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Continuous Glucose Monitoring in the Assessment of Low Energy Availability and Relative Energy Deficiency in Sport (REDs) in Athletes Without Diabetes: A Critical Narrative Review

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Abstract

Background: Continuous glucose monitoring (CGM) has been proposed to support assessment of low energy availability (LEA) and Relative Energy Deficiency in Sport (REDs), but its diagnostic value is uncertain.

Aim: To critically evaluate CGM evidence relevant to LEA and REDs in athletes without diabetes.

Material and methods: Two PubMed searches on 1 October 2026 yielded 145 unique records. AI-assisted assessment informed a purposive narrative synthesis of experimental, clinical, observational and measurement-agreement studies, supplemented by previously identified sources.

Results: Severe short-term energy restriction reduced sensor glucose in one experiment, whereas studies of controlled LEA, nutritional intervention and clinically diagnosed REDs did not consistently identify differences in aggregate glucose metrics. Dietary composition, training, recovery and measurement conditions complicated interpretation. No reviewed study established a validated CGM threshold for identifying or excluding REDs.

Conclusions: CGM remains an investigational adjunct. Low readings do not confirm LEA or REDs, and an unremarkable profile does not exclude them. Prospective validation must establish whether CGM adds information beyond nutritional and clinical assessment.

Keywords: continuous glucose monitoring; low energy availability; relative energy deficiency in sport; athletes; interstitial glucose

1 Introduction

Low energy availability (LEA) arises when dietary energy remaining after exercise expenditure is insufficient to support physiological functions. Its clinical consequences depend on the magnitude, duration and context of exposure. The 2023 International Olympic Committee consensus distinguishes transient, adaptable responses from problematic LEA associated with Relative Energy Deficiency in Sport (REDs). This distinction matters because a short period of under-fuelling and an established clinical syndrome are not interchangeable research outcomes [1].

Assessment is challenging: estimated energy availability depends on several measurements, while REDs requires a clinical evaluation that considers symptoms, signs and alternative explanations. A measure that responds to changes in food intake may therefore be informative without being sufficiently specific for diagnosis. The IOC REDs Clinical Assessment Tool, version 2, provides a multidimensional framework rather than a single-biomarker definition of the syndrome [2].

Continuous glucose monitoring (CGM) offers repeated measurements of interstitial glucose during daily life, exercise and sleep. These observations have prompted interest in identifying glucose patterns associated with inadequate energy provision. However, glucose concentration is not a direct measurement of energy availability. Broader reviews of CGM in endurance sport have already highlighted uncertainties concerning nutritional interpretation, training monitoring and measurement validity [3]. A focused appraisal must therefore separate biological responsiveness from evidence of diagnostic usefulness.

This critical narrative review examines whether CGM can contribute to assessment of LEA and REDs in athletes without diabetes. It distinguishes experimental energy restriction from clinically diagnosed REDs, evaluates daily

and nocturnal outcomes, and considers dietary, training-related and measurement influences. The central question is whether the available findings justify identifying or excluding LEA or REDs from a glucose profile, or support only a more limited role in research and contextual monitoring.

2 Materials and methods

This work is a critical narrative review. Two PubMed searches were performed on 1 October 2026 without filters, using terms in the title and abstract fields. The first combined CGM or interstitial glucose terms with athlete, cyclist or endurance terms and terms for energy availability or REDs. It returned four records. The second broadened the sports terms and omitted the energy-availability block, returning 145 records. All four records from the first search were present in the second set; the combined searches therefore contained 145 distinct PubMed identifiers.

The original export contained 145 unique PubMed identifiers. Title and metadata assessment set aside 75 records, chiefly because they concerned diabetes management, non-human populations or sweat-based sensing. For the remaining 70 records, 69 available PubMed abstracts were assessed; one report without an abstract was assessed as a full-text research letter. Independent duplicate screening was not performed.

Primary evidence was prioritised when it quantified EA, imposed an energy deficit or investigated clinically diagnosed REDs in people without diabetes. Selected studies of daily or nocturnal glucose, carbohydrate intake, training load and measurement agreement supplied interpretive context. Military experiments were treated as indirect evidence. Studies confined to diabetes management, animal research or sweat sensing were outside the main scope; pre-exercise reactive hypoglycaemia and performance-oriented feeding interventions were not the focus. Relevant contextual studies were selected purposively rather than exhaustively.

Thirteen reports from the original export were retained in the current narrative synthesis. Caldwell et al. [4], previously supplied with supplementary tables, provided an additional primary report outside that export. The evidence table therefore contains 14 publication-level entries, not 14 independent cohorts. Consensus documents and reviews supplied background. Retrieval on 7 October 2026 clarified already-identified sources, including Bowler, Coates and Ranahan, without adding these again to the search count. A record-level selection log and the exact search strings are retained in the review records.

The synthesis distinguishes experimental energy restriction, clinical REDs comparisons, contextual glucose profiles and measurement agreement. Study interpretation considered population, dietary exposure, device, monitoring window, comparator, missing data and access to the final publication. Findings were compared narratively; no meta-analysis or formal risk-of-bias score was performed. Companion reports and alternative versions were identified to avoid treating them as independent replication. Abstract-only findings and methods assessed from a thesis or preprint are identified explicitly.

3 Results of the literature review

3.1 Definitions and measurement of energy availability

Energy availability (EA) describes the dietary energy remaining for physiological functions after exercise-related energy expenditure has been deducted. It is calculated as dietary energy intake minus exercise energy expenditure, divided by fat-free mass, and expressed in kcal/kg fat-free mass/day. EA differs from energy balance, which considers total energy expenditure. Consequently, studies reporting an energy deficit cannot automatically be interpreted as having quantified EA using this definition [1].

The 2023 International Olympic Committee (IOC) consensus distinguishes adaptable from problematic low energy availability (LEA). Adaptable LEA involves changes that are generally mild and rapidly reversible, whereas problematic LEA is associated with more substantial and potentially persistent physiological disturbances. Relative Energy Deficiency in Sport (REDs) describes impaired physiological and/or psychological functioning resulting from exposure to problematic LEA. A short experimental reduction in energy intake therefore does not, by itself, establish a diagnosis of REDs [1].

Although an EA threshold of approximately 30 kcal/kg fat-free mass/day has frequently been used in research, it should not be treated as a universal diagnostic criterion. Individual responses vary, and the measurement of dietary intake, exercise expenditure and fat-free mass introduces uncertainty, particularly under free-living conditions. These limitations are relevant when interpreting associations between estimated EA and continuous glucose monitoring (CGM) outcomes [1].

Clinical assessment requires a broader approach. The IOC REDs Clinical Assessment Tool, version 2 (CAT2), incorporates screening, severity/risk assessment and a physician-led diagnosis informed by clinical findings and

differential diagnosis. Clinically low blood glucose is listed as a potential, non-scored indicator rather than a primary or secondary indicator. Although the IOC consensus includes CGM among methods used and recommended for investigating glucose metabolism, this does not establish a validated CGM threshold for diagnosing REDs [1, 2].

3.2 Experimental and field evidence on low energy availability

Smith et al. [5] investigated severe short-term energy deprivation combined with sustained exercise in a randomised, double-blind crossover trial. Twenty-three young military volunteers (17 men and six women) completed both conditions. During the approximately two-day intervention, reported energy intake was about 270 kcal/day in the deficit condition and 3940 kcal/day in the fed condition, with approximately three hours of exercise per day. This protocol examined an extreme energy deficit rather than calculated EA or clinically diagnosed REDs.

Mean interstitial glucose was lower during the deficit condition than during the fed condition (4.1 ± 0.8 versus 5.0 ± 1.2 mmol/L; $p < 0.0001$). The proportion of recorded time below 3.9 mmol/L was also greater (38.7 ± 25.0% versus 13.6 ± 13.7%; $p = 0.04$). However, the difference in time below 3.1 mmol/L was not statistically significant ($p = 0.10$). These observations demonstrate a glucose response to the experimental intervention, but they do not establish diagnostic performance for LEA or REDs. Energy and carbohydrate intake changed simultaneously, approximately 24% of CGM data were missing, and the extreme, short-term intervention differs from the conditions typically investigated in athletes with chronic under-fuelling [5].

Caldwell et al. [4] examined a more prolonged, controlled reduction in energy availability in endurance-trained women. In a randomised crossover study, 12 women contributed data from 14-day periods of low and optimal energy availability, averaging 22.3 ± 1.5 and 51.9 ± 2.0 kcal/kg fat-free mass/day, respectively. FreeStyle Libre 2 monitoring covered days 7–14 of each intervention. The authors reported no effect of diet on overall daily CGM-derived glucose regulation, despite impaired cycling time-trial performance following LEA. However, post-breakfast incremental glucose area under the curve was approximately 19.7% higher during LEA ($p = 0.039$), whereas total area under the curve and peak glucose did not differ significantly. These findings caution against assuming that reduced energy availability necessarily produces lower overall sensor glucose, while also showing that unchanged aggregate metrics do not imply an absence of changes in postprandial responses. CGM analyses included 10 participants during OEA and 11 during LEA (Supplementary Table S3). Interpretation is limited by the small sample and the simultaneous reduction in carbohydrate intake during LEA. The study did not evaluate diagnostic thresholds for LEA or clinically diagnosed REDs.

Field evidence provides a complementary perspective. Tarocchi et al. [6] investigated adolescent male cyclists across three periods of a competitive season. EA was estimated from dietary intake, exercise expenditure and fat-free mass, with LEA operationally defined as EA below 30 kcal/kg fat-free mass/day. During the final phase, completed by seven cyclists, an individualized nutritional intervention increased estimated EA from 29 ± 9 to 38 ± 6 kcal/kg fat-free mass/day ($p = 0.040$). Neither mean daily nor mean nocturnal glucose changed significantly.

Across these studies, glucose responses varied with the exposure and setting. Smith et al. [5] observed lower interstitial glucose during severe energy deprivation, whereas Caldwell et al. [4] did not detect changes in aggregate CGM metrics during a longer controlled LEA intervention, despite a change in post-breakfast incremental glucose AUC. Tarocchi et al. [6] found that improved estimated EA did not produce a significant change in mean daily or nocturnal glucose. Population, dietary composition, intervention magnitude and measurement differences limit direct comparison. None of these studies established a diagnostic CGM threshold for REDs.

3.3 Evidence in clinically diagnosed REDs

Matkin-Hussey et al. [7] conducted a cross-sectional pilot study comparing nine female athletes with clinically diagnosed REDs and nine matched controls under free-living conditions. Diagnosis was established by a sport and exercise physician and/or reproductive endocrinologist using clinical assessment informed by the original REDs Clinical Assessment Tool and guidelines for functional hypothalamic amenorrhoea. Participants wore an Abbott FreeStyle Libre 2 sensor for eight days. The methods specify analysis from Day 1 at 05:00 to Day 7 at 22:00, with nocturnal periods defined as 22:00–05:00. However, this stated interval is not directly consistent with the reported mean of 165 ± 5 hours of available data per participant, leaving some uncertainty about the exact analytical window.

No statistically significant between-group differences were detected in mean glucose or mean amplitude of glycaemic excursions (MAGE) across the overall, daytime or nocturnal periods. Nocturnal mean glucose was 5.38 ± 0.58 mmol/L in the REDs group and 5.07 ± 0.35 mmol/L in controls ($p = 0.21$); corresponding MAGE values were 1.36 ± 0.25 and 1.34 ± 0.44 mmol/L ($p = 0.92$). These findings do not demonstrate equivalence between groups or establish the diagnostic sensitivity of CGM.

Interpretation also requires consideration of participants' recovery status. Although personalised energy-intake plans were withheld until study completion, prior clinical and dietetic contact may already have influenced dietary behaviour. Three REDs participants had an estimated positive energy balance, which the authors interpreted as potentially reflecting early recovery efforts. A further four reported changing pre-exercise fuelling after observing CGM readings. Nevertheless, excluding REDs participants with positive energy balance and controls with negative energy balance did not reveal differences in glucose metrics.

Energy availability was not calculated because fat-free mass measurements were unavailable; estimated energy balance cannot substitute for this assessment. Together with the small sample and possible behavioural changes during monitoring, these limitations constrain generalisation to untreated REDs. The study therefore demonstrates a lack of detected group differences in this particular clinical sample, rather than validating CGM as a method for identifying or excluding REDs.

3.4 Overnight and daily glucose metrics

Overnight glucose has been investigated as a potential marker of inadequate energy provision. However, mean nocturnal glucose, minimum overnight glucose and time below a specified threshold represent different aspects of the glucose profile and should not be treated as interchangeable outcomes. Comparability is further limited by differences in observation windows. Matkin-Hussey et al. [7] defined night as 22:00–05:00, Tarocchi et al. [6] used 23:00–07:00, and Amatori et al. [8] used 00:00–06:00. Fixed clock-time windows may not correspond to individual sleep periods.

Controlled experimental evidence also indicates that nocturnal CGM metrics may not show a consistent response to reduced EA. In Caldwell et al. [4] (Supplementary Table S3), CGM data were available for 10 participants during OEA and 11 during LEA. Estimated nocturnal mean glucose was 5.0 mmol/L (95% CI, 4.7–5.3) during OEA and 4.8 mmol/L (95% CI, 4.5–5.1) during LEA. The estimated difference was −0.2 mmol/L (95% CI, −0.6 to 0.3; $p = 0.440$). No significant difference was detected in nocturnal minimum glucose ($p = 0.544$). These are estimated marginal means and confidence intervals, not means with standard deviations. Neither the main CGM methods nor Table S3 specifies the clock-time boundaries used to define night, limiting comparison with other studies.

The available findings do not establish a consistent relationship between nocturnal glucose and energy deficiency across settings. Smith et al. [5] observed lower glucose during sleep under severe experimental energy restriction. In contrast, Tarocchi et al. [6] found no significant change in mean nocturnal glucose following an improvement in estimated energy availability, while Matkin-Hussey et al. [7] detected no significant difference in nocturnal mean glucose between athletes with clinically diagnosed REDs and controls. These findings concern different exposures and populations and should not be interpreted as directly contradictory.

Additional evidence comes from professional cyclists monitored during two consecutive annual training camps. According to the available abstract, Zignoli et al. [9] found no statistically significant association between minimum overnight interstitial glucose and either daily exercise energy expenditure or carbohydrate intake. This evidence remains provisional within the present review because the full text was unavailable for methodological assessment. Moreover, exercise energy expenditure alone does not quantify EA, and a non-significant association does not establish the absence of a relationship.

Bowler et al. [10] studied 12 racewalkers during two four-day periods with standardised diet and exercise. Mean glucose was 5.7 ± 0.3 mmol/L over 24 hours and 5.1 ± 0.3 mmol/L overnight (22:00–06:00); mean daily difference was 0.7 ± 0.1 mmol/L. These descriptive values are not diagnostic thresholds. EA was not quantified, and between-day variation incorporates biological and sensor-related influences.

Hamilton et al. [11] monitored nine professional female cyclists during a nine-day camp. Mean nocturnal glucose (00:00–05:59) was lower than daytime glucose. Dietary intake and EA were not quantified, and low sensor readings were not confirmed by blood sampling. The authors considered compression during sleep as a possible

explanation for some events. These observations provide descriptive context, not evidence that nocturnal low readings identify LEA.

Overall, overnight monitoring may provide information about glucose responses under specific nutritional and training conditions. Its interpretation as an indicator of LEA or REDs requires further validation, including standardized outcome definitions and consideration of the timing of sleep, dietary intake and preceding exercise.

3.5 Carbohydrate intake and training as influences on CGM

Dietary carbohydrate intake can influence CGM outcomes without a demonstrated change in total energy availability. Amatori et al. [8] investigated 15 trained male cyclists in a randomized crossover trial comparing two seven-day diets with similar reported energy intake but different carbohydrate content. Actual carbohydrate intake averaged 5.15 ± 1.22 g/kg/day during the higher-carbohydrate condition and 2.71 ± 0.90 g/kg/day during the lower-carbohydrate condition.

The lower-carbohydrate condition was associated with lower mean glucose (model coefficient $\beta = -3.77$ mg/dL, SE = 1.47; $p = 0.011$) and a lower coefficient of variation ($\beta = -1.31$ percentage points; $p = 0.028$). However, the overall diet effect was not significant for MAGE ($p = 0.323$) or time below 70 mg/dL ($p = 0.530$). Thus, differences in carbohydrate intake did not affect all CGM outcomes uniformly [8].

Training-related associations were also observed. Across 144 exercise sessions, higher exercise intensity was associated with higher mean sensor glucose ($p < 0.001$). Across 180 nights, exercise on the preceding day was associated with lower nocturnal mean glucose after adjustment for carbohydrate intake ($\beta = -1.83$ mg/dL, SE = 0.92; $p = 0.047$). These associations were observed within the dietary trial and do not establish a causal effect of training independent of all other influences [8].

The diets were designed to be isocaloric, but EA was not directly assessed. Similar energy intake therefore does not establish equivalent or adequate EA. Nevertheless, the study demonstrates that carbohydrate intake and exercise are relevant alternative explanations for differences in CGM outcomes. This is particularly important when interpreting severe energy-restriction experiments, such as Smith et al. [5], in which carbohydrate intake was also substantially reduced.

Coates et al. [12] reported lower CGM glucose during heart-rate-matched submaximal cycling after overload training in 11 athletes ($p = 0.047$), but not during a 5-km time trial ($p = 0.07$). Energy intake was unmeasured; stable indirect nutritional markers cannot exclude LEA. Protocol details were assessed in the preprint [13], with principal findings checked against the published abstract.

In a companion analysis, Ranahan et al. [14] included 10 athletes, with CGM data available for eight. Daily and nocturnal mean glucose did not differ across training phases, whereas daily MAGE increased during recovery (2.6 ± 0.2 versus 2.3 ± 0.3 mmol/L at baseline). Dietary intake was not monitored. These linked reports must not be treated as independent replication.

3.6 Measurement agreement and methodological limitations

The interpretation of CGM as a potential marker of low energy availability requires consideration of its agreement with blood glucose measurements. CGM measures glucose in interstitial fluid, and its readings should not be assumed to be interchangeable with contemporaneous blood glucose values. Moreover, measurement agreement and diagnostic validity represent distinct requirements: agreement with blood glucose alone would not establish the ability of CGM to identify LEA or REDs.

Bauhaus et al. [15] compared a sport-oriented CGM device with laboratory analysis of capillary blood samples in ten athletes under resting and exercise conditions. Mean absolute relative deviation was lowest during fasted rest (8 ± 6%), compared with post-glucose rest (17 ± 12%) and protocols involving moderate- and high-intensity running (22 ± 24% and 18 ± 17%, respectively). The exercise protocols included glucose ingestion and a pre-exercise observation period; these estimates should therefore not be interpreted as reflecting exercise alone. Bland–Altman analyses showed wider limits of agreement under exercise conditions. These findings illustrate why correlation between measurement methods, or a small average difference, does not establish sufficient agreement for interpreting individual readings.

Dole et al. [16] compared CGM with capillary glucose in 12 experienced eumenorrheic female ultrarunners. A three-hour fasted outdoor run was followed by one hour of treadmill running with three 20-g oral glucose doses,

during both midfollicular and midluteal phases. Estimated mean differences between CGM and capillary glucose were −0.43 mmol/L (95% CI, −0.86 to −0.005) during fasted running and −1.02 mmol/L (95% CI, −1.63 to −0.42) during the subsequent carbohydrate-supplemented phase. Menstrual phase was associated with the discrepancy during treadmill running (p = 0.02), but not during the fasted phase. These average differences should not be applied as correction factors to individual readings. The principal results were checked against the published abstract; detailed methods were assessed in the thesis chapter [17].

The thesis chapter describing the Dole study identifies additional limitations relevant to interpretation [17] (Chapter 7). Capillary glucose was measured using a portable glucose meter rather than a laboratory reference analyser. Furthermore, the carbohydrate-supplemented treadmill phase always followed the fasted outdoor phase. Consequently, carbohydrate provision, accumulated exercise duration, and changes in the exercise setting cannot be separated as explanations for the larger discrepancy. The small sample and use of a single CGM model also limit generalisability.

Skroce et al. [18] assessed 10 trained men across cycling intensities. MARD remained below 15%, but the reported mean bias of 0.40 mg/dL coexisted with limits of agreement of approximately −26 to +27 mg/dL. CGM did not reproduce the observed increase in capillary glucose at higher intensity. Thus, favourable average accuracy should not be equated with precise individual readings or diagnostic validity for LEA.

The published abstract of Mason et al. [19] reports a similar distinction in 13 recreational runners: sensor readings tracked mean glucose trends, whereas point agreement was poorer, particularly at low glucose concentrations. This finding supports cautious interpretation of isolated readings; full-text methods were unavailable for assessment in this review.

For the present review, these studies provide evidence about measurement agreement, rather than diagnostic performance for LEA or REDs. These validation studies did not establish nocturnal measurement accuracy or validate a diagnostic glucose threshold for these conditions. A methodological implication is that low sensor readings require cautious interpretation before being attributed to inadequate energy availability. Future studies should evaluate measurement agreement under the intended monitoring conditions and, separately, determine whether CGM metrics provide diagnostic information beyond established nutritional and clinical assessment.

Table 1. Summary of primary reports included in the narrative synthesis

Study	Population and exposure	CGM finding and main qualification
Smith 2016 [5]	23 military volunteers completed crossover; severe energy deficit with exercise.	Lower mean sensor glucose and more time <3.9 mmol/L. Indirect population; energy and carbohydrate changed together; missing data.
Caldwell 2024 [4]	12 women; 14-day LEA/OEA crossover. CGM: OEA n=10, LEA n=11; days 7–14.	No significant difference in nocturnal mean (p=0.440) or minimum glucose (p=0.544). Post-breakfast iAUC increased; no clinical REDs validation.
Tarocchi 2024 [6]	8 adolescent male cyclists; 7 in final nutritional intervention. Estimated EA.	Improved EA without significant change in mean daily/night glucose. Small seasonal field study; no diagnostic validation.
Matkin-Hussey 2026 [7]	9 women with clinical REDs and 9 controls. Free-living monitoring.	No significant group differences in mean glucose or MAGE. Recovery-related behaviour; EA not calculated; analytical-window uncertainty.
Zignoli 2025 [9]	26 professional cyclists; 22 attended both annual camps.	Abstract only: no significant association of minimum overnight glucose with exercise expenditure or carbohydrate intake. Full methods unassessed.
Amatori 2026 [8]	15 male cyclists completed 7-day dietary crossover periods with similar reported energy intake.	Lower carbohydrate intake associated with lower mean glucose; training-related associations. EA not assessed.

Study	Population and exposure	CGM finding and main qualification
Bauhaus 2023 [15]	10 athletes; sensor vs laboratory capillary glucose at rest and in exercise protocols.	Agreement varied by condition; wide limits of agreement. One sensor model; no nocturnal or REDs validation.
Dole 2025 / thesis 2024 [16, 17]	12 female ultrarunners; prolonged running in two menstrual phases.	Sensor glucose is lower than capillary glucose. Published abstract and thesis assessed; final journal full text unavailable. One study.
Bowler 2025 [10]	12 racewalkers; repeated standardised periods.	Descriptive variability; no diagnostic thresholds.
Coates 2024 / preprint 2023 [12, 13]	11 athletes; overload training.	Exercise-specific findings; preprint methods. Linked to Ranahan.
Ranahan 2025 [14]	10 athletes; CGM n=8.	Recovery-related variability; linked analysis, not independent replication.
Hamilton 2024 [11]	9 female cyclists; 9-day camp.	Descriptive nocturnal profile; EA unmeasured and sensor lows unconfirmed.
Skroce 2025 [18]	10 male athletes; cycling validation.	Mean accuracy does not ensure precise individual readings; no LEA validation.
Mason 2024 [19]	13 runners; sensor agreement.	Trends versus point agreement; abstract-only assessment.

EA, energy availability; LEA, low energy availability; OEA, optimal energy availability; REDs, Relative Energy Deficiency in Sport. Counts refer to the stated analysis or intervention period, not necessarily complete paired datasets. Dole's article and thesis describe one study. Zignoli and Mason remain abstract-only; this table summarises a purposive narrative selection rather than an exhaustive systematic inventory. Fourteen publication-level entries are shown; Coates and Ranahan share a research programme. Article/preprint pairs and companion reports do not represent independent cohorts.

4 Discussion

The evidence examined in this review supports a distinction between the physiological responsiveness of interstitial glucose to under-fuelling and the diagnostic usefulness of CGM for identifying LEA or REDs. Glucose concentrations may decrease during substantial energy restriction, but this response does not establish that a particular CGM pattern can reliably distinguish athletes with REDs from those without the syndrome. The reviewed studies address different questions: responses to experimentally imposed energy deficits, associations with estimated EA, differences between clinically defined groups, and agreement between measurement methods. These forms of evidence are complementary, but they cannot substitute for one another.

The experimental evidence does not show a uniform glucose response to energy restriction. Smith et al. [5] observed lower interstitial glucose during severe deprivation combined with exercise, but energy and carbohydrate intake changed together and the sample comprised military personnel. Caldwell et al. [4] provide an important qualification: a longer, controlled LEA intervention in endurance-trained women did not produce significant changes in aggregate CGM metrics, including nocturnal mean glucose, although post-breakfast incremental glucose AUC increased and cycling performance declined. Tarocchi et al. [6] likewise found no significant change in mean daily or nocturnal glucose following improved estimated EA. These studies indicate that glucose responses depend on the exposure, population and metric, and that an apparently stable aggregate glucose profile cannot be taken as evidence of adequate EA.

The clinical findings of Matkin-Hussey et al. [7] highlight the importance of distinguishing a REDs diagnosis from nutritional exposure during the monitoring period. Prior clinical contact and emerging recovery behaviours may have influenced glucose profiles, while access to CGM readings prompted some participants to modify their fueling. Thus, monitoring itself may have altered the behaviour being observed. However, recovery-related changes remain a possible contributor to the findings rather than an established explanation for the absence of group differences, particularly because the additional analysis excluding participants with discordant estimated energy balance also found no glucose differences. Future longitudinal studies should document treatment timing, dietary changes and clinical status alongside CGM, while distinguishing energy balance from energy availability

and reporting monitoring windows consistently. This would help determine whether glucose metrics track current nutritional exposure, clinical recovery, or neither.

Diet and training further complicate interpretation. Amatori et al. [8] showed that lower carbohydrate intake was associated with lower mean glucose despite similar reported energy intake between conditions. Previous-day exercise was also associated with nocturnal glucose. Consequently, a lower CGM value may reflect several nutritional and exercise-related influences rather than a specific pathological state. Conversely, an apparently unremarkable glucose profile cannot be assumed to indicate adequate EA. Interpreting CGM without information on dietary intake, training and clinical presentation risks attributing excessive diagnostic meaning to a non-specific measurement.

Bowler et al. [10] provide descriptive context, while Coates et al. [12] and Ranahan et al. [14] highlight the importance of exercise conditions and recovery timing. Their findings do not establish diagnostic specificity for LEA or REDs.

Measurement uncertainty is a separate concern. Bauhaus et al. [15] demonstrated that small average differences between sensor and blood glucose could coexist with wide limits of agreement. This distinction matters when individual readings or threshold-based outcomes are used to classify athletes. It also indicates why correlation with blood glucose is insufficient to establish diagnostic validity. A sensor may detect a group-level response to an intervention while remaining insufficiently accurate for interpreting an isolated low value in an individual. Whether repeated or aggregated CGM measurements provide more reliable information for LEA or REDs assessment requires direct investigation. Dole et al. [16] additionally showed lower sensor than capillary glucose during prolonged running, but their protocol cannot isolate carbohydrate provision from accumulated exercise and changes in setting. These exercise findings do not directly establish nocturnal accuracy. Hamilton et al. [11] also identify unconfirmed nocturnal sensor lows as an interpretive limitation. Skroce et al. [18] and Mason et al. [19] reinforce the distinction between aggregate agreement and interpretation of individual readings.

The IOC framework provides an appropriate clinical context for these findings. Clinically low blood glucose is a potential, non-scored CAT2 indicator, and REDs assessment integrates multiple clinical domains and differential diagnoses. Although the consensus includes CGM among methods used to investigate glucose metabolism, this should not be interpreted as endorsement of a standalone diagnostic test [1, 2]. On the evidence reviewed here, CGM is best regarded as an investigational adjunct whose additional clinical value remains to be established.

Future studies should distinguish between detecting current energy restriction, identifying established REDs and monitoring recovery, because these aims may require different outcomes and reference assessments. Prospective research should combine standardized CGM protocols with clearly described clinical assessment, dietary intake, exercise expenditure and treatment status. Mean and minimum nocturnal glucose, variability and time below range should be evaluated separately, with explicit definitions of sleep periods, data completeness and artefact handling. Proposed thresholds would then require assessment of sensitivity, specificity and external validity in independent cohorts. Crucially, studies should determine whether CGM adds useful information beyond existing clinical assessment rather than merely demonstrating an association with one component of energy restriction.

4.1 Limitations of the review

This critical narrative review is not an exhaustive systematic review. The documented database searches were conducted in PubMed; database coverage, narrative selection and incomplete full-text access may have influenced the evidence considered. Zignoli et al. [9] were assessed only at abstract level. For Dole et al. [16], the published abstract was checked against a thesis chapter describing the study; the final journal full text was unavailable, and revisions between versions cannot be excluded. Caldwell et al. [4] were assessed with the supplied supplementary tables. Heterogeneous populations, devices, monitoring windows and definitions of energy deficiency limit direct comparisons. Military studies provide indirect evidence for athletes, and reports from the same experimental cohort must not be counted as independent confirmation. Study-level reporting uncertainties, including the analytical window in Matkin-Hussey et al. [7], further constrain interpretation. Coates et al. [12] were assessed through the published abstract and an earlier full preprint; post-review changes remain possible. Coates and Ranahan are linked analyses from the same research programme, with differing analysis samples. Contextual and validation studies were selected purposively; omission from the narrative does not establish ineligibility or poor quality. Mason et al. [19] were assessed only through the published abstract.

5 Conclusions

The reviewed evidence does not establish CGM as a standalone method for identifying or excluding LEA or REDs in athletes without diabetes. Glucose can respond to severe energy restriction, but aggregate profiles do not consistently distinguish nutritional exposures or clinical groups. Dietary composition, training, recovery and measurement uncertainty further limit specificity. CGM may contribute contextual information in research, but its added clinical value remains unproven. Future studies should use clearly defined nutritional and clinical reference assessments, standardised monitoring procedures and independent validation of any proposed diagnostic thresholds.

Disclosure

Author contributions

The manuscript has ten authors. Percentage contributions to the concept, methods and assumptions, and to the manuscript as a whole, are recorded in the separate author declaration. All authors have read and approved the manuscript.

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Institutional Review Board Statement

Not applicable. This narrative review analyses published literature and does not involve recruitment of human participants, animal experiments or access to identifiable individual-level data.

Informed Consent Statement

Not applicable. No individual participants were recruited and no identifiable personal data are reported.

Data Availability Statement

The evidence discussed is available in the cited publications, subject to publisher access conditions. The search export, selection log and extraction notes are retained in the review records.

Conflicts of Interest

The authors declare no conflicts of interest.

Declaration of generative AI and AI-assisted technologies in the writing process

During preparation of this manuscript, ChatGPT (OpenAI) was used to improve language and readability, formatting text, checking reference styles, and basic data analysis.

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