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Pre-Exercise Carbohydrate Intake and Reactive Low Glucose Concentrations in Athletes: Mechanisms, Individual Susceptibility and Practical Implications – A Narrative Review

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Abstract
Carbohydrate intake before endurance exercise can cause a transient decline in blood glucose at exercise onset. Its clinical and functional significance is less clear. This narrative review examines the underlying mechanisms, individual responses, and implications for nutrition and glucose monitoring. English-language literature identified through PubMed and Google Scholar searches in September 2026 was synthesised thematically. Experimental studies were considered alongside reviews, position statements, and methodological studies. Pre-exercise carbohydrate intake increases glucose and insulin concentrations. Muscle contraction adds a stimulus for glucose uptake, so disposal may temporarily exceed appearance. The resulting decline varies between individuals and nutritional conditions. Pre-exercise insulin concentrations and exercise modality do not consistently identify those who will develop low glucose. Evidence that susceptibility is reproducible under identical conditions remains limited. Transient low glucose is not consistently accompanied by symptoms, increased perceived exertion, or impaired performance. However, incomplete outcome assessment limits conclusions about its consequences. Continuous glucose monitoring adds uncertainty because

interstitial readings may differ from concurrent blood glucose. Interpretation should therefore integrate the glucose trajectory, episode duration, symptoms, performance, nutritional context, and measurement method. An early decline alone does not justify routinely avoiding pre-exercise carbohydrate. Strategies should meet exercise demands and accommodate individual tolerance. Repeated standardised studies are needed to establish which responses recur and affect performance.

Key words: pre-exercise carbohydrate; transient hypoglycaemia; endurance exercise; glucose kinetics; continuous glucose monitoring

1. Introduction

Carbohydrate is an important fuel during exercise, particularly at moderate-to-high intensities [1,2]. Muscle glycogen provides a major endogenous reserve. Its depletion during prolonged or repeated high-intensity exercise can limit the capacity to sustain work [1,2]. Carbohydrate provision should therefore reflect exercise duration, intensity, and the demands of training or competition [3]. Pre-exercise intake supports this provision by contributing to carbohydrate availability [2,3].

General recommendations suggest approximately 1–4 g/kg body mass within 1–4 h before endurance exercise [3]. The amount, timing, and source influence glucose and insulin responses, but metabolic differences do not consistently predict performance [4,5]. Intake shortly before exercise may leave insulin elevated when muscle contraction increases glucose uptake. Blood glucose can then decline during the transition from rest to exercise [6,7,8].

This decline can occur when glucose removal temporarily exceeds its entry into the circulation. Tracer studies show that blood glucose may fall even while glucose appearance remains elevated [4,9]. However, the magnitude and duration of the response differ between participants [6,7]. Understanding the mechanism is therefore insufficient to predict an individual's response or its consequences.

Prior feeding and insulin responses do not consistently explain this variation [6,7]. Low glucose is also not consistently accompanied by symptoms or increased perceived exertion [6,7,10]. Continuous glucose monitoring (CGM) adds a measurement issue: sensors measure interstitial glucose, which can differ from concurrent blood glucose [11,12,13]. Individual glucose profiles further complicate the interpretation of isolated readings [11,14].

This narrative review examines the mechanisms of transient low glucose after pre-exercise carbohydrate intake, individual susceptibility, and functional significance. It then considers how these findings can inform carbohydrate strategies and glucose monitoring during endurance exercise.

2. Materials and methods

English-language literature was identified through PubMed and Google Scholar searches conducted in September 2026. No publication-year restriction was applied. Both earlier experimental studies and recent work on exercise-associated glucose responses were considered.

Search terms addressed pre-exercise carbohydrate intake, blood glucose, transient or reactive hypoglycaemia, endurance exercise, performance, perceptual responses, individual variability, and CGM. Terms were combined according to the topic examined.

Abstracts were screened for relevance, followed by assessment of potentially relevant full texts. Selection focused on glucose metabolism after pre-exercise carbohydrate intake, individual responses, symptoms, and performance. Publications on nutritional strategies and glucose measurement were also considered.

Experimental studies were assessed alongside reviews, position or consensus statements, and methodological studies. Findings were synthesised thematically. No pooled effect estimate was calculated, and the certainty of evidence was not formally graded.

3. Results

The synthesis first examines glucose regulation after pre-exercise carbohydrate intake. It then considers nutritional decisions and the interpretation of low glucose during endurance exercise.

3.1. Physiological basis of the glycaemic response to pre-exercise carbohydrate

3.1.1. Metabolic response to pre-exercise carbohydrate ingestion

Carbohydrate ingestion increases circulating glucose through intestinal absorption and stimulates insulin secretion. The amount, timing, and type of carbohydrate influence the magnitude and duration of these responses. High-glycaemic-index (HGI) sources generally produce larger glucose and insulin responses than low-glycaemic-index (LGI) alternatives [4,5].

The ingested dose does not directly determine muscle carbohydrate oxidation. Exogenous carbohydrate must undergo gastric emptying, intestinal absorption, circulatory delivery, muscle uptake, and oxidation. Limitations at any of these stages can affect its use [15]. Blood glucose concentration captures only part of this process.

Febbraio et al. found greater glucose and insulin responses after an HGI meal than after an equivalent amount of LGI carbohydrate consumed 30 min before exercise [4]. Similarly, maltodextrin at 1 g/kg body mass increased pre-exercise glucose to approximately 7.4 mmol/L, compared with 4.7 mmol/L after placebo. Insulin was also higher after maltodextrin [7].

Insulin promotes muscle glucose uptake by stimulating glucose transporter 4 (GLUT4) movement to the cell membrane [16]. It also suppresses hepatic glucose production and inhibits lipolysis, reducing free fatty acid (FFA) availability [8,9]. The lower FFA availability observed after pre-exercise maltodextrin is consistent with this mechanism [7].

These changes can shift fuel use towards carbohydrate. Febbraio et al. reported greater glucose and total carbohydrate oxidation, with lower fat oxidation, after HGI intake [4]. This pattern was not universal. Kaviani et al. compared low- and moderate-GI bars providing 1.5 g/kg of available carbohydrate 1 h before cycling. Fat and carbohydrate oxidation did not differ significantly between the bar conditions [17]. GI therefore helps explain some metabolic responses but does not consistently predict substrate use.

Metabolic differences also need to be distinguished from performance outcomes. In Febbraio et al., different glucose, insulin, and substrate responses were not accompanied by a significant difference in work output [4,5].

Timing and prior nutrition shape the state at exercise onset. A longer interval after eating allows more time for glucose and insulin to decline [5]. Kondo et al. found higher FFA concentrations before glucose ingestion in the fasted condition. After 150 g of glucose, however, glucose and insulin at exercise onset were similar in fed and fasted conditions [6]. Earlier feeding can therefore alter the initial state without producing the same differences after a large glucose load. Exercise is introduced into this feeding-related metabolic state. Contraction adds a stimulus for muscle glucose uptake, making the balance between glucose appearance and removal central to the subsequent response [4,6,8].

3.1.2. Exercise onset and regulation of glucose kinetics

Blood glucose reflects the balance between the rate of appearance (Ra) and disappearance (Rd). Ra includes endogenous glucose production and absorbed glucose. Rd represents removal from the circulation, with contracting muscle becoming a major site of disposal. Blood glucose can

remain stable while both rates increase. Concentration alone therefore does not describe glucose turnover [8,9].

Contraction stimulates glucose uptake partly through GLUT4 movement to the muscle-cell membrane and transverse tubules. Insulin and contraction use partly distinct signalling pathways that converge on GLUT4 trafficking [8,16]. Their effects can occur together and may be additive.

Membrane transport is only one determinant of uptake. Glucose must also reach the muscle through the circulation, cross the capillary wall, and undergo intracellular phosphorylation and metabolism [18]. Insulin concentration alone therefore cannot describe the full uptake response during exercise.

Glucose appearance must increase sufficiently to match disposal. During fasting, endogenous production supplies most circulating glucose. Exercise stimulates hepatic glucose production, including glycogenolysis, to help meet muscle demand [8]. After feeding, absorbed glucose also contributes to Ra, while insulin suppresses hepatic output and promotes peripheral uptake [9]. The preceding nutritional state therefore affects both sides of the balance.

When carbohydrate is consumed shortly before exercise, insulin-mediated uptake may remain elevated as contraction begins. Rd can then rise faster than Ra [6,7,8]. Febbraio et al. demonstrated this imbalance using tracers: glucose appearance remained elevated after HGI intake, but disposal was disproportionately high early in exercise. Blood glucose consequently declined despite continued glucose delivery [4].

Studies without tracers can describe the trajectory but cannot separate the contributions of Ra and Rd. In Chryssanthopoulos et al., glucose fell by approximately 33–38% during the first 10 min after pre-exercise maltodextrin. Placebo values remained comparatively stable, and insulin also declined after exercise began [7]. These measurements establish the time course, not the specific change in glucose flux.

A temporary mismatch between appearance and disposal thus provides a mechanism for the early decline. Its interpretation requires a distinction between measured concentrations and directly assessed fluxes [4,8,9].

3.1.3. Transient decline in circulating glucose following pre-exercise carbohydrate ingestion

After glucose or HGI maltodextrin approximately 30 min before exercise, blood glucose can fall rapidly. In the examined protocols, the nadir occurred within roughly the first 15–20 min. Only some participants entered the study-defined hypoglycaemic range [6,7].

As glucose appearance and disposal become more closely matched, the decline may attenuate and glucose may recover during continued exercise [4,6]. Duration is therefore essential to interpretation. An early nadir followed by recovery differs from a sustained low-glucose episode.

Comparable carbohydrate intake also produces different trajectories between participants [6,7]. This variation leads to a separate question: which individual or contextual factors are associated with low glucose?

3.1.4. Interindividual variability and susceptibility to transient low glucose

Kondo et al. reported transient hypoglycaemia in 5 of 16 participants when fasted and 7 of 16 when fed. Only three developed it in both conditions [6]. Feeding status alone therefore did not consistently identify responders.

Insulin-related associations also depended on nutritional state. In the fasted condition, responders had higher insulin at exercise onset. A higher insulinogenic index was associated with lower glucose after 15 min. In the fed condition, onset insulin was similar between

responders and non-responders [6]. The association observed during fasting did not provide a consistent predictor across conditions.

Chryssanthopoulos et al. found transient hypoglycaemia in 6 of 11 participants during cycling and 7 of 11 during running. All six cycling responders also responded during running. Their nadir was approximately 3.3 mmol/L in both modalities, compared with approximately 5.4–5.5 mmol/L in non-responders [7]. This overlap suggests an individual component across modalities.

However, insulin at exercise onset was not significantly related to the subsequent decline or glucose after 20 min. Indirect estimates of insulin sensitivity did not explain responder status [7]. A mechanistic role for insulin therefore does not establish its value as a predictor.

Broader CGM evidence describes overall variability rather than responses to a specific carbohydrate challenge. Flockhart and Larsen report glucose coefficients of variation of approximately 21 ± 4% in endurance athletes and 16 ± 3% in healthy controls [11]. These secondary data cannot establish susceptibility to pre-exercise carbohydrate-induced low glucose.

Bowler et al. assessed glucose profiles across two four-day periods with standardised diet and exercise [14]. This design describes variability under controlled conditions. It does not directly test whether an identical pre-exercise carbohydrate challenge repeatedly causes hypoglycaemia. The evidence therefore demonstrates individual variation more clearly than stable susceptibility. Consistency across exercise modalities is suggestive, but repeated identical exposures are needed to establish reproducibility [6,7,14].

3.1.5. Functional relevance of transient low glucose

The mechanisms above explain how glucose can decline, but not whether the decline causes symptoms or impairs performance. Crossing a biochemical threshold and experiencing a functional consequence are separate outcomes [6,7,10].

Febbraio et al. found different metabolic responses without a significant difference in work output [4]. Chryssanthopoulos et al. likewise observed an early glucose decline without a significant reduction in time to exhaustion compared with placebo [10]. These findings argue against assuming that every early decline reduces endurance capacity.

Kaviani et al. found that low- and moderate-GI bars improved cycling time-trial performance compared with placebo [17]. This supports a potential benefit of carbohydrate provision, but the comparison did not directly test the consequences of transient hypoglycaemia.

An early decline followed by recovery also differs from sustained carbohydrate insufficiency. During prolonged exercise, declining endogenous stores can increase reliance on circulating and ingested carbohydrate [2]. Evidence from that later state cannot automatically explain a brief early nadir [2,4].

Functional interpretation therefore requires symptoms, perceived exertion, and direct performance outcomes alongside the glucose trajectory [6,7,10]. These distinctions provide the basis for practical decisions about pre-exercise intake.

3.2. Practical implications for pre-exercise carbohydrate intake

3.2.1. Should athletes avoid carbohydrate intake shortly before exercise?

Intake within 30–60 min before exercise can raise glucose and insulin, followed by an early glucose decline. Reviews do not show a consistent adverse effect on performance under these conditions [5,19].

For example, Chryssanthopoulos et al. administered 75 g of glucose 30 min before running at approximately 70% of maximal oxygen uptake (VO_2max). Glucose fell during early exercise, but time to exhaustion was not significantly lower than after placebo [10]. Febbraio et al. also

found no significant difference in work output between HGI, LGI, and control conditions [4]. These results apply to the tested protocols rather than proving that all low-glucose episodes are inconsequential.
Only a subset of participants develops low glucose after comparable intake [6,7]. Applying the same restriction to every athlete would therefore disregard both individual variation and the potential value of carbohydrate provision.
Food-based studies reviewed by Naderi et al. generally reported neutral or beneficial performance outcomes after intake approximately 45–60 min before exercise [3]. The possibility of an early decline should consequently be considered alongside the fuel demands of the task.
The evidence does not support routine carbohydrate avoidance during the final hour before exercise. Timing, amount, and source should instead be selected according to exercise demands and the athlete's response [3,5].

3.2.2. Timing of pre-exercise carbohydrate intake

Timing influences the glucose and insulin concentrations present when exercise begins. The recommendation of approximately 1–4 g/kg body mass within 1–4 h offers a practical range, not a universally optimal interval [3].
Earlier intake allows more time for glucose and insulin to decline. Intake within approximately 60 min may leave both elevated and produce a larger early glucose fall [19]. However, a different glucose profile does not by itself establish a performance disadvantage.
Timing comparisons reviewed by Rothschild et al. produced limited or inconsistent performance differences. One study favoured intake 30 min rather than 120 min before exercise [5]. Trials using intake approximately 30 min before exercise also found pronounced glucose changes without consistent performance impairment [4,10]. A longer feeding interval therefore does not reliably improve performance.
Timing alone cannot explain the response because individuals differ after comparable exposure [6]. Carbohydrate amount and food form also influence the outcome. Studies using food approximately 30–90 min before exercise reported beneficial, neutral, and food-dependent effects [3].
The feeding interval should therefore be considered alongside amount, food form, previous intake, gastrointestinal tolerance, and exercise demands. No single interval is supported as optimal for all athletes or tasks [3,5].

3.2.3. Carbohydrate amount and glycaemic characteristics

Carbohydrate amount and GI influence the feeding response, but neither independently guarantees better performance [3,5]. The dose must first be considered in relation to the task and starting carbohydrate availability.
The reviewed studies do not show a consistent proportional performance improvement as the pre-exercise dose increases [5,19]. Larger intakes may therefore provide no additional benefit in some protocols.
Prior underconsumption or incomplete glycogen restoration can increase the importance of pre-exercise intake [5,20]. The recommended range of approximately 1–4 g/kg body mass should be adapted to preceding nutrition, exercise demands, and tolerance [20].
GI informs expectations about glucose and insulin responses. LGI carbohydrate generally produces smaller increases, which may preserve FFA availability and favour fat oxidation. HGI intake tends to suppress lipolysis and increase carbohydrate oxidation [19]. These mechanisms justify comparing sources but do not establish which source will improve performance.

Performance findings remain mixed. Some studies favour LGI intake, while others show no difference [5,19,21]. Benefits have been reported more often in time-to-exhaustion tests than in time trials [5]. The outcome measured therefore affects interpretation.
The HGI–LGI comparison by Febbraio et al. illustrates this limitation: metabolic responses differed, but work output did not [4]. GI should consequently be treated as one characteristic of a feeding strategy rather than a stand-alone performance predictor.
Whole foods also differ in carbohydrate density, water and fibre content, processing, structure, and digestibility [3]. Equal carbohydrate doses can therefore require different portion sizes and have different tolerability.
In one study reviewed by Naderi et al., approximately 70 g of carbohydrate required about 650 g of cooked potatoes or 430 g of cooked lentils [3]. Large portions can be difficult to consume close to exercise, even when they meet the carbohydrate target.
Food comparisons also make the specific effect of GI difficult to isolate. Some studies favoured oats or lentils, whereas other comparisons found no performance difference [3]. Differences in processing, fibre, and food structure prevent attributing these results solely to GI.
Food-based options can perform similarly to sports products. Raisins and a sports gel, each providing 1 g/kg body mass of carbohydrate, produced comparable performance in trained cyclists [3]. Products offering smaller portions or convenient dosing may nevertheless suit some practical settings.
The amount should meet exercise demands, and the source should deliver it in a tolerable form. GI can help explain the metabolic response, but feasibility and tolerance also determine the usefulness of the strategy [3,5].

3.2.4. Individualised decision-making for pre-exercise carbohydrate intake

Planning begins with exercise duration, intensity, and purpose. Prolonged or high-intensity sessions generally require greater carbohydrate availability [5,20]. Competition and key performance sessions usually prioritise this availability. Selected lower-intensity sessions may use reduced availability when it matches the intended training stimulus [5].
Previous intake and recovery define the nutritional starting point. Incomplete glycogen restoration can increase the need for pre-exercise carbohydrate [20]. The same athlete may therefore require different strategies before otherwise similar sessions.
The amount and source must then fit the available feeding interval. Portion size, fibre, digestibility, palatability, and gastrointestinal tolerance affect how much can be consumed comfortably [3]. The review of cross-country skiing nutrition also emphasises individual tolerance [22].
Glucose measurements can inform this process, but one low reading does not establish persistent susceptibility. Similar exposures produce different responses between participants [6,7]. A strategy should therefore be evaluated through the athlete's repeated experience as well as its biochemical effects.
If adverse responses recur, timing, amount, or form can be adjusted while maintaining adequate carbohydrate availability [20]. The change should address the observed problem within its nutritional and exercise context.
The revised strategy should be tested during training before competition. Assessment should include symptoms, performance, gastrointestinal comfort, and practical feasibility [3]. This connects the feeding plan to outcomes that matter to the athlete.

3.3. Interpreting low glucose during endurance exercise

3.3.1. Biochemical classification and physiological significance

Study-defined glucose thresholds classify a biochemical response [6,7]. They do not establish its duration, symptoms, or performance consequences [10,11]. These outcomes require separate assessment.

Only some participants cross the threshold after comparable carbohydrate intake. Low glucose often occurs early and may resolve during continued exercise [6,7,10]. Interpretation therefore requires the trajectory, duration, and recovery as well as the nadir.

Exercise changes both muscle glucose uptake and hepatic output [8,9]. Similar concentrations can thus arise under different nutritional and exercise conditions. The reviewed evidence does not establish an athlete-specific threshold that reliably identifies functional impairment [11,14].

The measurement compartment must also be identified. A low interstitial sensor reading is not equivalent to laboratory-confirmed low blood glucose [11,12]. Biochemical classification is therefore a starting point for interpretation rather than a complete assessment.

3.3.2. Symptoms and perceptual responses

A glucose decline describes a metabolic response; symptoms describe the participant's experience. The reviewed studies do not establish a consistent relationship between the nadir and symptom severity [6,7]. However, the completeness of symptom assessment limits this conclusion.

Perceived exertion is also distinct from hypoglycaemia-specific symptoms. Fatigue or weakness during exercise is not specific to low glucose. Coincidence in time is therefore insufficient to establish causation.

Chryssanthopoulos et al. reported a marked early glucose decline, but perceived exertion was identical to placebo at 15 and 30 min. Glucose later recovered while perceived exertion continued to rise [10]. These different trajectories show that the outcomes need not change together. Hypoglycaemia-specific symptoms were not systematically assessed, so the study cannot establish that every episode was asymptomatic.

Incomplete symptom assessment also limits other work [23]. Absence of a reported symptom must be distinguished from systematic assessment showing no symptoms. Standardised measurements are needed to relate the biochemical response to the participant's experience.

Reviews likewise describe early glucose declines without consistent changes in symptoms or perceived exertion [5,19]. Nutritional state, exercise characteristics, and individual responses can influence these perceptions [5].

Glucose, symptoms, and perceived exertion should therefore be recorded separately and compared over time. This allows their relationship to be assessed without assuming that one establishes the significance of another.

3.3.3. Low glucose and exercise performance

Performance effects require direct performance measurements. Crossing a biochemical threshold does not establish reduced exercise capacity [6,7,10].

Some studies documented transient low glucose without a time trial or time-to-exhaustion test [6,7]. They establish occurrence under the tested conditions but cannot quantify a performance cost. Completing a prescribed exercise bout is insufficient to exclude impairment.

Chryssanthopoulos et al. measured both glucose and time to exhaustion. Glucose recovered after the early nadir, and endurance capacity was not significantly lower than after placebo [10]. This result does not support a decrement in that protocol. It also does not directly isolate the effect of the low-glucose episode from the other effects of carbohydrate intake.

Reviews report no consistent detrimental performance effect associated with transient low glucose [5,19]. This argues against inferring impairment from glucose alone. However, a non-significant difference does not prove equivalence or exclude an effect under other conditions.
Neither the magnitude of an early decline nor a biochemical threshold provides a validated estimate of performance loss in the reviewed evidence [5,11]. This is a limitation of current knowledge, rather than proof that glucose and performance are unrelated.
Sustained carbohydrate insufficiency presents a different problem. During prolonged exercise, declining endogenous stores increase reliance on circulating and ingested carbohydrate [2]. Performance may be affected when supply cannot meet demand [24].
These findings should not be transferred directly to an early nadir followed by recovery. Mata et al. describe relatively few studies with adverse performance effects after pre-exercise intake; most reported unchanged or improved performance [2].
The functional meaning of low glucose therefore depends on its time course, nutritional context, and measured performance outcome [4,6,7,10]. A brief decline should neither be assumed to impair performance nor be treated as universally inconsequential.

3.3.4. Individual susceptibility and reproducibility

Differences between participants suggest an individual component to the response [5,6,7]. A single trial, however, identifies only what occurred under the conditions tested. Stable susceptibility requires evidence that the response recurs.
Baseline glucose has not been established as a reliable predictor in healthy endurance athletes. Porter et al. studied post-meal exercise in groups with different glycaemic status, including type 2 diabetes [23]. Their findings cannot validate prediction in an athletic population. Insulin-related associations also vary across pre-exercise carbohydrate studies and feeding conditions [6,7].
Consistency across modalities is relevant but does not establish test–retest reproducibility. Cycling and running expose participants to different exercise stimuli [7]. Reproducibility requires repeated exposure to the same stimulus under comparable conditions.
Bowler et al. assessed two four-day periods with standardised diet and exercise [14]. This addresses variability during repeated controlled periods, not an identical pre-exercise carbohydrate challenge. Broader CGM profiles describe another aspect of variation [11].
The reviewed designs therefore answer related but distinct questions. Kondo et al. compared feeding states, Chryssanthopoulos et al. compared modalities, and Bowler et al. examined repeated glucose profiles [6,7,14]. Together with the review literature, these studies provide limited evidence for reliably reproducible susceptibility [5].
Repeated trials should standardise prior nutrition, carbohydrate exposure, and exercise. Until such evidence is available, a single episode should be interpreted within its observed context rather than used to assign a permanent responder status.

3.3.5. CGM measurement and interpretation

CGM measures interstitial rather than blood glucose [11,13]. Differences between the two compartments can become important during rapid changes after feeding or exercise onset.
Bauhaus et al. compared CGM with laboratory measurements of capillary blood glucose in healthy athletes. Mean absolute relative difference (MARD) was 8 ± 6% during fasted rest, 17 ± 12% during postprandial rest, 22 ± 24% during moderate-intensity exercise, and 18 ± 17% during high-intensity exercise [12]. Agreement limits were wider during exercise. Accuracy was therefore dependent on the measurement conditions.
Near a classification threshold, a modest measurement difference can change whether a value is labelled low. A sensor-derived nadir should consequently be identified as interstitial glucose, not assumed to represent simultaneous blood hypoglycaemia.

Thresholds introduce a further limitation. CGM targets were developed mainly for diabetes care; their functional meaning in healthy athletes is less established [13]. Crossing a sensor threshold alone does not demonstrate symptoms or impaired performance.

DuBose et al. illustrate the distinction between a nadir and a sustained event in healthy individuals without diabetes. Among participants with exercise data, 36% recorded a sensor nadir below 70 mg/dL and 8% below 54 mg/dL. Only two met the event definition of glucose below 54 mg/dL for at least 15 consecutive minutes [25]. The population was not restricted to endurance athletes, and low sensor values were not systematically confirmed with concurrent blood measurements. These findings therefore distinguish isolated readings from sustained sensor events without establishing the prevalence of confirmed hypoglycaemia in athletes.

The direction of change also matters. The same value may occur during a post-meal rise, an exercise-related decline, or recovery. Recent intake, activity, and the surrounding glucose trajectory are therefore needed for interpretation [11,25].

Short monitoring periods may overemphasise isolated readings or miss recurring patterns [11,14]. Longer observation can describe these patterns more fully, but does not replace repeated identical challenges when the question concerns susceptibility to pre-exercise intake.

CGM can thus describe trajectories but cannot automatically confirm low blood glucose. When precise biochemical classification is needed, an appropriate blood glucose reference measurement is required [11,12,13].

3.3.6. Final synthesis

Interpretation begins with the measurement method, glucose trajectory, and episode duration. Symptoms and performance must then be assessed separately. Reproducibility requires an additional step: repeated comparable exposures.

The reviewed studies show that early low glucose can occur without documented symptoms or performance impairment. Incomplete outcome assessment prevents treating this as proof that every episode is harmless. An early nadir followed by recovery must also be distinguished from sustained carbohydrate insufficiency.

Individual variation is evident, but prediction and reproducibility remain uncertain. Interstitial measurement differences add another source of uncertainty. Together, these limitations favour assessment of the complete response over interpretation based on a threshold alone.

4. Discussion

4.1. Interpretation of the main findings

The central finding is a distinction between a plausible mechanism and an established consequence. Feeding and contraction can jointly increase glucose uptake, allowing disposal to exceed appearance temporarily [8,9]. This explains an early decline. It does not, by itself, establish inadequate carbohydrate availability or impaired performance. The metabolic differences without changes in work output reported by Febbraio et al. illustrate this distinction [4].

Study design limits the conclusion. Trials without direct performance endpoints cannot establish a performance cost [6,7]. Trials that found no significant decrement do not exclude effects under all conditions [4,10]. Moreover, comparing carbohydrate with placebo tests the combined effects of feeding, rather than isolating the causal effect of low glucose. Interpretation should therefore remain specific to the exposure, trajectory, and outcome measured.

The time course further separates a brief early nadir from sustained low glucose during prolonged exercise. Recovery argues against interpreting the early episode as persistent carbohydrate insufficiency. Conversely, unchanged performance after a brief decline does not make prolonged low glucose inconsequential [2,24].

4.2. Individual susceptibility and practical interpretation

Overlap between responders suggests an individual component, but the available studies do not establish stable susceptibility. Feeding-state comparisons, modality comparisons, and repeated CGM profiles address different questions [6,7,14]. A single episode can guide further observation but cannot support a permanent classification.

Practical decisions should begin with exercise demands and preceding nutrition. Timing, amount, source, and tolerance then guide carbohydrate provision. A stable glucose profile is not necessarily evidence of better performance. If adverse responses recur, targeted changes can be tested during training while preserving adequate carbohydrate availability [3,5,20].

4.3. Measurement and limitations of the evidence

CGM introduces uncertainty in the timing and magnitude of a glucose nadir because interstitial readings can differ from concurrent blood measurements [11,12,13]. Episode duration and trajectory add context, but precise biochemical classification still requires an appropriate reference method. Sensor-defined events in non-athletic populations cannot directly establish the prevalence or consequences of hypoglycaemia in endurance athletes [25].

The evidence is heterogeneous in prior nutrition, carbohydrate exposure, exercise protocol, measurement, and outcome assessment. Some studies lack direct performance endpoints, and symptom assessment is inconsistent [6,7,10]. Findings from other populations offer context but limit generalisation [23]. Reviews and position statements support interpretation; they are not independent experimental observations [5,20].

This narrative synthesis provides neither a pooled estimate nor a formal assessment of evidence certainty. Its conclusions describe patterns in the selected literature. They do not quantify an individual's probability of low glucose or the expected performance cost.

4.4. Priorities for further research

Research should test repeated identical exposures in the same participants. Prior nutrition, carbohydrate intake, and exercise should be standardised. Validated glucose measurements should be combined with symptom assessment and direct performance tests.

These designs could link the magnitude and duration of a decline to its consequences. They would also separate reproducibility under controlled conditions from variation between contexts. This would provide a stronger basis for individual nutritional decisions than an isolated threshold crossing [6,7,10,11,12,14].

5. Conclusions

Transient low glucose after pre-exercise carbohydrate intake may reflect a temporary excess of disposal over appearance. Its occurrence varies between individuals and conditions. A low value alone does not establish symptoms or impaired performance. Interpretation requires the time course, nutritional and exercise context, measurement method, and directly assessed outcomes.

The reviewed evidence does not support routine avoidance of pre-exercise carbohydrate solely to prevent an early decline. Strategies should meet exercise demands and accommodate individual tolerance. CGM readings require contextual interpretation, and one episode does not establish stable susceptibility. Repeated standardised studies are needed to identify responses that recur and have functional consequences.

Disclosure

Author Contributions:

Conceptualization: K.D. Methodology: K.D and K.G. Literature search and selection: K.G, N.W, and A.G. Analysis and interpretation of the reviewed literature: M.S, S.K, and K.L. Data

curation: K.S and J.D. Verification of study descriptions and citation accuracy: K.S and A.G. Writing—original draft preparation: K.D., N.W, M.S, and J.D. Writing—review and editing: K.G, S.K, K.S, A.G, J.D, K.L, and L.J. Supervision: L.J and J.D. Project administration: K.D. All authors have read and agreed to the published version of the manuscript.

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AI

During the preparation of this manuscript, the authors used OpenAI tools (ChatGPT and Codex) to assist with locating relevant literature, improve academic English, correct grammatical and stylistic errors, and identify inconsistencies. These tools also assisted with drafting and restructuring passages to improve clarity, coherence, and the logical flow of the manuscript. AI tools are not listed as authors. The human authors remain responsible for verifying the cited sources, assessing the accuracy of the content and its interpretation, and approving the final manuscript.

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