restricted to English-language, peer-reviewed full reports. Exclusions comprised underage or untrained participants, non-athletics samples, exercise-only interventions, macronutrient manipulation without an eligible energy or physique target, pharmacological or surgical weight loss, acute dehydration-based weight-making, conference abstracts, and cross-sectional or retrospective designs.

2.3. Information Sources and Search Strategy

PubMed, Scopus and SPORTDiscus were searched from inception to 30 August 2026, providing biomedical, multidisciplinary and sport-specific literature (460, 707 and 330 records, respectively; total 1497). An updated search was conducted on 30 September 2026: all three databases were re-run in full from inception (461, 709 and 339 records), and Web of Science (Core Collection) was additionally searched from inception (731 records), translating the Scopus strategy into TS syntax. These four updated searches retrieved 2240 records, which constitute the identification total before deduplication. Because the updated searches were re-run in full from inception, records from the original search round are contained within them and were not counted twice. Population and intervention concepts were combined with AND and synonyms within each concept with OR, using subject

headings and title/abstract terms adapted to each database; individual athletics events were named rather than the generic term "athletics", which retrieves large numbers of institutional records; outcome and comparator terms were deliberately omitted from the strings and applied at screening, as both are inconsistently reported in abstracts. No database-level filters were applied. Full search strings for all four databases, with per-database retrievals for both search dates, are provided in Supplementary File S1. Backward citation searching covered the reference lists of all included reports and of the most closely related review [18]; forward citation searching of all included reports was conducted in Google Scholar (most recently 18 October 2026; 1129 citing-list entries in total).

2.4. Selection Process and Data Collection

Records were deduplicated in Zotero and screened in Rayyan [21]. One reviewer (L.B.) screened all records at title/abstract and full-text level and performed data extraction; a second reviewer (S.I.) verified all screening decisions at both stages and checked the extracted data and record handling against the source records and reports. Disagreements were resolved by discussion. Extracted fields included design; sample size, sex, age, event and calibre; dietary prescription, achieved intake, duration and protein intake; training; measurement methods and time points; and outcomes. Companion reports were linked as single studies for participant counting and appraisal while retaining report-specific samples, methods and time points. For Kojima et al. [22] and Ishibashi et al. [23], participant overlap is presumed from the shared protocol and research group rather than explicitly confirmed in the reports. Tabulated values were preferred; graph-only values were not treated as exact, and missing dispersion was not imputed. The complete screening record, including all full-text exclusions with reasons and the citation-searching log, is provided as Supplementary File S2.

2.5. Outcomes

Primary outcomes were event-specific performance and exercise capacity; secondary outcomes were body composition and physiological determinants of performance; tertiary outcomes were bone, iron and endocrine markers, with resting metabolic rate (RMR) retained as a supplementary metabolic outcome.

2.6. Quality Appraisal and Risk of Bias

Critical appraisal was conducted using the CASP Checklist for Randomised Controlled Trials and the CASP Checklist for Cohort Studies, selected according to the design of the appraised comparison [24,25]. Nine studies were appraised with the RCT checklist and seven with the cohort checklist; for Bouguerra et al. [26], the RCT checklist was applied because allocation to training-time groups was randomised, although the Ramadan exposure itself was not. Both authors completed appraisal, verified all item-level judgements, and disagreements were resolved by discussion. The full per-item justifications are provided in Supplementary File S4. Overall assessments were qualitative, with no numerical scores or predefined cut-offs, and informed interpretation rather than eligibility. In addition, risk of bias in the nine randomised studies was assessed with the Cochrane RoB 2 tool [27]: the version for individually randomised parallel-group trials for the three parallel-group trials and the variant for crossover trials, including its additional period and carry-over domain, for the six randomised crossover studies. Where the randomised element was the time of training or an exercise co-intervention rather than the dietary exposure, the assessment concerns that randomised comparison. Judgements across the five domains (randomisation process; deviations from intended interventions; missing outcome data; measurement of the outcome; selection of the reported result) were categorised as low risk, some concerns

or high risk, made by both reviewers and resolved by consensus; domain-level judgements are provided in Supplementary File S5. The single-arm cohort designs were appraised with CASP only.

2.7. Synthesis Methods

Heterogeneity of exposures, comparators, outcomes and measurement schedules precluded meaningful meta-analysis, even within exposure classes, where few studies shared comparable designs. Findings were therefore synthesised narratively in line with Synthesis Without Meta-analysis (SWiM) guidance [28], distinguishing within-arm changes from between-condition effects and, for Ramadan studies, outcomes assessed during fasting from those assessed afterwards. Structured descriptive comparisons and exploratory restrictions of the evidence base are presented with the results (Section 3.6); the exploratory restrictions serve as sensitivity analyses of study quality and design. Reviewer-calculated weekly mass-change equivalents and the relative contributions of fat and fat-free mass to measured losses are defined in the corresponding display legends; these are descriptive aids and should not be interpreted as estimates of between-condition effects.

2.8. Reporting Bias and Certainty of Evidence

Risk of bias due to missing results was assessed for each synthesis by comparing the methods and available trial records of each study with its reported results; gaps were linked to the relevant outcome or comparison and distinguished from missing participant data, incomplete statistics and results outside the eligibility criteria, and a gap alone was not taken as evidence of selective reporting. Registration was classed as prospective when the record was registered or first posted before the study started. Funnel-plot-based assessment was not feasible because no synthesis included ten or more studies.

The certainty of evidence for each outcome and comparison within each exposure category was rated with the GRADE approach [29], considering risk of bias, inconsistency, indirectness, imprecision and publication bias, starting at high certainty for bodies of evidence from randomised studies and low for those from non-randomised studies, which were rated separately. Very small trials without an informative comparative interval were downgraded two levels for imprecision. Both reviewers made the missing-results and certainty judgements independently and resolved disagreements by discussion; the outcome-specific assessments and agreed ratings are reported in Section 3.8 and Supplementary File S6.

3. Results

3.1. Study Selection

The updated four-database searches retrieved 2240 records; after removal of 987 duplicates, 1253 were screened at the title/abstract level, and 1228 were excluded. Twenty-five reports were sought; one was not retrieved, and 24 were assessed at full text, of which seven were excluded (wrong intervention n = 3, wrong design n = 3, no eligible outcome n = 1), yielding 17 reports through the database stream. Citation searching identified 30 candidate reports, of which one was not retrieved, 29 were assessed, and 26 were excluded (wrong population n = 17, wrong intervention n = 5, wrong design n = 4), yielding three additional reports. In total, 20 reports represented 16 studies (Figure 1) involving approximately 234 athletes, including 34 women (14.5%). Participant totals used the largest analysed sample from the eligible comparisons of each study.

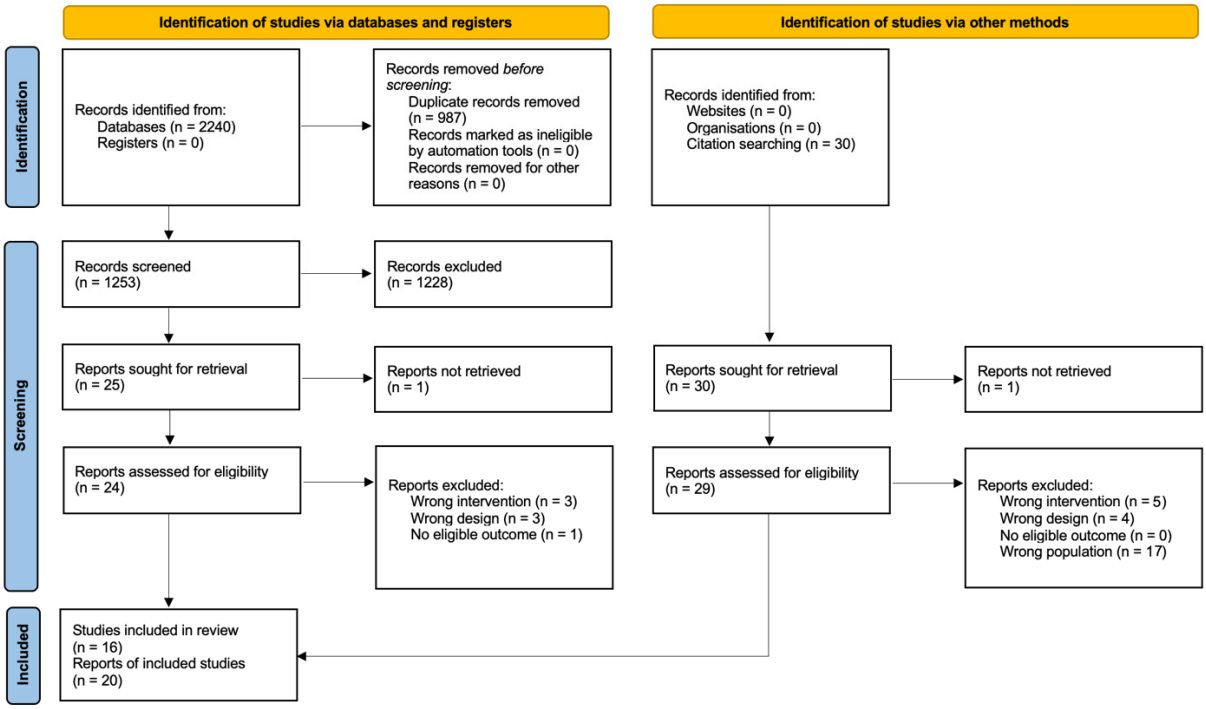

Figure 1. PRISMA 2020 flow diagram outlining the process of study selection [19].

3.2. Study Characteristics

Four exposure categories emerged (Table 1): prescribed energy restriction or weight-loss programmes (three studies; 300–750 kcal/day deficits, energy intake at approximately 50% of estimated requirements, or 5% body mass over four weeks); controlled LEA (five studies; targets of 15–30 kcal/kg FFM/day for 3–9 days with food provided; adequate-EA comparators, where present, targeted 40–45 kcal/kg FFM/day); 16:8 TRE without a prescribed deficit (three studies; 4–8 weeks); and Ramadan fasting (five studies; approximately one month of daytime food and fluid abstinence with altered meal timing and sleep, two with non-fasting control groups [30,31]). Participants spanned recreational to world-class level, corresponding approximately to Tiers 1–5 of the participant-classification framework [20]; no female participants were reported in the Ramadan studies. Protein intake was 2.0–2.1 g/kg/day in Huovinen et al. [32], Burke et al. [2] and Kuikman et al. [33]; elsewhere it varied or was unreported. In Brisswalter et al. [30], reported energy intake did not change significantly in either group.

3.3. Quality Appraisal

Qualitative CASP appraisal rated overall methodological quality as high in one study, moderate in eleven and low in four (Table 2), with recurring limitations in allocation concealment, blinding, self-selected group formation and the reporting of estimate precision. The four studies rated as low quality were uncontrolled pre–post designs or lacked non-fasting comparators. Risk-of-bias assessment of the randomised studies rated one parallel-group trial at low and two at high risk of bias and of the six crossover studies three at low risk and three with some concerns; the main concerns arose from the randomisation process and missing outcome data (Supplementary File S5).

Table 1. Study characteristics, interventions and selected physiological outcomes.

Study/Report	Design and Timing	Exposure Category	Participants	Dietary Intervention; Comparator; Training	Measurement Methods/Outcome Domains	Key Findings
Huovinen et al., 2015 [32]	Randomised parallel-group trial; 4 weeks.	Prescribed energy deficit	15 male national/international sprinters and jumpers, aged 20–35 y, with ≥5 years of competitive experience. Low weight reduction (LWR), n = 7; high weight reduction (HWR), n = 8. CMJ data were available for 7/6 participants and sprint data for 5/7 participants in the LWR/HWR groups, respectively.	HWR: target deficit 750 kcal/d; LWR: 300 kcal/d. Carbohydrate and fat reduced; protein maintained at ~2.1 g/kg/d. Habitual training monitored.	DXA; body composition, sprint/jump performance, endocrine and blood chemistry.	HWR produced larger observed BM and FM losses than LWR, but no between-arm performance superiority was demonstrated. No significant changes in testosterone, cortisol or SHBG; baseline cortisol was higher in HWR. DXA bone mass showed no significant change. Bicarbonate decreased in both groups (HWR ~19%; LWR ~13%); pH and ionised calcium decreased in HWR. Haemoglobin did not change significantly.
Toyoshima et al., 2005 [34]	Uncontrolled pre–post; 4 weeks; measurements before, at week 2 and at week 4.	Dietary weight loss	6 male collegiate relay runners; age: 19.0 ± 0.9 y; VO_2max: 66.9 ± 8.1 mL/kg/min; body fat: 18.2%. Complete dietary data: n = 4.	Target: 5% BM reduction over 4 weeks; dietitian-arranged meals, with water only between meals. Recorded EI: 2096→1266 kcal/d and protein 81.6→40.3 g/d by week 4 (complete records: n = 4). Earlier EI differences non-significant; earlier deficit is not excluded. Usual training continued. Comparator: baseline.	BIS; body composition, treadmill capacity, knee strength and blood chemistry.	BM, FM and FFM decreased, while treadmill capacity showed no significant change and isometric knee-flexion strength declined. Total body water decreased by 1.3 L (intracellular −0.9 L); parallel FFM and water changes support, but do not establish, a fluid-shift explanation. Osmolality and blood urea nitrogen increased. Haemoglobin decreased at week 2 but did not differ significantly from baseline at week 4.

Table 1. *Cont.*

Study/Report	Design and Timing	Exposure Category	Participants	Dietary Intervention; Comparator; Training	Measurement Methods/Outcome Domains	Key Findings
Zanker & Swaine, 2000 [35]	Randomised crossover; two 3-day restriction/exercise periods, 2 weeks apart; each within a 5-day measurement protocol.	Prescribed energy deficit	8 trained male distance runners; age: 25.1 ± 5.9 y; ≥4 years training and ≥50 km/wk; VO_2max: 61.8 ± 4.9 mL/kg/min.	EI: ~50% versus ~100% of estimated requirement. Identical 60 min treadmill runs on 3 days at alternating 65%, 75% and 85% VO_2max. Protein prescribed as 15% of energy; g/kg/d not reported.	BIA and skinfolds at baseline only; bone turnover and endocrine.	No performance test was included; bone formation and IGF-1 declined after restriction but not after energy balance. P1NP decreased by 15% ($p = 0.008$) and IGF-1 by 17% ($p = 0.007$); declines correlated ($r = 0.97$, $p < 0.001$). Osteocalcin and urinary NTx/Dpd showed no significant changes. Blood n = 8; urinary outcomes n = 6.
Kojima et al., 2020 [a] [22]	Randomised crossover; two 3-day dietary/exercise periods, with testing on day 4; 2-week washout.	Controlled LEA	7 trained male university distance runners (8 recruited; 1 injury withdrawal); age: 19.9 ± 1.1 y; VO_2max: 67.5 ± 4.3 mL/kg/min; training 2 h/d, 6 d/wk.	LEA: target 15, achieved 17.9–19.5 kcal/kg FFM/d; NEA: target 45, achieved 51.7–54.7. Supervised running 75 min/d at 70% VO_2max; meals provided. Protein 20–22% of energy in LEA versus ~16% in NEA.	BIA (InBody770); glycogen by ^{13}C-MRS; endurance capacity, endocrine markers, resting gas exchange and fatigue. RMR analysis n = 6.	LEA reduced BM and FFM, with no significant change in FM; day-4 endurance capacity did not differ significantly between LEA and NEA. Muscle glycogen fell by ~30% in LEA and was lower than in NEA. IGF-1 was lower in LEA on day 4 (trial $p = 0.002$); free testosterone decreased within LEA without a significant trial difference; creatine kinase decreased in both trials. RER decreased in LEA; RMR showed no significant trial, time or interaction effects. Fatigue increased within LEA; urinary 3-MH/8-OHdG showed no significant effects.

Table 1. *Cont.*

Study/Report	Design and Timing	Exposure Category	Participants	Dietary Intervention; Comparator; Training	Measurement Methods/Outcome Domains	Key Findings
Ishibashi et al., 2020 [a] [23]	Randomised crossover; 3 training days plus day-4 measurements; ≥1-week washout. Companion report grouped with Kojima et al. [22]; participant overlap presumed, not explicitly confirmed.	Controlled LEA	6 trained male distance runners; age: 19.8 ± 0.4 y and VO_2max: 67.9 ± 1.5 mL/kg/min (mean ± SE); 5000 m performance 15:18 ± 0:18; training 4 h/d, 6 d/wk.	LEA: target < 20, achieved 17.3–19.1; NEA: target > 45, achieved 51.2–54.7 kcal/kg FFM/d. Supervised running 75 min/d at 70% VO_2max and meals provided.	BIA (InBody720); glycogen by ^{13}C-MRS; iron regulation and inflammation.	Iron-regulatory biomarkers shifted under LEA; the companion report found no significant LEA–NEA difference in endurance capacity. Hepcidin AUC was higher in LEA than NEA across days 1–4 ($p = 0.047$; d = 1.25); resting hepcidin showed no interaction ($p = 0.337$). Ferritin increased from 35.2 to 56.6 ng/mL in LEA and remained stable in NEA (interaction $p = 0.010$). Post-exercise IL-6 showed an interaction ($p = 0.039$); post-exercise hepcidin did not.
Sim et al., 2024 [36]	Randomised crossover; three 4-day conditions; ≥10-day washout.	Controlled LEA	12 trained male runners (17 recruited; 5 withdrew); age: 25.8 ± 3.8 y; VO_2max: 52.2 ± 0.4 mL/kg/min; training: 28.8 ± 9.1 km/wk.	EA targets: 15 (LEA2), 30 (LEA1) and 45 (AEA) kcal/kg FFM/d. Standardised running at 65% VO_2max; EEE: 15 kcal/kg FFM/d. Meals provided; macronutrient distribution: ~50% carbohydrate, 30% fat and 20% protein.	BIA; body composition, RMR, bone turnover, endocrine and mood.	No performance test was included; testosterone decreased in LEA2 (interaction $p = 0.03$). Percentage-change analysis showed greater testosterone decreases in both LEA conditions than AEA ($p = 0.009$). IGF-1, insulin, leptin, glucose and P1NP decreased over time, with no EA × time interactions. RMR and β-CTX showed no interactions ($p = 0.26$ and $p = 0.78$, respectively). Fatigue increased in LEA2 (interaction $p = 0.012$).

Table 1. *Cont.*

Study/Report	Design and Timing	Exposure Category	Participants	Dietary Intervention; Comparator; Training	Measurement Methods/Outcome Domains	Key Findings
Sterringer et al., 2026 [37]	Randomised crossover of running with/without jumping during LEA; two 5-day conditions, ≥1 menstrual cycle apart.	Controlled LEA	14 female athletes randomised: 7 Tier 1 and 7 Tier 2; 12 analysed. Condition-specific samples: running without jumping (RUN), n = 10; running with jumping (RUN + J), n = 12. Age: 26.4 ± 3.9 y; VO_2max: 45.3 ± 7.2 mL/kg/min; training: 4.3 ± 1.3 d/wk.	EA target: 15 kcal/kg FFM/d (provided EI: 30; prescribed EEE: 15 kcal/kg FFM/d). Running ~10 km/d at 65–70% VO_2max; RUN + J added 50 jumps, with running adjusted to match EEE. Protein: 1.1 ± 0.1 g/kg/d. No adequate-EA control; LEA effects are based on pre–post comparisons.	DXA at screening only; body mass, bone turnover and endocrine. Running economy measured but results not reported in the article.	BM decreased similarly with and without added jumping; no direct performance outcome was reported. β-CTX increased over time ($p = 0.005$), whereas P1NP and sclerostin showed no significant changes ($p = 0.517$ and $p = 0.091$, respectively); no condition × time interaction supported an added-jumping effect. fT3 decreased over time ($p = 0.007$), with a significant within-group reduction only in RUN. fT4, IGF-1, leptin and insulin decreased, whereas rT3 increased. PTH, cortisol and estradiol showed no significant changes.
Burke et al., 2023 [b] [2]	Non-randomised controlled parallel trial in two camps; 6-day baseline then 9-day adaptation. Allocation incorporated dietary preference and matching.	Controlled LEA	23 race walkers, Tier 3–5 (20 men, 3 women); HCHO n = 12, LEA n = 11. Age 25.5 ± 6.9 versus 29.3 ± 4.5 y. LEA had faster baseline personal-best times. An unrelated injury reduced follow-up VO_2peak/race testing to n = 22.	LEA: target 15, achieved 15 ± 2; HCHO target: 40, achieved 43 ± 4 kcal/kg FFM/d. Protein ~2.0–2.1 g/kg/d; EEE ~1300 kcal/d. Each race followed 24 h carbohydrate loading (8 g/kg) plus a 2 g/kg pre-race meal.	DXA; body composition, race performance, economy, substrate use and well-being.	LEA produced greater body-mass and fat loss but no additional performance benefit. Post-race RPE increased in LEA relative to HCHO ($p = 0.049$). RER and heart rate decreased more in LEA (both $p < 0.001$), with increased fat oxidation. Stress increased and recovery decreased only in LEA.

Table 1. *Cont.*

Study/Report	Design and Timing	Exposure Category	Participants	Dietary Intervention; Comparator; Training	Measurement Methods/Outcome Domains	Key Findings
Fensham et al., 2022 [b] [14]	Non-randomised parallel report from the same camps; outcomes after 6 days of adaptation.	Controlled LEA	28 male race walkers, Tiers 3–5: CON, n = 10; LEA, n = 10; LCHF, n = 8. The reporting sample differs from the mixed-sex cohort of Burke et al. [2]. Median age (IQR): CON, 27 (21–33) y; LEA, 30 (28–33) y. VO_2max: CON, 63.2 ± 3.6; LEA, 62.1 ± 6.1 mL/kg/min.	EA: ~15 (LEA) versus ~40 (CON) kcal/kg FFM/d; protein: ~2.1 (LEA) versus 2.3 (CON) g/kg/d. An additional ketogenic LCHF arm had high EA. Fasted and exercise-associated blood samples were collected around a 25 km walk with condition-specific feeding.	DXA baseline BMD; fasting and exercise-associated bone-turnover markers.	Bone formation declined under LEA while exercise-associated resorption rose; performance for this cohort is reported under Burke et al. [2]. Fasting P1NP decreased by ~14% ($p = 0.02$) and undercarboxylated osteocalcin by ~24% ($p = 0.049$); P1NP decreased more than in CON ($p < 0.001$). Fasting CTX increased across groups without a group difference. Exercise-associated CTX AUC increased by 30.9% and undercarboxylated osteocalcin AUC decreased by 19.8% in LEA (both $p = 0.01$); no exercise-associated AUCs changed in CON.
McKay, Peeling et al., 2022 [b] [15]	Non-randomised parallel report from the same camps; 6-day adaptation and a paced exercise protocol.	Controlled LEA	Same cohort as reported by Fensham et al. [14]: CON, n = 10; LEA, n = 10; LCHF, n = 8. Athlete calibre: Tier 5, n = 4; Tier 4, n = 20; Tier 3, n = 4. Age: CON, 26.7 ± 7.0 y; LEA, 30.0 ± 4.0.	EA: ~15 (LEA) versus ~40 (CON) kcal/kg FFM/d; dietary iron: 16.2 (LEA) versus 23.9 (CON) mg/d. Additional ketogenic LCHF arm with high EA. Diets and training monitored. Exercise protocol: 25 km hybrid treadmill/field walk at ~75% VO_2max, not a maximal time trial.	DXA used for EA calculation; iron regulation, blood cells, inflammatory/stress responses and lipids.	Performance for this cohort is reported under Burke et al. [2]. No significant LEA–CON differences were detected in iron-regulatory, immune or exercise-stress responses. Hepcidin increased 3 h post-exercise across groups. Testosterone decreased over time without a significant group difference. Total cholesterol and LDL decreased within LEA.

Table 1. *Cont.*

Study/Report	Design and Timing	Exposure Category	Participants	Dietary Intervention; Comparator; Training	Measurement Methods/Outcome Domains	Key Findings
Kuikman et al., 2025 [33]	Non-randomised controlled parallel trial: 7-day EA manipulation after ~2 weeks at altitude; whole camp 3 weeks.	Controlled LEA	20 female race walkers recruited; 19 analysed (LEA n = 10; HEA n = 9). Tier 3–5. Recruited age 26.5 ± 6.5 y; VO_2max: 58.2 ± 4.2 mL/kg/min. One injury exclusion before manipulation.	LEA: target 15, achieved 15.1 ± 0.6; HEA: target 45, achieved 45.1 ± 1.0 kcal/kg FFM/d. Conducted at ~1800 m altitude, with individualised training and provided meals adjusted to actual EEE. Protein 2.0 g/kg BM/d in both arms. Preference-based allocation with strategic balancing.	GE Lunar iDXA before/after the camp; endocrine and lipid indicators. RMR by indirect calorimetry (ParvoMedics TrueOne 2400)	BM decreased more in LEA across the full DXA interval; FM and FFM interactions were not significant. No significant 7-day LEA effect on absolute or FFM-relative RMR was detected ($p = 0.347$ and $p = 0.547$, respectively); no athlete met the study's suppressed-RMR thresholds. Within LEA, T3 was lower 36 h after descent than before altitude ($p = 0.002$) and after 2 weeks at altitude ($p = 0.025$); cortisol was higher after 2 weeks at altitude and 36 h after descent. IGF-1, insulin and LDL showed interactions without significant post hoc contrasts; total cholesterol decreased in both groups before the manipulation ($p = 0.041$).
Tovar et al., 2021 [c] [38]	Randomised crossover; two 4-week conditions; washout 2.1 ± 0.7 weeks.	TRE	15 male endurance runners analysed. Age: 28.7 ± 5.2 y; VO_2peak: 55.5 ± 5.7 mL/kg/min; training: ≥32 km/wk; competition history: ≥1 race of ≥5 km in the previous year.	16:8 TRE versus 12:12 normal diet. Energy and macronutrient intake intended to match habitual intake; same self-selected training. Recorded EI 2421 versus 2513 kcal/d; protein ~1.6 g/kg/d in both conditions.	DXA; body composition, 10 km time trial and exercise substrate utilisation.	FM and body-fat percentage decreased with TRE, although body-fat percentage showed a sequence effect. Exercise VCO_2 decreased with TRE ($p \leq 0.01$), although a carry-over effect limited interpretation; lactate was lower at 90% VO_2peak ($p = 0.03$; diet × intensity $p = 0.02$). RER and substrate oxidation showed no significant diet effect.

Table 1. *Cont.*

Study/Report	Design and Timing	Exposure Category	Participants	Dietary Intervention; Comparator; Training	Measurement Methods/Outcome Domains	Key Findings
Richardson et al., 2023 [c] [39]	Companion report of the Tovar et al. [38] crossover experiment.	TRE	Same 15 participants as in Tovar et al. [38]. VO_2max: 55.2 ± 6.7 mL/kg/min; body fat: 16.0 ± 5.6%.	Same as reported by Tovar et al. [38]; 4 weeks per condition.	DXA; segmental composition, BMD, resting metabolism, glucose/insulin, lipids and blood pressure.	Leg FM decreased more with TRE, whereas arm and trunk FM did not differ; no additional performance outcome was reported. BMD, resting energy expenditure, fasting glucose and insulin, HOMA-IR, QUICKI, lipids and blood pressure showed no significant diet differences.
Correia et al., 2024 [40]	Randomised counterbalanced crossover; two 30-day conditions; 2-week washout.	TRE	16 male recreational runners enrolled; 15 analysed. Age: 23.7 ± 2.6 y; running experience: ≥3 years; training: >3 sessions/wk and 15–30 km/wk. Pre-TRE VO_2max: 61.6 ± 6.0 mL/kg/min.	Ad libitum 16:8 TRE (13:00–21:00) versus habitual diet. Supervised running: 30 km/wk in weeks 1–2, ~39 km/wk in weeks 3–4. Dietary records only before each condition; achieved EI and protein intake during intervention are unknown.	DXA; body composition, aerobic determinants and metabolic markers.	Aerobic determinants improved over time without an additional TRE effect. No significant between-condition body-composition difference was detected. Fasting glucose and measured lipids did not differ significantly between conditions.
Brady et al., 2021 [41]	Randomised parallel trial; 8 weeks. Post-intervention analyses were blinded to allocation.	TRE	28 runners recruited; 23 randomised; 17 analysed (TRE, n = 10; CON, n = 7). Age: TRE, 35.9 ± 8.6 y; CON, 39.9 ± 3.0 y. All competed in events ≥1500 m and had trained ≥5 d/wk for ≥24 months.	Ad libitum 16:8 TRE, typically 12:00–20:00, versus habitual diet; habitual training monitored. Protein 1.4–1.7 g/kg/d. Recorded EI reduction in TRE: 394 ± 228 kcal/d at week 4 and 265 ± 606 kcal/d at week 8; neither was a prescribed deficit.	BIA; body composition, aerobic determinants, glucose/insulin and triglycerides.	TRE reduced BM relative to CON; adjusted FM and FFM contrasts were not significant. Aerobic capacity, running economy, RER, submaximal lactate, fasting glucose, insulin, HOMA-IR and triglycerides showed no significant adjusted between-group differences.

Table 1. *Cont.*

Study/Report	Design and Timing	Exposure Category	Participants	Dietary Intervention; Comparator; Training	Measurement Methods/Outcome Domains	Key Findings
Chennaoui et al., 2009 [42]	Uncontrolled prospective observational pre–post study during a 30-day Ramadan fast.	Ramadan fasting	8 male middle-distance runners; age: 25.0 ± 1.3 y (mean ± SEM); training: 6–10 sessions/wk for ≥3 years.	Ramadan fasting (~13 h/d), with no prescribed energy deficit; habitual training continued. MAV assessed 5 days before and on days 7 and 21 of Ramadan; BM on days 0, 7, 21 and 31; blood before, at the end of and 1 week after Ramadan. Sleep patterns, food and fluid intake changed concurrently.	Skinfolds; body mass/fat percentage, maximal aerobic velocity, endocrine, inflammation, sleep and mood.	MAV decreased during Ramadan without significant BM change. Energy intake and nocturnal sleep decreased; fatigue increased. IL-6, catecholamines and free fatty acids increased; melatonin decreased narratively. Free fatty acids remained elevated 1 week later, despite the abstract's general statement of recovery. Performance was not retested after Ramadan.
Bouguerra et al., 2017 [26]	Prospective repeated-measures study with randomised training-time allocation; before, midway through and after Ramadan during a 6-week programme.	Ramadan fasting	24 elite male middle- and long-distance runners from Tunisian national civilian and military teams. Afternoon (AG), morning (MG) and evening (EG) training groups: n = 8 each. Age: 28.5 ± 10.6 y	Ramadan fasting, including abstinence from food and fluids. Training times: AG, 14:00–16:00; MG, 09:00–11:00; EG, 22:00–24:00. All groups followed the same programme (6 sessions/wk) and meal schedule. EI, EA and protein intake were not quantified. No non-fasting comparator.	Four skinfolds; BM and fat percentage. Track 3000 m time, MAV, VO_2max, tlim100 and dlim100. FFM calculation described but results absent.	The 3000 m time increased midway through Ramadan; the pre- to post-Ramadan difference was not significant. After Ramadan, TTE increased with afternoon training and decreased with evening training. BM decreased in morning and afternoon groups, while body-fat percentage decreased across groups.

Table 1. *Cont.*

Study/Report	Design and Timing	Exposure Category	Participants	Dietary Intervention; Comparator; Training	Measurement Methods/Outcome Domains	Key Findings
Havenetidis, 2015 [31]	Non-randomised controlled parallel study; four-week training block; pre/post tests in weeks 1 and 6.	Ramadan fasting	20 male 3000 m runners from an army academy athletics team, ≥2 years of training. Fasting, n = 10; non-fasting, n = 10. Age: fasting, 21.7 ± 2.0 y; non-fasting, 21.4 ± 1.7 y. Baseline 3 km time: fasting, 565 ± 29 s; non-fasting, 577 ± 22 s. Groups were determined by religious affiliation.	Ramadan versus non-fasting; identical supervised running, strength and callisthenics training: 4 d/wk, 2 sessions/d (34 sessions reported). No prescribed deficit; compensatory dinner intake allowed. Protein intake 1.5 → 2.0 g/kg/d in the fasting group and 1.7 → 1.8 g/kg/d in the non-fasting group.	Body mass only; shuttle-test predicted VO_2max, HR, training, RPE and TQR.	Shuttle-run-predicted VO_2max improved in both groups, with no significant group difference, and fasting produced no clear between-group BM effect. TQR was lower during fasting (14.5 ± 1.4 versus 15.7 ± 1.3; group $p = 0.004$; group × week $p = 0.001$), chiefly in week 2; HR, RPE and training volume and intensity did not differ.
Brisswalter et al., 2011 [30]	Non-randomised controlled study; testing in the week before Ramadan and the last week of fasting. Four tests over 3 days at each assessment; same test order and individual testing time of day.	Ramadan fasting	18 well-trained Muslim runners regularly competing at 1500, 5000 or 10,000 m; fasting/control n = 9 each. Age 23.6 ± 2.9 y; training and living in France. Allocation followed religious practice.	No prescribed deficit. Seven-day food records: reported EI unchanged over time; fasting/control 135.2 ± 8.4/140.5 ± 6.2 kJ/kg/d, respectively (not pre/post values). Protein dose NR. Estimated water intake ~1.2 L/d in both groups. Standardised training: 3 sessions/wk.	BM (force platform), four-site skinfolds; 5000 m track time trial; maximal voluntary isometric knee extension and EMG; VO_2max, speed at VO_2max, ventilatory thresholds and submaximal VO_2 kinetics.	Significant group × period interactions: slower 5000 m performance, lower knee-extension MVC and speed at VO_2max, and a longer VO_2 time constant in fasting. Authors report −5% performance, −3.2% MVC and +51% time constant. No significant BM, fat %, VO_2max or running-efficiency effects. Source discrepancies are described in Table 3, note k.

Table 1. *Cont.*

Study/Report	Design and Timing	Exposure Category	Participants	Dietary Intervention; Comparator; Training	Measurement Methods/Outcome Domains	Key Findings
Al-Nawaiseh et al., 2021 [43]	Uncontrolled prospective pre–post (observational); testing 3 d before Ramadan and in the last week of fasting, at the same time of day. Comparator: baseline only.	Ramadan fasting	15 trained male distance runners; age 23.9 ± 3.1 y; VO_2peak 71.1 ± 3.4 mL/kg/min; running ≥150 min/d on ≥5 d/wk.	Ramadan fasting ≥15 h/d for approximately one month; no dietary prescription. Recorded EI 2990→2523 kcal/d ($p = 0.002$); carbohydrate, lipid and water intakes decreased; protein intake unchanged ($p = 0.124$). Training not prescribed; daily training duration and exercise energy expenditure decreased.	BIA (InBody720); digital-scale BM; food records; treadmill GXT (Conconi protocol): VO_2, time to exhaustion, maximal speed, HR, RPE; sleep diary.	Body mass, fat mass and lean body mass unchanged. Time to exhaustion and maximal running speed improved in the last week of fasting ($p = 0.049/0.048$); VO_2max, energy availability and sleep duration unchanged; heart rate lower at submaximal speeds.

Note. Numerical body-composition and performance outcomes are reported in Table 3. Superscript letters identify reports from the same study: [a] Kojima et al. [22] and Ishibashi et al. [23]; [b] Burke et al. [2], Fensham et al. [14] and McKay, Peeling et al. [15]; [c] Tovar et al. [38] and Richardson et al. [39]. Abbreviations. 3-MH: 3-methylhistidine; 8-OHdG: 8-hydroxy-2′-deoxyguanosine; ^{13}C-MRS: carbon-13 magnetic resonance spectroscopy; AEA: adequate energy availability; AG/MG/EG: afternoon/morning/evening training groups; AUC: area under the curve; BIA: bioelectrical impedance analysis; BIS: bioimpedance spectroscopy; BM: body mass; BMD: bone mineral density; CI: confidence interval; EMG: electromyography; MVC: maximal voluntary contraction; TTE: time to exhaustion; CMJ: countermovement jump; CON: control; CTX/β-CTX: C-terminal/beta-isomerised C-terminal telopeptide of type I collagen; Dpd: deoxypyridinoline; DXA: dual-energy X-ray absorptiometry; EA: energy availability; EEE: exercise energy expenditure; EI: energy intake; FFM: fat-free mass; FM: fat mass; fT3/fT4: free triiodothyronine/free thyroxine; GXT: graded exercise test; HCHO: high energy/carbohydrate availability; HEA: high energy availability; HOMA-IR: homeostatic model assessment of insulin resistance; HR: heart rate; IGF-1: insulin-like growth factor 1; IL-6: interleukin-6; IQR: interquartile range; LCHF: low-carbohydrate, high-fat diet; LDL: low-density lipoprotein cholesterol; LEA: low energy availability; MAV: maximal aerobic velocity; NEA: normal energy availability; NTx: cross-linked N-telopeptide of type I collagen; P1NP: N-terminal propeptide of type I procollagen; PTH: parathyroid hormone; QUICKI: quantitative insulin-sensitivity check index; RER: respiratory exchange ratio; RMR: resting metabolic rate; RPE: rating of perceived exertion; rT3: reverse triiodothyronine; RUN/RUN + J: running without/with additional jumping; SD: standard deviation; SE: standard error; SEM: standard error of the mean; SHBG: sex hormone-binding globulin; T3: triiodothyronine; tlim100/dlim100: time to exhaustion/distance completed at 100% MAV; TQR: Total Quality Recovery scale; TRE: time-restricted eating; VCO_2: carbon dioxide production; VO_2max/VO_2peak: maximal/peak oxygen uptake; VT1/VT2: first/second ventilatory thresholds.

Table 2. Critical appraisal of the 16 included studies (20 reports) using the Critical Appraisal Skills Programme (CASP) checklists: (**a**) studies appraised with the CASP Checklist for Randomised Controlled Trials (RCTs); (**b**) studies appraised with the CASP Checklist for Cohort Studies.

(a) CASP Checklist for Randomised Controlled Trials (RCTs)

	Authors	1	2	3	4a	4b	4c	5	6	7	8	9	10	11	Assessment
1	Huovinen et al., 2015 [32]	Y	Y	Y	U	U	U	U	Y	Y	N	U	Y	U	Moderate
2	Zanker & Swaine, 2000 [35]	Y	Y	U	U	U	U	Y	Y	Y	N	U	Y	N	Moderate
3	Kojima et al., 2020 [22] and Ishibashi et al., 2020 [23]	Y	Y	U	U	U	U	Y	Y	U	N	U	Y	U	Moderate
4	Sim et al., 2024 [36]	Y	Y	U	N	N	U	Y	Y	Y	N	U	Y	U	Moderate
5	Sterringer et al., 2026 [37]	Y	Y	U	N	N	U	N	Y	U	N	U	Y	N	Moderate
6	Tovar et al., 2021 [38] and Richardson et al., 2023 [39]	Y	Y	U	N	N	U	U	Y	U	N	U	Y	U	Moderate
7	Correia et al., 2024 [40]	Y	Y	Y	N	N	U	Y	Y	U	U	U	Y	N	Moderate
8	Brady et al., 2021 [41]	Y	Y	U	N	N	Y	U	Y	Y	Y	U	Y	U	Moderate
9	Bouguerra et al., 2017 [26]	Y	Y	U	N	N	U	N	U	N	N	U	Y	U	Low

(b) CASP Checklist for Cohort Studies

	Authors	1	2	3	4	5a	5b	6a	6b	7	8	9	10	11	12	Assessment
1	Toyoshima et al., 2005 [34]	Y	U	N	U	N	N	U	Y	Y	N	U	Y	Y	U	Low
2	Burke et al., 2023 [2], Fensham et al., 2022 [14] and McKay, Peeling et al., 2022 [15]	Y	U	Y	Y	U	Y	Y	U	Y	U	Y	Y	Y	Y	High
3	Kuikman et al., 2025 [33]	Y	U	Y	Y	Y	U	Y	U	Y	N	Y	Y	Y	Y	Moderate
4	Chennaoui et al., 2009 [42]	Y	U	U	Y	U	N	Y	Y	U	N	U	Y	Y	U	Low
5	Havenetidis, 2015 [31]	Y	U	Y	Y	Y	U	Y	Y	Y	N	Y	Y	Y	Y	Moderate
6	Brisswalter et al., 2011 [30]	Y	U	Y	Y	Y	U	Y	Y	Y	N	Y	Y	Y	Y	Moderate
7	Al-Nawaiseh et al., 2021 [43]	Y	U	U	Y	U	N	U	Y	Y	U	U	Y	Y	U	Low

CASP criteria for randomised controlled trials: 1. Did the study address a clearly formulated research question?; 2. Was the assignment of participants to interventions randomised?; 3. Were all participants who entered the study accounted for at its conclusion?; 4a. Were the participants 'blind' to intervention they were given?; 4b. Were the investigators 'blind' to the intervention they were giving to participants?; 4c. Were the people assessing/analysing outcome/s 'blinded'?; 5. Were the study groups similar at the start of the randomised controlled trial?; 6. Apart from the experimental intervention, did each study group receive the same level of care (that is, were they treated equally)?; 7. Were the effects of intervention reported comprehensively?; 8. Was the precision of the estimate of the intervention or treatment effect reported?; 9. Do the benefits of the experimental intervention outweigh the harms and costs?; 10. Can the results be applied to your local population/in your context?; 11. Would the experimental intervention provide greater value to the people in your care than any of the existing interventions? (Y Yes, N No, U Cannot tell). Items 4a and 4b are N where the intervention was necessarily transparent to those involved and U where blinding was merely undescribed [25]. CASP criteria for cohort studies: 1. Did the study address a clearly focused issue?; 2. Was the cohort recruited in an acceptable way?; 3. Was the exposure accurately measured to minimise bias?; 4. Was the outcome accurately measured to minimise bias?; 5a. Have the authors identified all important confounding factors?; 5b. Have they taken account of the confounding factors in the design and/or analysis?; 6a. Was the follow-up of subjects complete enough?; 6b. Was the follow-up of subjects long enough?; 7. What are the results of this study?; 8. How precise are the results?; 9. Do you believe the results?; 10. Can the results be applied to the local population?; 11. Do the results of this study fit with other available evidence?; 12. What are the implications of this study for practice? (Y Yes, N No, U Cannot tell; [24]). Judgements were qualitative, with no summary scores calculated.

Table 3. Body-composition and performance findings.

Study/Condition	Exposure (d)/ BM Interval [d]	Protein (g/kg/d) [f]	Comparator/Contrast	Δ BM (kg)	Weekly BM Change (%) [d]	Δ FM (kg)	Δ FFM/Lean (kg) [e]	Performance and Exercise-Capacity Findings
Huovinen et al., 2015, HWR [32]	28	2.1	LWR; target deficits 750 versus 300 kcal/d.	−2.2 ± 1.0	−0.68	−1.7 ± 1.6	−0.5 ± 1.2 (NS)	Within HWR, CMJ increased by 2.6 ± 2.5 cm (n = 6) and flying 20 m time decreased by 0.04 ± 0.04 s (n = 7; both $p \leq 0.05$). Between-arm performance superiority was not demonstrated.
Huovinen et al., 2015, LWR [32]	28	2.1	HWR; same study, not an independent experiment.	−0.4 ± 1.2 (NS)	−0.13	−0.2 (NS)	−0.2 (NS)	CMJ changed from 53 to 55 cm (n = 7) and flying 20 m time from 2.13 to 2.10 s (n = 5); neither change was significant.
Toyoshima et al., 2005 [34]	28	NR; 81.6 to 40.3 g/d (week 4; n = 4)	Baseline only; 5% body-mass reduction target.	−2.4	−1.03	−1.1	−1.3	Absolute/relative VO_2max and incremental treadmill time: no significant changes. Isometric knee flexion decreased ($p < 0.05$); other strength measures did not.
Zanker & Swaine, 2000 [35]	3	NR; 15% of energy	Energy-balanced condition versus ~50% of requirement.	Not measured	—	Not measured	Not measured	No performance test or longitudinal body-composition result.
Kojima et al., 2020/Ishibashi et al., 2020 [a] [22,23]	3	~1.8 (Kojima et al. [22]; estimate) [f]	Normal EA. Body-composition values here are those of Kojima et al. [22].	LEA −1.2; NEA +0.3 (interaction $p < 0.001$)	−4.52 (LEA)	LEA −0.2; NEA −0.3 ($p = 0.33$)	LEA −1.0; NEA +0.5 ($p = 0.01$)	Day-4 time to exhaustion: LEA 1170 ± 312 versus NEA 1361 ± 480 s ($p = 0.39$), not a pre–post change.
Sim et al., 2024, LEA2 [36]	4	~1.3 [f]	AEA 45 kcal/kg FFM/d; LEA2 target 15.	LEA2 −1.3; AEA −0.2 (interaction $p = 0.001$)	−3.78 (LEA2) [d]	LEA2 −0.4; AEA +0.1 ($p = 0.260$)	LEA2 −0.9; AEA +0.3 ($p = 0.134$)	No performance test.

Table 3. *Cont.*

Study/Condition	Exposure (d)/ BM Interval [d]	Protein (g/kg/d) [f]	Comparator/Contrast	Δ BM (kg)	Weekly BM Change (%) [d]	Δ FM (kg)	Δ FFM/Lean (kg) [e]	Performance and Exercise-Capacity Findings
Sim et al., 2024, LEA1 [36]	4	~1.9 [f]	AEA 45 kcal/kg FFM/d; LEA1 target 30. Same crossover participants.	LEA1 −0.7; AEA −0.2	−2.05 (LEA1) [d]	LEA1 −0.32; AEA +0.1	LEA1 −0.4; AEA +0.3	No performance outcome was assessed; this was the same crossover sample as LEA2.
Sterringer et al., 2026 [37]	5	1.1	RUN + J versus RUN tests the effect of added jumping; baseline is the comparator for the shared LEA exposure.	RUN −1.3 ± 0.7; RUN + J −1.4 ± 0.7 ($p = 0.651$)	—	Not measured after intervention	Not measured after intervention	Running economy was measured but not reported; no direct performance outcome was available.
Burke et al., 2023 [2]/Fensham et al., 2022 [14]/McKay, Peeling et al., 2022 [b] [15]	9 LEA; 6 for bone/iron	2.0–2.1	HCHO control. DXA interval includes baseline and adaptation, not only 9 days.	DXA: LEA −2.1; HCHO −0.9 ($p = 0.005$)	—	LEA −1.6; HCHO −0.9 [g] ($p = 0.009$)	LEA −0.4 ± 0.8; HCHO +0.03 ± 0.4 ($p = 0.131$)	Following 24 h carbohydrate refuelling, 10,000 m performance improved by 3.5 ± 1.8% in LEA and 4.5 ± 4.1% in HCHO (both $p < 0.001$), with no between-group difference; VO_2peak was unchanged. BM loss persisted after refuelling and was unrelated to performance change (r = −0.08 [−0.49 to 0.35], $p = 0.717$).
Kuikman et al., 2025 [j] [33]	7-day LEA; DXA across 3-week camp + return	2.0 in both arms	Concurrent HEA at ~1800 m; preference allocation.	LEA −0.89 ± 0.47; HEA −0.35 ± 0.61 (interaction $p = 0.030$)	—	LEA −1.00 ± 0.60; HEA −0.54 ± 0.57 ($p = 0.053$; time $p < 0.0001$)	FFM: LEA +0.12 ± 0.81; HEA +0.16 ± 0.65 ($p = 0.909$; time $p = 0.408$)	No longitudinal performance outcome was reported.

Table 3. *Cont.*

Study/Condition	Exposure (d)/ BM Interval [d]	Protein (g/kg/d) [f]	Comparator/Contrast	Δ BM (kg)	Weekly BM Change (%) [d]	Δ FM (kg)	Δ FFM/Lean (kg) [e]	Performance and Exercise-Capacity Findings
Tovar et al., 2021 [38]/Richardson et al., 2023 [c] [39]	28	1.6	Normal diet (ND), intended energy/macronutrient match.	TRE −0.8 ± 1.9; ND +0.3 ± 1.1 ($p = 0.09$)	−0.27 (TRE)	TRE −0.8 ± 1.3; ND +0.1 ± 4.3 ($p = 0.05$)	Lean: TRE +0.1 ± 1.7; ND +0.7 ± 2.4 ($p = 0.45$)	The 10 km time change was −20 ± 214 s with TRE and −36 ± 177 s with ND (diet $p = 0.53$). Body-fat percentage changed by −1.0 ± 1.5 versus +0.1 ± 1.3 percentage points with TRE and ND, respectively ($p = 0.04$). A sequence effect was detected for body-fat percentage ($p = 0.05$). Leg FM decreased more with TRE ($p = 0.03$); arm and trunk FM did not differ.
Correia et al., 2024 [40]	30	NR	Non-TRE + same training; baseline protein is not intervention intake.	TRE −0.5; non-TRE +0.5 (NS)	−0.15 (TRE)	TRE −0.4; non-TRE +0.3 (NS)	TRE −0.2; non-TRE +0.2 (NS)	No race or time trial was conducted. VO_2max increased in both TRE (61.6→63.7) and non-TRE (60.3→61.7 mL/kg/min; time $p = 0.044$). VT1, VT2 and velocity at VO_2max also increased over time ($p = 0.004$, $p = 0.019$ and $p = 0.002$, respectively), with no condition effects.

Table 3. *Cont.*

Study/Condition	Exposure (d)/ BM Interval [d]	Protein (g/kg/d) [f]	Comparator/Contrast	Δ BM (kg)	Weekly BM Change (%) [d]	Δ FM (kg)	Δ FFM/Lean (kg) [e]	Performance and Exercise-Capacity Findings
Brady et al., 2021 [41]	56	1.4–1.7	Habitual-diet CON. Raw changes are distinct from adjusted differences.	TRE −1.11; CON +0.77 (adjusted −1.92 kg, 95% CI −3.52 to −0.32; $p = 0.022$)	−0.19 (TRE)	TRE −0.67; CON −0.06 (adjusted −0.51, 95% CI −1.21 to 0.19; $p = 0.139$)	TRE −0.44; CON +0.78 (adjusted −1.15, 95% CI −2.46 to 0.16; $p = 0.081$)	No race or time trial was conducted. VO_2peak, speed and running economy at 2 mmol/L blood lactate, RER and submaximal lactate showed no significant adjusted between-group differences. The observed TRE loss comprised approximately 60% FM and 40% FFM.
Chennaoui et al., 2009 [42]	30-day fast; BM measured on days 0–31	NR	Baseline only. Energy intake lower on day 21, not a fixed 30-day deficit.	−1.5 (not significant)	−0.50 (days 0–31)	NR	NR	MAV decreased from 20.7 km/h before Ramadan to 20.1 km/h on Day 7 and 19.9 km/h on Day 21 (both $p < 0.05$; −3.9% by Day 21). The Day 7 decrement occurred after only 0.5 kg BM loss; body-fat percentage changed from 10.0% to 9.4% (NS). MAV was not retested after Ramadan.
Bouguerra et al., 2017, AG [h] [26]	Ramadan month; exact BM interval NR	NR	Other training times, all fasting; baseline for Ramadan.	−2.7 ($p < 0.001$)	—	NR in kg; fat % −1.2 pp	NR	The 3000 m time worsened midway through Ramadan but showed no significant pre- to post-Ramadan difference, with no training-time interaction. VO_2max decreased transiently midway through Ramadan ($p < 0.05$), also without an interaction. After Ramadan, tlim100 and dlim100 increased (both $p < 0.001$).

Table 3. *Cont.*

Study/Condition	Exposure (d)/ BM Interval [d]	Protein (g/kg/d) [f]	Comparator/Contrast	Δ BM (kg)	Weekly BM Change (%) [d]	Δ FM (kg)	Δ FFM/Lean (kg) [e]	Performance and Exercise-Capacity Findings
Bouguerra et al., 2017, MG [h] [26]	Ramadan month; exact BM interval NR	NR	Other training times, all fasting; baseline for Ramadan.	−1.5 ($p < 0.001$)	—	NR in kg; fat % −1.2 pp	NR	The 3000 m time worsened midway through Ramadan but showed no significant pre- to post-Ramadan difference, with no training-time interaction. MAV, VO_2max, tlim100 and dlim100 showed no significant changes.
Bouguerra et al., 2017, EG [h] [26]	Ramadan month; exact BM interval NR	NR	Other training times, all fasting; baseline for Ramadan.	−0.3 (NS)	—	NR in kg; fat % −1.1 pp	NR	The 3000 m time worsened midway through Ramadan but showed no significant pre- to post-Ramadan difference, with no training-time interaction. MAV and VO_2max decreased transiently midway through Ramadan (both $p < 0.05$), without significant interactions. After Ramadan, tlim100 decreased ($p < 0.01$) and dlim100 decreased ($p < 0.05$).
Havenetidis [i], 2015 [31]	30-day Ramadan; 4-week training; tests weeks 1/6	Fasting 1.5→2.0; control 1.7→1.8. Pre/post only.	Concurrent non-fasting group; non-random allocation by religion.	Fasting −1.0 (within $p = 0.012$); control change NR; group × time NS	—	NR	NR	Shuttle-run-predicted VO_2max increased from 59.8 ± 2.5 to 62.2 ± 1.6 mL/kg/min during fasting and from 59.3 ± 2.1 to 62.6 ± 2.0 mL/kg/min without fasting (time $p = 0.001$), with no significant group difference. The 3 km time was measured only at baseline.

Table 3. *Cont.*

Study/Condition	Exposure (d)/ BM Interval [d]	Protein (g/kg/d) [f]	Comparator/Contrast	Δ BM (kg)	Weekly BM Change (%) [d]	Δ FM (kg)	Δ FFM/Lean (kg) [e]	Performance and Exercise-Capacity Findings
Brisswalter et al., 2011, Ramadan fasting group [k] [30]	Ramadan month; exact interval NR/before Ramadan to last week of fasting	NR	Non-fasting control (n = 9); fasting n = 9	Fasting −0.8 (62.6→61.8); control −0.6 (65.2→64.6); both NS	—	NR in kg. Fat %: fasting 11.4→10.6; control 12.5→11.2; both NS	Not reported	5000 m time: fasting 14:18→15:03 versus control 14:23→14:21 min:s; significant group × period interaction. VO_2 time constant: 22.3→33.9 versus 23.1→21.7 s. Knee-extension MVC: 205.2→197.5 versus 211.3→210.6 (source units inconsistent). No significant change in reported EI, BM or fat %. See note k.
Al-Nawaiseh et al., 2021 [43]	3 d before Ramadan to the last week of fasting (approximately 4.5 weeks)	~1.9→~1.5	Baseline only (all participants fasted)	+0.3 (60.5→60.8); NS ($p = 0.201$)	—	+0.1 (9.0→9.1); NS ($p = 0.488$)	LBM +0.2 (51.5→51.7); NS ($p = 0.525$)	Time to exhaustion improved 1392.9→1448.4 s and maximal speed 18.6→19.7 km/h ($p = 0.049/0.048$); VO_2max unchanged ($p = 0.960$). EI 2989.7→2523.2 kcal/d ($p = 0.002$); carbohydrate, lipid and water intakes decreased; protein 118.1→88.6 g/d ($p = 0.124$). Daily training duration and exercise energy expenditure decreased; sleep duration unchanged.

Data conventions. Δ denotes within-arm post minus pre unless a between-condition or adjusted estimate is explicitly identified. Mean ± SD of change is shown only when reported; other differences are calculated from published group means. No SD, confidence interval or *p*-value was imputed. Rounding and differing source definitions can prevent body-composition components from summing exactly. [a–c] Linked reports from the same study are identified in Table 1. The Kojima et al. [22] report supplies the body-composition values shown for Kojima/Ishibashi; the reports are not pooled or counted twice. The Tovar et al. [38] report supplies the repeated whole-body composition and performance results for Tovar/Richardson. The three Bouguerra et al. [26] rows represent one study (n = 24).[d] Weekly equivalent. (ΔBM/matching pre-BM) × 100 × 7/elapsed days; this is descriptive rescaling, not an observed or sustainable weekly fat-loss rate. Sim et al. [36] values use the authors' reported mean percentage changes (−2.16% and −1.17%), not percentages recomputed from rounded means; the Chennaoui et al. [42] equivalent uses days 0–31. No equivalent is calculated where anthropometry exceeds the restriction interval (Burke et al. [2], Kuikman et al. [33]), anthropometric

timing is unspecified (Bouguerra et al. [26], Havenetidis [31], Brisswalter et al. [30]) or only screening BM is tabulated (Sterringer et al. [37]); no equivalent is calculated for Al-Nawaiseh et al. [43], where body mass did not decrease. [e] FFM/lean labels follow each source and are not interchangeable with muscle mass. The Huovinen et al. [32] "FFM" is tabulated separately from bone mass; the Tovar et al. [38] outcome is labelled "lean mass". The Kuikman et al. [33] FFM includes bone mineral; the authors' separately reported LBM excluding bone mineral changed near-identically (LEA +0.12 ± 0.81, HEA +0.16 ± 0.66 kg; interaction $p = 0.901$). [f] Units are g/kg BM/d unless otherwise stated. Kojima et al. [22] (~1.8), Sim et al. [36] (~1.3/~1.9) and Al-Nawaiseh et al. [43] (~1.9→~1.5) are reviewer estimates from reported energy/macronutrient or absolute protein values and BM, not reported mean individual intakes. Zanker & Swaine [35] report 15% of energy, not g/kg/d. The Toyoshima et al. [34] complete dietary series is n = 4; the Correia et al. [40] baseline dietary records do not measure intervention intake. Havenetidis [31] values are pre/post dietary assessments. [g] Values follow the main Results of Burke et al. [2] (DXA BM −2.1 kg, FM −1.6 kg in LEA); the source abstract gives BM −2.0 kg, rounded group means imply FM −1.5 kg, and scale BM decreased 2.0 kg. These source inconsistencies are noted, not reconciled. [h] BM changes derive from mean pre/post anthropometry; endpoint SEs are not SDs of change. Fat percentage fell 1.2/1.2/1.1 percentage points in AG/MG/EG (all $p < 0.001$), not kilograms. FM/FFM in kilograms were not reported. Numerical performance changes were not digitised from figures. The 3000 m group × phase interaction was non-significant; tlim100 was not assessed mid-Ramadan. Training-time × phase interactions were significant for tlim100 and dlim100 (both $p < 0.001$). [i] Four training weeks are not the exact BM measurement interval: testing is described in weeks 1 and 6. VO_2max was predicted from the 20 m shuttle test, not measured by gas exchange; raw shuttle time/distance and follow-up 3 km results were not reported. The source tabulates the post-dietary record under week 5, whereas the methods say week 6. The source reports baseline BM SD inconsistently (7.2 and 7.3). [j] DXA covers the interval from before altitude exposure to 36 h after descent, including acclimatisation, the 7 d dietary manipulation and return. Reported change means and SDs take precedence over rounded endpoint subtraction. The FM interaction ($p = 0.053$) does not establish greater LEA fat loss. [k] The source provides group means. Times printed as 14.18/15.03 under "min and sec" are shown as 14:18/15:03. The reported 3.2% MVC decrease differs from the ~3.8% implied by rounded means, force/torque units are inconsistent, and the VO_2 time-constant increase (22.3→33.9 s) is given as +51% in the results but +16% in the discussion; these discrepancies are retained. The second visit fell in the last week of fasting; with no exact elapsed-day denominator, no weekly equivalent is calculated. Abbreviations. MVC, maximal voluntary contraction; SD, standard deviation; LBM, lean body mass; ND, normal diet; pp, percentage points; NS, not statistically significant; NR, not reported for the relevant measurement; —, not calculated or not applicable. All other abbreviations are defined in Table 1.

3.4. Primary Outcomes: Performance and Exercise Capacity

No intervention demonstrated a between-condition performance benefit. Huovinen et al. [32] reported improved jumping and sprinting within the larger-deficit group, without between-group superiority. After refuelling, race walkers improved 10,000 m performance by 3.5 ± 1.8% after LEA and 4.5 ± 4.1% after higher energy availability [2]. Tovar et al. [38] detected no significant diet effect on 10 km time change, and Kojima et al. [22] reported time to exhaustion of 1170 versus 1361 s under low and normal energy availability ($p = 0.39$). During Ramadan, maximal aerobic velocity declined in an uncontrolled cohort [42], and in Brisswalter et al. [30], 5000 m time increased from 14:18 to 15:03 min:s during the last week of fasting, versus 14:23 to 14:21 in controls, with a significant group × period interaction. The authors reported a 3.2% decrease in maximal voluntary knee-extension performance. The oxygen-uptake time constant increased from 22.3 to 33.9 s and speed at maximal oxygen uptake decreased, without significant changes in maximal oxygen uptake or running efficiency. Bouguerra et al. [26] reported slower 3000 m performance mid-Ramadan without a significant pre–post difference; post-Ramadan time to exhaustion increased with afternoon training and decreased with evening training. In Al-Nawaiseh et al. [43], treadmill time to exhaustion and maximal running speed improved from before Ramadan to its final week ($p = 0.049$ and $p = 0.048$), without a non-fasting comparator and alongside significantly reduced daily training duration and exercise energy expenditure. Toyoshima et al. [34] reported reduced isometric knee-flexion strength after a four-week dietary weight-loss programme. Havenetidis [31] reported improved shuttle-run-predicted maximal oxygen uptake in both fasting and non-fasting runners, without a significant group difference.

3.5. Secondary Outcomes: Body Composition

Body mass was measured in 15 studies; within-condition changes and the composition of measured losses are summarised in Figure 2 and Table 3. Burke et al. [2] reported DXA-derived total-mass losses of 2.1 versus 0.9 kg and fat losses of 1.6 versus 0.9 kg in the LEA and higher-energy conditions. Brady et al. [41] reported an adjusted body-mass difference of −1.92 kg (95% confidence interval [CI] −3.52 to −0.32) after TRE, without significant differences in FFM (−1.15 kg; 95% CI −2.46 to 0.16) or aerobic determinants. Neither Tovar et al. [38] nor Correia et al. [40] detected significant between-condition body-mass differences. Richardson et al. [39], reporting the Tovar et al. [38] trial, found no between-diet difference in bone mineral density. Kuikman et al. [33] reported greater body-mass loss with LEA across an altitude camp, without significant fat-mass ($p = 0.053$) or FFM interactions. In the controlled Ramadan comparison, neither body mass nor skinfold-derived body-fat percentage changed significantly in either group, despite the performance decrement during fasting [30]. In Al-Nawaiseh et al. [43], body mass, bioimpedance-derived fat mass and lean mass were likewise unchanged from before Ramadan to its final week (all $p \geq 0.201$), despite significantly reduced energy, carbohydrate and water intakes. Significant FFM reductions occurred in only two of the five bioimpedance-based studies [22,34] and in none of the five longitudinal DXA studies (Table 3).

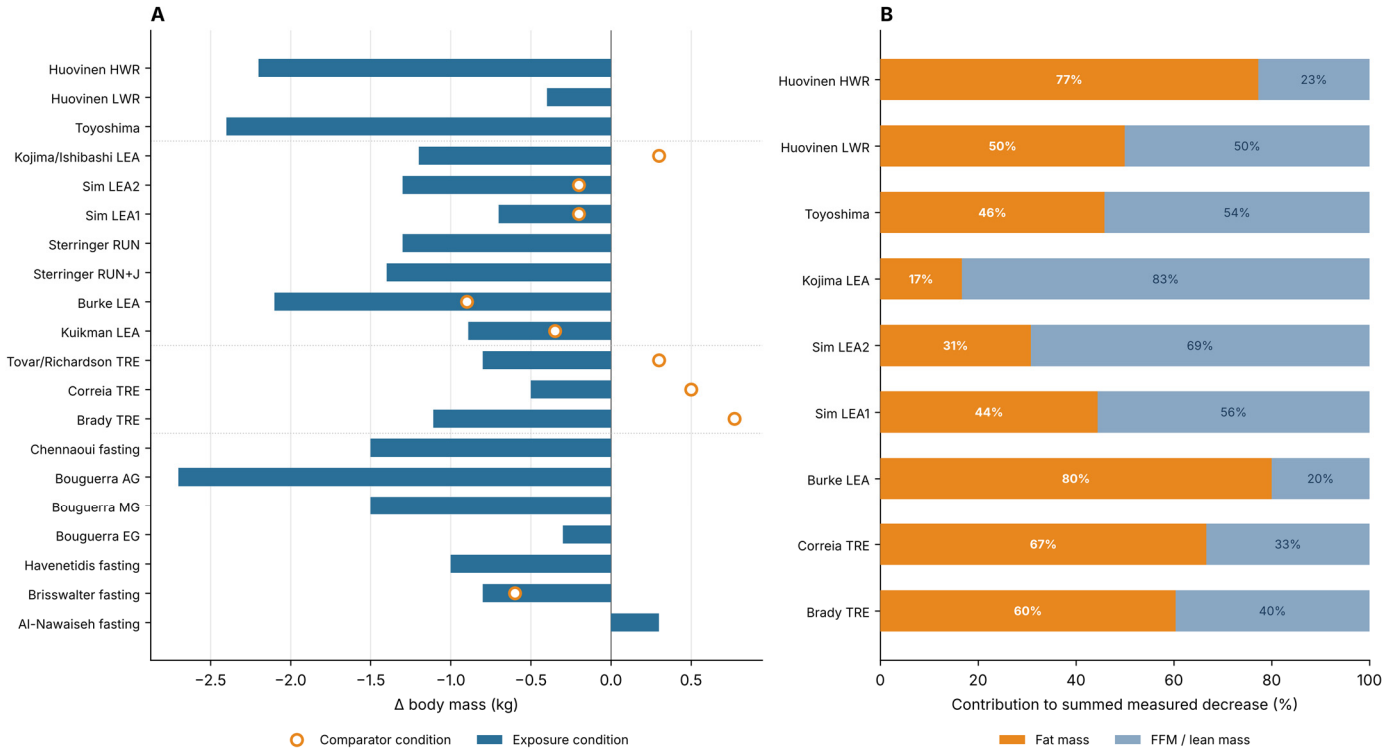

Figure 2. Body-mass change and composition of measured body-mass loss across study conditions. (**A**) Within-condition change in mean body mass from pre- to post-intervention; filled bars denote exposure conditions and open circles the corresponding comparator conditions where longitudinal data were reported. (**B**) Relative contributions of fat mass (FM) and fat-free or lean mass (FFM) to the summed measured decrease, calculated as |component change|/(|ΔFM| + |ΔFFM or lean|) × 100 and shown only for conditions in which both compartments decreased. Condition labels follow the source studies: HWR and LWR, higher and lower weight-reduction groups (Huovinen et al. [32]); LEA, low energy availability; LEA1 and LEA2, the two LEA conditions of the crossover by Sim et al. [36]; RUN and RUN + J, running without and with jumping during LEA (Sterringer et al. [37]); TRE, time-restricted eating; AG, MG and EG, afternoon, morning and evening training groups during Ramadan (Bouguerra et al. [26]); "fasting", the whole-group Ramadan-fasting condition (Chennaoui et al. [42], Havenetidis [31], Brisswalter et al. [30] and Al-Nawaiseh et al. [43]). Values are descriptive within-condition changes, not between-condition effects: bars do not encode statistical significance, measurement intervals differ across studies, multiple conditions from one study are not independent, and fluid and glycogen shifts limit interpretation as tissue partitioning. Exact values and interval qualifications are given in Table 3.

3.6. Tertiary Outcomes and Exploratory Restrictions

P1NP decreased by approximately 15% after three days of restriction [35] and by 14% after six days of LEA [14]; exercise-associated β-CTX area under the curve (AUC) rose 30.9% within LEA [14]. Sterringer et al. [37] reported increased β-CTX across two LEA conditions without P1NP change. Hepcidin AUC over days 1–4 was higher under LEA than normal availability [23], whereas McKay, Peeling et al. [15] detected no LEA–control difference after six days. Severe LEA reduced testosterone [36]. Self-reported recovery was impaired under restriction in Burke et al. [2] and Havenetidis [31]. Kuikman et al. [33] reported no LEA effect on RMR, although the final LEA RMR assessment preceded descent from altitude while triiodothyronine and DXA outcomes were assessed afterwards. In exploratory restrictions (Table 4), excluding the three Low-rated single-group studies removed the strength and maximal-aerobic-velocity decrements and the within-group performance improvements observed in uncontrolled designs; excluding all Low-rated studies additionally removed the findings of Bouguerra et al. [26], but did not remove

the during-fasting 5000 m performance decrement, which persisted in a Moderate-rated controlled study [30]. In a further post hoc restriction to distance-running and race-walking studies (excluding the sprint-and-jump trial of Huovinen et al. [32]), the principal findings were unchanged: no between-condition performance benefit emerged in any category, and the direction of body-mass and fat-mass changes was unaltered. The direction of measured outcomes across studies and outcome domains is summarised in a descriptive direction-of-findings plot (Figure 3).

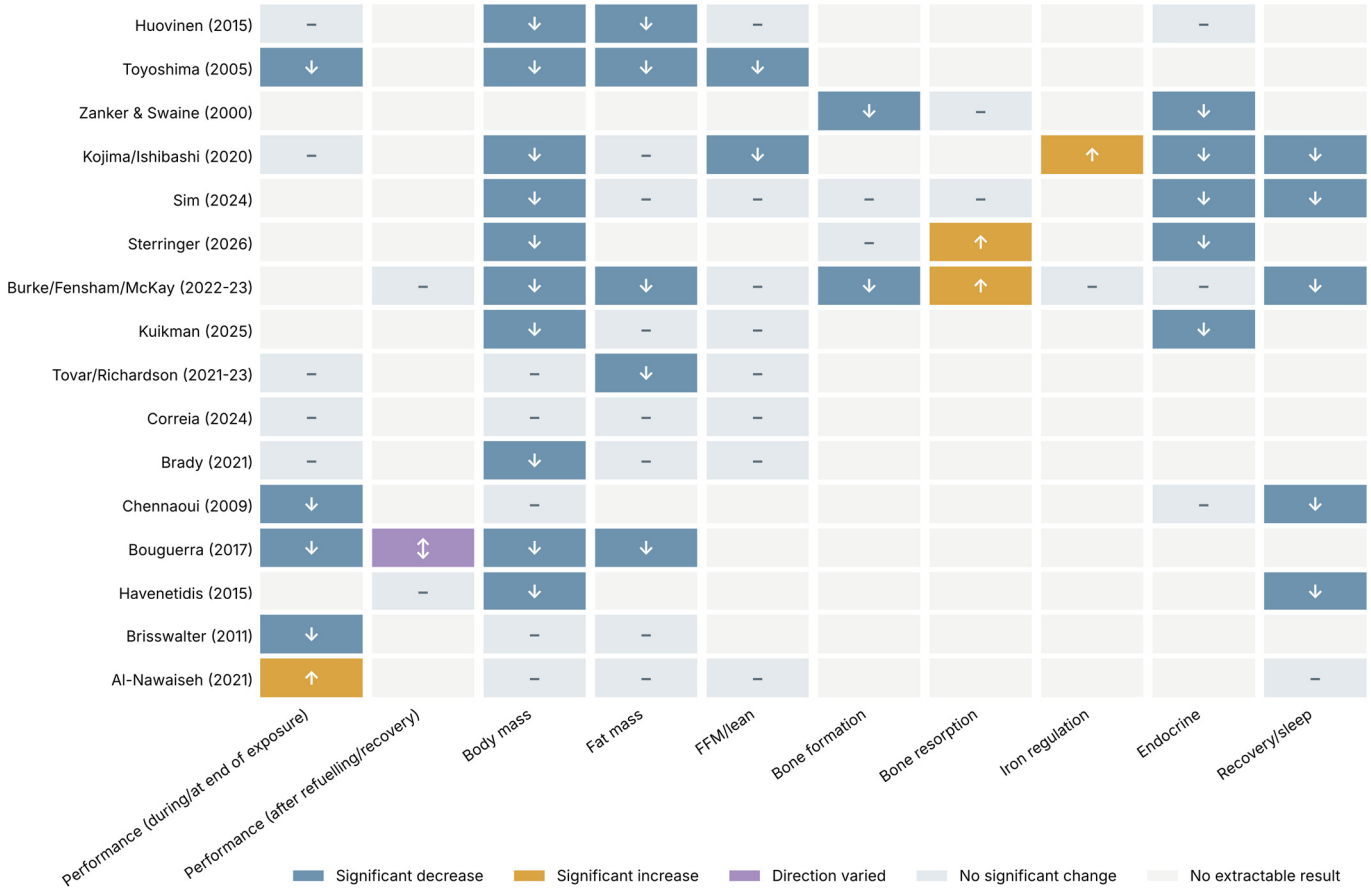

Figure 3. Direction of reported findings across studies and outcome domains. Downward arrows denote lower measured values, except in performance and recovery/sleep columns, where they denote poorer function. Upward arrows denote higher measured values or, in the performance columns, better function. A dash denotes no significant difference; blank cells denote no extractable result, not necessarily an unmeasured outcome. Performance contrasts prioritise between-condition comparisons where available; other domains also include within-condition changes. Performance columns include aerobic determinants; fat-mass cells include body-fat percentages. Endocrine outcomes comprise thyroid, reproductive, adrenal and IGF-axis markers. A double-headed arrow ("Direction varied") denotes differences across outcomes or groups. Colours do not encode clinical benefit, effect size, precision or certainty. The cells are descriptive summaries, not votes for intervention efficacy; comparison-specific details are in Tables 1 and 3 [2,14,15,22,26,30–43].

Table 4. Descriptive comparisons and exploratory restrictions of the evidence.

Comparison	Contributing Evidence	Descriptive Finding and Limitation
Intervention class	16 studies: prescribed deficit/weight-loss programme (3); controlled LEA (5); TRE (3); Ramadan (5).	Controlled LEA lasted 3–9 d, TRE 4–8 weeks and Ramadan approximately one month. Exposure class and duration overlap. Ramadan also changes fluid intake and daily schedules; it is not an equivalent, prescribed energy-deficit dose.
Exposure duration and measurement interval	All 16 studies; measurement intervals are summarised in Table 3.	Short-term mass changes may include glycogen/fluid shifts and should not be interpreted as sustainable weekly tissue-loss rates. DXA in Burke et al. [2] spans baseline plus adaptation, and in Kuikman et al. [33] the whole altitude camp. Exact anthropometric intervals are unspecified in Bouguerra et al. [26] and Havenetidis [31]; Sterringer et al. [37] tabulate screening rather than condition-specific starting BM.
Protein intake (exploratory)	Huovinen et al. [32], Burke et al. [2] and Kuikman et al. [33] at approximately 2.0–2.1 g/kg/d; the remaining reports provide only partial intake or composition data.	These three DXA studies reported fat loss without significant FFM reduction, but none randomised protein independently. The FM interaction in Kuikman et al. [33] was $p = 0.053$. The complete dietary records in Toyoshima et al. [34] covered four runners; Correia et al. [40] did not record intervention intake. Sterringer et al. [37] and Havenetidis [31] do not provide post-intervention FM/FFM. No protective threshold is established.
Sex	12 women in Sterringer et al. [37], 3 in Burke et al. [2] and 19 in Kuikman et al. [33].	Under the linked-report convention, 34/234 participants (14.5%) were reported as women. Kuikman et al. [33] provide a female-only controlled altitude cohort; Sterringer et al. [37] compare jumping during LEA. Differences in intervention, environment, reproductive-status reporting and outcomes preclude a reliable sex-specific effect estimate.
Event and calibre	Distance runners; one sprinter/jumper study; two race-walker studies.	The Kuikman et al. [33] female altitude cohort is distinct from the Burke/Fensham/McKay group [2,14,15]. Havenetidis [31] studied 3 km athletics-team runners, not an undifferentiated military population. Calibre spans recreational to world-class. No eligible report specifically tests hurdles or combined events; throws are outside the review scope.
DXA-only comparison (exploratory)	5 studies: Huovinen et al. [32], Burke et al. [2], Kuikman et al. [33], Tovar et al./Richardson et al. [38,39] and Correia et al. [40].	No significant group-level FFM/lean-mass decrease was established. Significant FFM reductions in the included evidence were reported with bioimpedance. This method-associated pattern neither invalidates those findings nor proves lean-tissue preservation; measurement method, protein, duration and population are confounded.

Table 4. *Cont.*

Comparison	Contributing Evidence	Descriptive Finding and Limitation
Adequate-intake/habitual-diet/non-fasting comparator	10 studies: Zanker & Swaine [35], Kojima et al./Ishibashi et al. [22,23], Sim et al. [36], Burke et al. [2], Kuikman et al. [33], Tovar et al./Richardson et al. [38,39], Correia et al. [40], Brady et al. [41], Havenetidis [31] and Brisswalter et al. [30].	Between-condition effects, rather than the contrast between significant and non-significant within-arm results, are interpretable. Shared time effects do not isolate exposure. Havenetidis [31] and Brisswalter et al. [30] included non-fasting controls; only Brisswalter et al. [30] reported a longitudinal 5000 m test. Sterringer et al. [37], Bouguerra et al. [26] and Al-Nawaiseh et al. [43] lack adequate-EA or non-fasting controls.
Excluding single-group studies (exploratory)	Excluding the single-group studies (Toyoshima et al. [34], Chennaoui et al. [42] and Al-Nawaiseh et al. [43]) leaves 13 studies; excluding all four Low-rated studies also removes Bouguerra et al. [26], leaving 12.	Single-group exclusion removes the strength findings of Toyoshima et al. [34], the MAV findings of Chennaoui et al. [42] and the within-group performance improvements of Al-Nawaiseh et al. [43], but retains the Bouguerra et al. [26] training-time comparison. The Brisswalter et al. [30] performance decrement remains after either exclusion. These restrictions change the exposures represented and do not alter non-randomised allocation; no pooled or subgroup effect was calculated.
Timing of performance and RMR testing	Burke et al. [2], Kuikman et al. [33] and the five Ramadan studies [26,30,31,42,43].	Burke et al. [2] tested racing after refuelling. The Kuikman et al. [33] DXA/blood outcomes extend to 36 h after descent; the last LEA RMR measurement preceded descent. Brisswalter et al. [30] and Al-Nawaiseh et al. [43] retested during the last week of fasting. Bouguerra et al. [26] tested before/during/after Ramadan; Havenetidis [31] reported pre/post predicted VO_2max and weekly recovery. Chennaoui et al. [42] did not retest MAV after Ramadan.

Note. These are structured narrative comparisons, not meta-analyses or formal subgroup tests. Protein, measurement-method, comparator and evidence-exclusion comparisons are exploratory. Companion reports are counted once; study-level sample sizes may differ by outcome. A non-significant difference does not establish equivalence or safety. Abbreviations. BM, body mass; DXA, dual-energy X-ray absorptiometry; EA, energy availability; FFM, fat-free mass; FM, fat mass; HEA/LEA, high/low energy availability; MAV, maximal aerobic velocity; RMR, resting metabolic rate; TRE, time-restricted eating; VO_2max, maximal oxygen uptake.

3.7. Evidence in Female Athletes

Thirty-four of approximately 234 participants (14.5%) were reported as women, concentrated in two female-only studies with three additional women in Burke et al. [2]. Sterringer et al. [37] exposed recreational female runners to five days of LEA and reported increased β-CTX without P1NP change and decreased free triiodothyronine; the design compared added jumping, not energy availability levels. Kuikman et al. [33] provide the only controlled female cohort: greater body-mass loss under LEA at altitude, lower triiodothyronine after descent and no detected RMR suppression. The three women in Burke et al. [2] were not analysed separately, and no TRE or Ramadan study reported female participants. Menstrual status and hormonal-contraceptive use were inconsistently reported, so no sex comparison was possible (Table 4).

3.8. Reporting Bias and Certainty of Evidence

Comparison of study methods and trial records with published results identified outcome-specific gaps (Supplementary File S6). Trial records were identified for five of the 16 studies, three registered prospectively and two retrospectively. Results for registered resting metabolic rate and serial hormone profiles were not located in the included reports [2,14,15]. Bouguerra et al. [26] described the FFM calculation without reporting FFM values. Running economy, a registered secondary endpoint, was not reported in the article of Sterringer et al. [37]. No gap was in itself taken as evidence of selective reporting. Certainty was low for time to exhaustion under controlled LEA and for the TRE 10 km comparison, and very low for sprint and jump performance with prescribed energy restriction, the post-refuelling LEA race comparison and the controlled Ramadan 5000 m comparison. Ratings for body composition and physiological outcomes were low or very low, chiefly because of imprecision and study limitations, and no rating exceeded low (Supplementary File S6).

4. Discussion

The main finding of this systematic review is that reducing body mass during athletics training does not necessarily confer an additional performance benefit and that fasting-based exposures can impair performance during the exposure itself even when body mass does not change. The review also identifies a mismatch between functional and physiological outcomes: the absence of detected performance differences can coexist with changes in bone turnover, endocrine function, iron regulation or perceived recovery, and conversely, a performance decrement can occur without measurable changes in body composition. Interpretation, therefore, depends on the dietary exposure, comparator, measurement method and timing, rather than weight-loss magnitude alone.

4.1. Performance: What the Strongest Evidence Does and Does Not Show

Burke et al. [2] provide the most direct competition-based evidence addressing this research question. High-calibre race walkers lost more body mass during LEA, but this did not translate into a greater improvement in 10,000 m performance than under higher energy availability, and pre-race changes in body mass and performance were not significantly associated. The design could not disentangle the effects of lower body mass from fuel availability, training quality or psychological readiness. Dietary provision was adjusted to actual training loads, strengthening ecological validity, although preference-based allocation increased the risk of selection bias. Importantly, both groups were refuelled for approximately 24 h before racing, so performance was assessed after restriction and refuelling rather than during LEA itself. Despite greater fatigue, stress and poorer perceived recovery during LEA, race performance after refuelling was preserved. The authors therefore suggested that,

when body-composition change is judged necessary, a brief, strategically placed period of LEA could be integrated within the annual training plan to allow recovery and refuelling before competition. These findings are consistent with preserved race performance after brief LEA followed by refuelling, without demonstrating an additional performance benefit from greater body mass loss. They do not address prolonged or repeated restriction across a season. The parallel improvements do not demonstrate equivalence between conditions. Practical relevance depends on effect magnitude and its uncertainty rather than on statistical significance alone [44], so a practically important between-group difference cannot be excluded.

The same caution applies in reverse to Huovinen et al. [32]: within-group improvements in jumping and sprinting under the larger deficit are consistent with concurrent training adaptation. Without between-group superiority, such improvements are not evidence that restriction itself enhanced performance.

4.2. Timing of Assessment and the Ramadan Evidence

The interpretation of findings depended on the timing of outcome assessment. Bouguerra et al. [26] reported slower 3000 m performance midway through Ramadan despite no significant pre–post difference, showing how pre–post comparisons may miss changes occurring during an intervention, and Havenetidis [31] reported poorer recovery despite predicted aerobic capacity improving in both groups. Al-Nawaiseh et al. [43] likewise tested in the final week of fasting and reported improved time to exhaustion and maximal running speed, but without a non-fasting comparator and with concurrently reduced training load, the improvement cannot be attributed to fasting itself. Post-intervention measurements therefore cannot establish whether performance and recovery were maintained throughout restriction. The nature of the exposure poses a further interpretive problem. Ramadan fasting typically alters fluid intake, meal timing and sleep, making its effects difficult to isolate without controls [42]. Because observance is determined by religious practice rather than allocation, fasting and non-fasting athletes may also differ in unmeasured ways [30]. Brisswalter et al. [30] provide a particularly informative controlled comparison: in the only Ramadan study with repeated race-distance testing against a non-fasting control, 5000 m time deteriorated in the fasting group while the control group was stable, despite no statistically significant changes in body mass, body-fat percentage or reported energy intake. Chennaoui et al. [42] and Bouguerra et al. [26], lacking non-fasting comparators, could not provide this information. Excluding the Low-rated studies removed the functional decrements reported by Chennaoui et al. [42] and Bouguerra et al. [26], whereas the Brisswalter et al. [30] decrement and the physiological changes in better-controlled studies persisted. Null performance findings also warrant caution: Brady et al. [41] retained fewer participants than required by their power calculation, and Kojima et al. [22] powered their study for glycogen rather than performance.

4.3. Body Composition: Compartments and Methods

Body-composition outcomes must be read against the measured compartment and the method used; this evidence base spans three methodologies with different vulnerabilities: bioimpedance, DXA and anthropometry. Bioimpedance estimates FFM from total-body water and is therefore sensitive to exactly the constituents that short-term restriction perturbs [45]. Kojima et al. [22] reported approximately 1 kg lower bioimpedance-derived FFM alongside approximately 30% lower muscle glycogen; a glycogen-and-fluid shift is therefore a plausible contributor to the measured FFM difference [45], but these data cannot establish whether contractile tissue was lost. Toyoshima et al. [34] similarly reported parallel losses of FFM and body water. Conversely, the absence of significant DXA-derived

FFM loss does not establish preservation: DXA estimates of lean tissue mass are influenced by hydration and glycogen status, and scan timing relative to training and feeding was not uniformly standardised [46]. Statistical non-significance is equally not preservation: in Brady et al. [41], the adjusted between-group FFM difference by bioimpedance was −1.15 kg (95% CI −2.46 to 0.16), so a potentially relevant loss could not be excluded. Measurement method, diet, population and duration also varied together across the DXA studies, so the method contrast cannot be isolated from these differences. In terms of measurement hierarchy, multi-compartment models are least assumption-dependent, DXA is intermediate, and bioimpedance and anthropometry are most vulnerable to acute shifts in hydration and glycogen [45,46]; no included study used a multi-compartment model. Taken together, significant FFM losses were confined to bioimpedance-based studies [22,34], one of which measured a 1.3 L decrease in total body water [34], whereas the controlled DXA comparisons detected no significant FFM loss [2,32,33,38–40]. This pattern is consistent with fluid and glycogen shifts and does not establish loss of contractile tissue, although it equally cannot exclude it. The controlled Ramadan study illustrates the converse dissociation: function declined while skinfold-derived body composition did not [30], and skinfolds cannot directly quantify glycogen, body water or contractile tissue [45]. Finally, extrapolating brief interventions to weekly rates can magnify apparent rates of change, and the DXA intervals in Burke et al. [2] and Kuikman et al. [33] extended beyond the restriction period itself, in the latter case across an altitude descent, so measured intervals and exposure windows should be read together (Figure 2; Table 3).

4.4. Protein Intake

No included study isolated the effect of protein intake, so the evidence permits only an observation of co-occurrence: Huovinen et al. [32], Burke et al. [2] and Kuikman et al. [33] provided approximately 2.0–2.1 g/kg/day and reported fat loss without significant DXA-derived FFM loss. The mechanistic rationale for elevated protein during restriction is well described: it supports muscle protein synthesis when energy deficit suppresses it, increases satiety and carries a modestly higher thermic effect [47,48]. Syntheses in resistance-trained populations associate higher intakes with better lean-mass retention during energy deficit [49,50]. In elite athletes with supplementary resistance training, slower weight loss produced better lean-mass and strength outcomes, although rate was confounded with intervention duration [51]. The present pattern is consistent with those recommendations but establishes neither a protective effect nor a threshold in athletics: no study compared protein doses under matched deficits, endurance training loads differ fundamentally from the resistance-training contexts in which the evidence was generated, and adherence outside provided-food designs was self-reported. Tissue-specific protection should also not be assumed: outside the included evidence, increasing protein from 0.8 to 1.7 g/kg/day did not attenuate LEA-associated bone-turnover changes in a small cycling study [52], cautioning against extending a lean-mass argument to skeletal outcomes. To our knowledge, a protein dose comparison under a controlled energy deficit in trained runners has not yet been conducted.

4.5. Carbohydrate Availability and Marker Kinetics

Carbohydrate availability further complicates causal attribution. In companion reports on race walkers, an energy-adequate ketogenic diet altered selected bone turnover, hepcidin and immune responses despite adequate energy provision [14,15]. In Fensham et al. [14], exercise-associated P1NP declined only in the ketogenic arm, whereas CTX area under the curve increased in both ketogenic and LEA arms. Between-arm differences were not established for every marker or assessment period. Although ineligible as a restriction

intervention for this study, the ketogenic arm illustrates why energy and carbohydrate effects should not be conflated, a conclusion that converges with the biochemical synthesis of Guisado-Cuadrado et al. [18]. In the trial of Burke et al. [53], adaptation to a ketogenic low-carbohydrate, high-fat diet impaired exercise economy and negated the performance benefit of intensified training in elite race walkers, with energy intake matched across diets. Metabolomic evidence in elite endurance athletes similarly showed that a low-carbohydrate, high-fat diet induced greater changes in selected circulating lipid-related metabolites than short-term LEA [54]. Whether carbohydrate timing prevents adverse responses during energy restriction remains untested.

The iron findings are only partially consistent with this interpretation: hepcidin is stimulated by inflammatory signalling and suppressed by erythropoietic drive, and the discordant results after three days (higher hepcidin AUC under LEA [23]) and six days (no LEA–control difference [15]) in separate cohorts cannot define response kinetics, because assay timing relative to exercise, carbohydrate provision during testing and baseline iron status all differed. An energy-deficit-driven hepcidin increase has likewise been reported outside athletics, during simulated military operations [55], supporting biological plausibility without resolving the time course.

Endocrine responses showed a similar exposure dependence: severe LEA reduced testosterone within days [36], while bone-formation suppression appeared at 15 kcal/kg FFM/day within roughly the same window [14], suggesting that formation markers and hypothalamic–gonadal readouts respond on comparable, short timescales. By contrast, the included designs did not detect significant LEA effects on resting metabolic rate, although in Kuikman et al. [33], final LEA RMR testing preceded descent from altitude, whereas triiodothyronine and DXA were assessed afterwards, precluding interpretation as concurrent responses; these null findings do not establish the absence of metabolic adaptation.

4.6. Positioning Within the Wider Evidence

This review complements and extends Guisado-Cuadrado et al. [18] in population, exposure and outcomes: it is specific to athletics rather than mixed sports; it covers prescribed restriction, TRE and Ramadan fasting, including two non-fasting comparisons, rather than induced LEA alone; and it integrates competitive performance and body composition with biomarkers rather than synthesising biomarkers alone. Dose–response work in non-athlete populations [12,56,57] characterises physiological responses to reductions in energy availability under laboratory conditions; translating these findings to athletics requires competition-relevant testing during restriction and fasting and after refuelling. Evidence from rapid weight-making in weight-category sports [13] addresses a different exposure and recovery context and should not be generalised to athletics. Three gaps limit interpretation. First, the central unresolved question is whether short-term functional and physiological changes predict competition performance, recovery or cumulative risk across repeated cycles. Bone-turnover markers are not surrogates for skeletal strength or injury risk, and no included study examined repeated restriction across a season. Injury outcomes were absent altogether, despite the injury burden that motivates concern about LEA in athletics [16,17]. Second, women constituted 14.5% of participants, were absent from the Ramadan evidence entirely, and menstrual status or hormonal contraceptive use was inconsistently reported where women were included, so sex-specific inference remains limited [8]. Third, the evidence base is too small to stratify by event or athletic calibre, despite participants spanning Tiers 1–5 of the classification framework [20]. The rationale for body-mass reduction differs between sprinters, jumpers and marathon runners, yet findings from well-trained middle- and long-distance runners dominate the synthesis.

4.7. Limitations

This review has limitations at two levels. The evidence base constrains inference: samples were small, randomisation was often absent or partial, exposures were brief, and comparators and assessment schedules were heterogeneous (Table 1). At the review level, screening and data extraction were performed by L.B. and verified by S.I., rather than conducted independently in parallel; quality appraisal was completed by both authors. Single-reviewer abstract screening can miss approximately 13% of relevant studies [58]. As safeguards, the second reviewer verified all screening decisions and extracted data, and a pre-submission verification of the screening record against the database harvests identified one additional eligible report, which was taken to full text and included (Supplementary File S2). The review was not registered. However, the eligibility criteria, search strategy, study selection procedures, and synthesis methods are transparently documented, and the complete search, screening, and critical appraisal materials are publicly available through the Open Science Framework (Supplementary Materials). We acknowledge that the absence of registration limits independent verification of prespecified methodological decisions and represents a limitation of the review. The search was limited to four databases and English-language, peer-reviewed full reports, so studies indexed elsewhere, published in other languages or available only as grey literature may have been missed. CASP checklists are structured appraisal instruments rather than domain-based risk-of-bias tools, although the randomised studies were additionally assessed with the Cochrane RoB 2 tool (Supplementary File S5), and the same heterogeneity precluded meta-analysis. Exploratory restrictions are descriptive only. Women constituted 14.5% of participants and were entirely absent from the Ramadan evidence, and well-trained middle- and long-distance runners dominate the included samples, so the findings cannot be assumed to generalise to sprint, jump and combined events, to women observing Ramadan, or to other weight-sensitive sports. Overall, no body of evidence reached moderate certainty in the GRADE assessment (Section 3.8).

4.8. Practical Implications and Future Research

Body-mass loss alone should not be interpreted as evidence of performance benefit, and stable body mass should not be interpreted as evidence that a fasting exposure is functionally neutral. Where physique change is judged necessary, the included evidence and current consensus support embedding it in planning rather than improvising it: scheduling physique-focused phases away from key competitions, as documented in elite practice [7]; maintaining adequate protein intake during restriction; preserving carbohydrate availability around demanding sessions; and refuelling before competition. Monitoring should extend beyond the scale to training quality, perceived recovery and, where feasible, relevant biomarkers, because physiological and recovery responses did not consistently track performance. Practitioner awareness cannot be assumed: a survey of athletics and short-track coaches found limited awareness of REDs [59], so implementation includes educating coaching staff, not only athletes. These are prudent considerations, not prescriptions validated by this review. For athletes observing Ramadan, the controlled comparison suggests that performance decrements in the final week of fasting are possible even without statistically significant weight loss; competition scheduling warrants individual planning, and the training-time interactions reported by Bouguerra et al. [26] do not identify a universally preferable schedule.

Future studies should independently manipulate deficit magnitude or protein intake under matched conditions, standardise body-composition assessment and scan timing, and include competition-relevant testing during restriction and after refuelling, with sample sizes calculated to detect a prespecified smallest worthwhile difference in the primary perfor-

mance outcome. Experimental models that manipulate body mass directly (external loading and hypohydration) provide complementary causal evidence on the mass–performance relationship [60,61] and could be integrated with dietary designs. Repeated restriction cycles with injury and illness surveillance, and greater female representation with menstrual status documented, including in Ramadan research, are priorities. Multicentre studies embedded within existing training camps may facilitate recruitment without disrupting competitive schedules [2,14].

5. Conclusions

Dietary restriction and fasting can reduce body mass or fat mass during continued athletics training, but the available evidence does not demonstrate a consistent performance advantage, and a controlled comparison showed that performance can decline during Ramadan fasting without statistically significant changes in body mass or reported energy intake. The certainty of this evidence is low to very low, reflecting small samples, brief exposures and heterogeneous designs. Interpretation must distinguish outcomes measured during restriction or fasting from those assessed after refuelling, and changes in body composition from changes in physiological function. Short-term changes in biomarkers or recovery may occur without a statistically significant performance decrement. Conversely, neither non-significant performance differences nor the absence of significant FFM loss establishes physiological tolerance. Protein intake and carbohydrate availability warrant consideration, but neither a protective protein threshold nor an optimal restriction schedule can be identified from the current evidence. Practical decisions should be individualised to the athlete, event and training phase rather than guided by weight loss alone. Future studies should distinguish energy-restriction effects from those of low carbohydrate availability and recruit sufficient numbers of women and men to assess outcomes through restriction, fasting, recovery and repeated cycles. The available evidence does not establish that reducing body mass improves performance in athletics. Body mass can fall without performance improving, and performance can fall without a statistically significant change in body mass.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/nu18233904/s1, S1: full search strategies; S2: screening and updated-search record; S3: PRISMA 2020 checklist; S4: per-item CASP justifications; S5: Cochrane RoB 2 assessments of the randomised studies; S6: reporting-bias assessments and GRADE certainty-of-evidence ratings.

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