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Continuous Glucose Monitoring (CGM) in Long-Distance Runners: A Narrative Review of Performance Optimization and Hypoglycemia Prevention

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Abstract

Background. Continuous Glucose Monitoring (CGM) is increasingly used by ultra-endurance athletes to refine complex fueling strategies. However, transitioning this clinical tool into a performance monitoring remains difficult, largely due to the interstitial-to-blood lag time that can distort real-time data interpretation.

Aim. This study aims to synthesize current literature to evaluate the application of CGM data during extreme exercise, examining how exercise intensity, dietary protocols, and technical artifacts influence metabolic data interpretation and fueling efficacy.

Material and Methods. A review of literature was performed with a primary focus on literature from 2020 to 2026, while including foundational technical research. The analysis examined the impact of a broad range of physical and technical factors on the practical application and limitations of CGM.

Results. CGM accuracy, measured by MARD, often drops during high-intensity exercise and dehydration. There are clear differences between interstitial readings and blood glucose levels that athletes must account for. Data integrity is affected by factors like thermal stress, caffeine intake, and localized tissue compression. Findings also show that metabolic responses to CGM vary depending on sex and dietary status, such as LCHF. While the technology helps track carbohydrate absorption during 120 g/h protocols, it can also mask a systemic energy deficit if the athlete relies only on stable readings. Sensor lag remains a critical issue, where glucose levels on the screen do not always reflect actual glycogen availability or energy status.

Conclusions. To improve performance, athletes should focus on trend arrows rather than static numbers. CGM is a monitoring tool, not an absolute source of physiological truth. Using a structured protocol helps manage sensor lag and prevents metabolic crashes. This approach makes it easier to stay on track with fueling and pacing during extreme effort.

Keywords: Continuous Glucose Monitoring (CGM), ultra-endurance, metabolic pacing, carbohydrate intake, glycemic variability, MARD, sports nutrition

1. Introduction

Long-distance running at marathons and ultra-marathon level creates an extreme physiological demand [1,2]. It is a challenge of metabolic efficiency and in order to meet high energy requirements, runners rely heavily on carbohydrates for fuel [3,4]. Maintaining this fuel supply is not merely beneficial but essential for sustaining exercise intensity. As stated by Hearris et al. [3] glycogen, which is stored in the liver and skeletal muscles, is limited to roughly 1800 to 2000 kilocalories. Once the energy reserves are exhausted, runners experience a severe drop in performance, clinically recognized as bonking or hitting the wall [1,3,5]. Crucially, maintaining blood glucose homeostasis is necessary not only for powering the working muscles but also for functioning of the central nervous system [3]. Should blood sugar levels drop, the nervous system faces immediate compromise, manifesting clinically as dizziness, loss of motor coordination, and a rapid decline in running performance [4,6,7].

For many years, blood glucose monitoring relied on standard finger-prick glucometers [8]. This method has significant disadvantages during a race, because it is invasive, highly inconvenient to execute while running, but most importantly it provides only a static snapshot of blood sugar [4,8]. Without real-time data, athletes depended on subjective measures such as the Rating of Perceived Exertion (RPE) [7,9]. Stress and adrenaline often mask early signs of physiological depletion, thus perceived exertion can be inaccurate during a race [4]. The revolutionary shift towards Continuous Glucose Monitoring (CGM) was a technological breakout from clinical diabetes care into high-performance athletics [6,9]. By measuring interstitial glucose levels continuously, CGMs act like a dynamic, 24/7 metabolic "video" rather than a traditional "still photo" [4,9].While recent clinical research highlights the effectiveness of CGM in diagnosing metabolic dysfunctions [10], translating this diagnostic tool into ultra-endurance settings introduces physiological challenges.

The main clinical reason for using CGMs in endurance sports is the prevention of hypoglycemia [6,11]. Rather than reacting to symptoms of hypoglycemia post-onset, runners can use real-time glucose trend arrows to detect falling blood sugar and ingest carbohydrates pre-emptively [11]. Monitoring glucose continuously highlights the crucial concept of metabolic inter-individuality for precision fueling [3,4]. Athletes have unique metabolic profiles and consuming the same carbohydrate gel can have vastly different glycemic responses [4,9]. The objective of this review is to synthesize the current literature to equip distance runners and sports medical professionals with a clear evidence-based strategy for integrating CGM into modern marathon training and racing protocols [4,6,9].

2. Methods

2.1. Search Strategy

To establish a solid evidence base for this review, a search was performed focusing on high-impact databases, specifically PubMed, Google Scholar and Scopus. The search strategy was designed to combine classic metabolic theories with the latest practical applications of wearable technology. To achieve this, the review includes foundational research on muscle glycogen and psychophysiological models dating back to 2003 such as the work of Nybo [12] and Hearris et al. [3]. This ensures a strong understanding of the biological mechanisms behind endurance. At the same time, the analysis of sensor technology and performance protocols was intentionally restricted to articles published between 2020 and 2026. By focusing on the last six years for technical data, the discussion remains relevant to the latest sensors of third-generation e.g. Libre

3 and Dexcom G7. Including older data would risk including models with higher error margins (MARD), which do not meet the precision standards required for elite marathon fueling.

The search of literature was categorized into four thematic domains, which were intersected using Boolean operators (AND to integrate the separate categories and OR to expand search within specific clusters). This approach ensured a comprehensive retrieval of data at the intersection of bioengineering, sports science and endocrinology:

- The Technology domain focused on the hardware and data acquisition methods, utilizing keywords such as “Continuous Glucose Monitoring”, “CGM”, “interstitial glucose dynamics”, “wearable metabolic sensors”, “MARD” and “trend arrow algorithms”.
- The Physiological domain targeted the metabolic and molecular impact of long-distance exertion with terms such as “glycemic variability”, “exercise-induced hypoglycemia”, “GLUT4 translocation”, “metabolic flux” and “central fatigue”.
- The Performance domain contextualized the findings within the specific demands of distance running with a focus on “marathon running”, “endurance performance”, “precision fueling”, “120 g/h carbohydrate intake”.
- The Sex-specific and Recovery domain addressed emerging research gaps, focusing on “female athlete metabolism”, “menstrual cycle metabolism”, “nocturnal glycemia” and “overtraining detection”.

2.2. Selection Process

Specific criteria were applied to the gathered literature to maintain the focus of this review on high-performance athletics and ensuring the synthesis of all 31 selected sources. The selection prioritized studies with high methodological quality, specifically peer-reviewed randomized controlled trials, systematic reviews and meta-analyses.

- Inclusion criteria: Articles were eligible if they were published in English-language, peer-reviewed journals. The scope was strictly limited to studies involving healthy endurance athletes (marathon, ultra-marathon). Research was considered eligible if it explored CGM as a primary tool for fueling optimization, real-time metabolic monitoring, sex-specific metabolic profiling or the mitigation of exercise induced hypoglycemia. Seminal physiological studies were included regardless of the 2020 threshold if they provided primary evidence of core biological mechanisms.
- Exclusion criteria: Since a significant portion of CGM literature is dedicated to clinical diabetes management without athletic performance component, these studies were

largely excluded unless they offered fundamental physiological principles applicable to healthy cohorts. Moreover, research involving sedentary populations, animal models or sensors with Mean Absolute Relative Difference (MARD) exceeding 15% was discarded to ensure the final recommendations are based on accurate and human-centric data relevant to modern athletic standards.

3. Physiological Background

3.1. Glycogen Depletion and the Metabolic Demands of Endurance

Endurance performance in disciplines like the marathon relies heavily on carbohydrate oxidation. While fats contribute significantly to the total energy pool, glucose remains arguably the most critical fuel for sustaining high-intensity efforts [13]. The challenge is primarily physiological. An average adult typically stores only about 100 g of glycogen in the liver and 400 g in skeletal muscle [3]. Murray and Rosenbloom [13] note, elite athletes can push these boundaries; through chronic training adaptations and supercompensation protocols, their total reserves may expand to between 700 and 900 g.

Despite these adaptations, the metabolic flux required to sustain a competitive pace (often between 75% and 92% of VO_2 max) can rapidly deplete even the most well-stocked reserves [14]. At these competitive intensities, the active muscle mass becomes highly dependent on carbohydrate oxidation, accelerating the degradation of endogenous glycogen stores [13,15]. This depletion triggers metabolic fatigue, often referred to as hitting the wall, where the body can no longer sustain the ATP resynthesis rates necessary to maintain speed [13]. To delay this decline, modern fueling strategies focus on high-rate exogenous carbohydrate delivery [2].

Recent insights from Morton et al. [2] highlight that ingesting carbohydrates during a race does more than just power the working muscles; it serves a vital role in sparing hepatic glycogen. By maintaining blood glucose levels, athletes protect the liver's capacity to fuel the central nervous system, effectively delaying the onset of both peripheral and central fatigue [2,12]. Ultimately, these endogenous stores are insufficient to sustain a competitive pace for the duration of the event. These internal energy reserves are ultimately insufficient to reach the finish line at elite speeds, This limitation prompts Burke et al. [14] to underscore the need for greater precision in personalized intake.Traditional guidelines provide the solid theoretical basis for carbohydrate requirements. Real-time glucose monitoring, however, offers a practical means to manage these metabolic constraints for the individual athlete [9]. Yet, these constraints are not uniform. Biological sex, for instance, introduces entirely new complexities into the fueling equation.

3.2. Sex-Based Differences in Metabolism and Fueling

These metabolic complexities are frequently overlooked in the literature, largely because foundational sports nutrition guidelines are still predominantly based on male cohorts. As Kuikman et al. [16] highlight, women account for a mere 11% of participants in studies examining acute carbohydrate fueling. Yet, biological sex fundamentally alters the parameters of substrate oxidation and energy storage. Elite female runners typically possess less lower-body fat-free mass than their male counterparts. This anatomical difference translates to an absolute glycogen storage capacity that is approximately 32% smaller, leaving a much narrower margin for error during prolonged exertion [17].

To compensate for these smaller reserves, the female endocrine system facilitates distinct metabolic adaptations. Estrogen improves insulin action and prioritizes lipid oxidation over carbohydrates during exercise, effectively sparing limited glycogen stores [18]. This metabolic advantage, however, is continuously challenged by the hormonal fluctuations of the menstrual cycle. During the follicular phase, higher estrogen levels generally improve insulin sensitivity and maximize glucose tolerance [19]. The luteal phase presents a contrast. Marked by a lower estrogen-to-progesterone ratio, this phase frequently induces decreased insulin sensitivity and triggers highly variable glycemic responses during exertion [18,19]. It must be noted that the magnitude of these physiological shifts remains a subject of ongoing debate. Several studies report no significant differences in insulin sensitivity or glycogen synthesis capacity between sexes or across menstrual phases, highlighting the lack of universal consensus [16,19].

These physiological shifts can make standardized and male-centric fueling recommendations less effective for women. A more individualized approach is necessary. Here, continuous glucose monitoring emerges as a tool for navigating the unique metabolic profile of the female athlete. Rather than relying on static guidelines, real-time data allow runners to dynamically adapt their nutritional interventions to their immediate hormonal and metabolic status [9].

3.3. Glycemic Stability and Management of Central Fatigue

Beyond preserving peripheral muscle function, dynamic glucose management is critical for sustaining cognitive drive. The brain acts as the ultimate governor of endurance performance. Its capacity to recruit working muscle is highly sensitive to systemic energy availability, and as Nybo [12] demonstrated, exercise-induced hypoglycemia directly attenuates central nervous system activation. This reduces voluntary force production. This central fatigue mechanism

illustrates a protective response: when circulating glucose drops, the brain actively downregulates motor output to protect cerebral metabolism.

These metabolic perturbations are not limited to the late stages of a race. Poorly timed pre-exercise carbohydrate ingestion can trigger reactive hypoglycemia right at the onset of exertion. This causes a rapid decline in blood glucose that compromises psychological motivation and metabolic stability [9]. As glucose remains the primary fuel for the central nervous system, mitigating these sharp fluctuations influences the subjective racing experience. Backhouse et al. [20] established that stabilizing blood glucose through continuous carbohydrate delivery significantly enhances positive affective states and lowers the rating of perceived exertion (RPE). By maintaining euglycemia, athletes experience less perceived suffering, enabling them to tolerate high-intensity efforts for longer durations [12,20].

The necessity of this psychological and physiological stability is particularly evident in extreme ultra-endurance environments. Recent continuous glucose monitoring data from the 100-mile Lake Biwa ultramarathon revealed that top finishers consistently exhibited smaller glycemic fluctuations and avoided the severe glucose drops that affected slower runners [21]. Faster athletes proactively consumed higher amounts of carbohydrates to stabilize their glucose curves, especially during the early stages of the race [5,21]. In this context, real-time glucose monitoring transcends basic metabolic tracking. It emerges as a tool for managing the body-brain axis, allowing runners to proactively avoid the glycemic crashes that precipitate central fatigue and psychological collapse [5,9].

3.4. Carbohydrate Absorption Limits and Muscle Protection

While continuous glucose delivery shields the central nervous system, it must also satisfy the metabolic demands of the working muscles. During prolonged exercise, muscle glucose uptake increases via the translocation of GLUT4 transporters [22]. However, the rate of intestinal absorption remains a limiting factor. Since the SGLT1 transporter saturates at approximately 60 g/h, athletes must utilize multiple transportable carbohydrates [15]. By combining glucose and fructose to engage the GLUT5 pathway, oxidation rates can reach 90–120 g/h [7,23].

Pushing these limits to 120 g/h offers structural benefits beyond mere energy provision. Field data from Viribay et al. [23] demonstrate that high carbohydrate intake during intense efforts significantly limits exercise-induced muscle damage (EIMD). This is evidenced by a reduction in the systemic efflux of biomarkers such as creatine kinase (CK) and lactate dehydrogenase (LDH). Preventing this damage is critical, as EIMD compromises GLUT4 function and limits

subsequent glycogen resynthesis [7]. Consequently, these authors indicate that ultra-high carbohydrate delivery preserves neuromuscular function and accelerates recovery . Achieving such ultra-high intake rates without gastrointestinal distress requires progressive gut training to increase the density of intestinal transporters [15,23]. For the athlete, this management requires high precision, further justifying the use of real-time monitoring to ensure that fueling strategies maintain systemic stability [4,9]. While the physiological rationale for ultra-high carbohydrate delivery and continuous monitoring is compelling, translating these theoretical advantages into real-world performance requires navigating significant technological constraints and metabolic nuances. These practical limitations, along with their implications for fueling strategies, will be explored in the subsequent sections.

4. Technological Considerations

4.1. The Interstitial Gap: Blood-to-Interstitial Dynamics and Lag-Time

Continuous glucose monitoring (CGM) systems do not provide direct measurements of systemic blood glucose. Instead, they estimate glycemia based on the chemical composition of the interstitial fluid (ISF). The sensor filament resides within the dermal layer, where it monitors glucose availability in the interstitial compartment. This measurement depends entirely on the passive movement of glucose from the capillary bed into the interstitial compartment. This movement creates a physical barrier, separating the real-time metabolic state of the blood from the environment accessible to the sensor [8,24].

The diffusion process results in an unavoidable delay known as lag-time [8]. While this offset is relatively stable under resting conditions, Bauhaus et al. [24] emphasize that the physiological demands of endurance exercise disrupt these kinetics. Factors such as fluctuations in body temperature, the redistribution of blood flow toward working muscles and changes in systemic acidity all alter the diffusion equilibrium between metabolic compartments. As a result, the standard resting lag-time of 5–10 minutes often extends to 15 minutes. During periods of rapid metabolic flux, this delay can reach 25 minutes. Such a gap typically grows in proportion to exercise intensity, meaning the sensor is often least accurate exactly when the runner requires the most precise data [4,24]. These technological constraints have a direct impact on athletic decision making. During rapid shifts in workload, such as a high-intensity start or a steep uphill incline, the immediate uptake of blood glucose is not reflected in the ISF until much later [24]. The numerical value on the screen represents a metabolic history rather than the current physiological state. Relying on these historical figures to trigger nutritional interventions is a

strategic error. It leads to delayed underfueling. To manage this inherent delay, runners must adopt a proactive fueling protocol. Carbohydrate intake must be timed to match upcoming physical demands instead of responding to visible drops on a graph [4].

4.2. Analytical Accuracy During Exertion: MARD, Dietary Influences and Physical Interferences

The Mean Absolute Relative Difference (MARD) is the standard metric for CGM accuracy. It represents the average deviation between sensor readings and reference blood glucose measurements.

According to Bauhaus et al. [24], while a MARD below 10% indicates clinical reliability (e.g. 8% during fasted rest), precision declines during physical exertion, with error rates reaching 18-22%. At higher intensities, the rapid turnover of glucose alters its flux into the ISF. These shifts in localized blood flow widen the gap between capillary blood and interstitial readings. The device is often at its least accurate exactly when the runner requires the most reliable data.The extreme distances of ultramarathons introduce additional complexity. John et al. [25] noted that athletes running up to 230 kilometers often maintain stable glucose lines, even while experiencing severe energy deficits. This stability can be misleading and mask underlying endocrine disruptions. As distance increases, the correlation between interstitial glucose and actual energy status weakens. It leads to a metabolic drift, where CGM data no longer accurately reflect systemic fuel reserves.

Beyond physiological stress, the stability of the CGM signal is influenced by the athlete's background diet. For instance, following a 28-day low-glycemic index (LGI) diet attenuates glycemic variability and reduces the amplitude of glucose spikes compared to high-glycemic index diets . Rather than simply altering the clarity of the monitor's data, this dietary approach stabilizes the underlying physiological baseline.It demonstrates that CGM readings are not just a reflection of the race itself, but are modulated by nutritional choices made weeks in advance [26].

Mechanical and environmental factors distort signal reliability of the device. Variations in skin temperature and fluid loss impact the electrochemical stability of the sensors, causing the data to drift. Athletes also frequently deal with pressure-induced sensor dips. Hydration packs or tight clothing restrict local perfusion and trigger false hypoglycemic readings [26]. Chemical interference poses a similar challenge. High doses of paracetamol or vitamin C can cause falsely elevated levels, while salicylates may lower the values. The number on the screen is a product

of biological, dietary, mechanical and chemical variables. It remains a proxy rather than a direct measurement of blood glucose [27].

4.3. Data Translation: Rate of Change (ROC) and Proactive Fueling

Static glucose values provide limited insight during high-intensity exercise due to the physiological lag-time in interstitial monitoring [8]. Because of this delay, the Rate of Change (ROC), represented by trend arrows, serves as the primary navigational tool. As Eichenlaub et al. [28] outline,visually categorize the direction and magnitude of this immediate glucose flux, forming the critical basis for proactive interventions. In an endurance context, these arrows act as predictive vectors, allowing runners to anticipate a metabolic drop before the deficit appears in the interstitial fluid [11].

Downward arrows during high-intensity efforts signal a strict requirement to ingest carbohydrates. This proactive intake accounts for the physiological lag and mitigates the impending drop. While CGM-informed fueling aligns intake with real-time fluctuations, it does not eliminate the sensor's inherent latency. The metabolic pacing modifies systemic glucose dynamics but fails to alter subjective fatigue or blood lactate levels [4]. Data tracking must therefore be integrated with the athlete's physiological tolerance. Continuous monitoring requires a structured protocol to prevent data overload and glucorexia, which is an obsession with minor, non-physiological fluctuations [9].

Performance optimization fundamentally differs from clinical management. For healthy individuals, CGM is a tool for self-optimization rather than disease treatment [8]. Endurance athletes regularly experience extreme glucose variability that exceeds medical norms, including episodes of exercise-induced hyperglycemia [6]. In a race, the objective is metabolic availability, not glycemic stability. Proactive fueling serves as an anticipatory strategy to stay ahead of the metabolic curve. Ultimately, the CGM remains a decision-support tool, not a source of absolute physiological truth [9]. Table 1 outlines how to adjust carbohydrate intake based on specific ROC trend arrows.

Table 1. Metabolic Pacing Strategy: CGM Trend Interpretation and Strategic Response

Glycemic Trajectory (ROC)	Metabolic Etiology	Acute Nutritional Intervention	Confounding Variables
Rapid Drop (↘↘)	Acute exogenous fuel deficit; high glycolytic flux.	Immediate 15-30 g fast-acting CHO bolus; prioritize glucose/maltodextrin.	Potential compression lows if postural; caffeine masks subjective perception of this deficit.
Stable Low (→)	Maintenance of normoglycemia despite severe energy deficit; decoupling of glucose and actual energy status.	Proactive fueling mapped to physical demands (e.g., 60-90 g/h); avoid reactionary fasting.	Normal physiological baseline in LCHF/fat-adapted profiles.
Sudden Rise (↗↗)	Acute workload shift (e.g., steep incline) triggering hepatic glucose output; or rapid gastric emptying of HGI.	Maintain planned intake; do not cease fueling to avoid rebound hypoglycemia.	Interstitial sensor lag (5-15 min delay vs. capillary blood) during rapid glycemia excursions.
High Variability (〜)	Gastrointestinal ischemia and disrupted epithelial barrier function; gut mucosal stress.	Reduce acute intake rate; prioritize small, frequent sips using 2;1 glucose-fructose ratio.	High risk of severe GI distress at workloads requiring >90-120 g/h
Nocturnal Low (↓)	Post-exercise glycogen resynthesis; skeletal muscle glucose extraction.	Review pre-sleep nutrition; evaluate systemic Low Energy Availability (LEA) risk.	Frequently overlaps with postural mechanical failures (artifacts).

Sources: Metabolic etiologies, acute nutritional interventions, and confounding variables synthesized from [4,6,9,11,23,25,29,30]

Notes: ROC: Rate of Change; CHO: Carbohydrates; LEA: Low Energy Availability; LCHF: Low-Carbohydrate High-Fat; HGI: High-Glycemic Index; GI: Gastrointestinal.

5. Discussion

5.1. High-Volume Protocols (120 g/h): Metabolic Efficacy vs. Intestinal Limits

Historically, the maximum rate for exogenous carbohydrate oxidation was limited to 90 g/h [2,15]. Ingesting 120 g/h tests the physiological limits of intestinal absorption and imposes extreme metabolic demand.

Viribay et al. [23] confirmed this rate is feasible for elite athletes, where high-volume feeding blunts post-race creatine kinase efflux, indicating reduced muscle damage. High-volume feeding also sustains neuromuscular function and accelerates the recovery of high-intensity running capacity [7]. High exogenous carbohydrate availability saturates the systemic circulation, which spares intramyofibrillar glycogen and protects excitation-contraction coupling [3,7]. However, maximal supply carries a high physiological cost for the gastrointestinal (GI) tract. Arribalzaga et al. [1] identified GI distress as the primary barrier to reaching these intake levels. Unabsorbed nutrients in the intestinal lumen cause osmotic stress and fluid shifts, disrupting gastric emptying and triggering acute diarrhea and vomiting [1,2]. Elite runners, however, can tolerate 120 g/h without GI breakdown [23]. Tolerance depends strictly on systematic gut training. Repeated exposure to massive carbohydrate loads upregulates intestinal transporters and without this preparation, 120 g/h protocols significantly increase the risk of GI failure [15].

A rigid 120 g/h target ignores biological heterogeneity. Exogenous carbohydrate requirements vary based on metabolic rates, body mass, and sex [17]. John et al. [25] observed that during extreme ultramarathons, the efficiency of high-intake protocols fluctuates as energy deficits accumulate and metabolic responses to sugars become unstable over long distances. The 120 g/h threshold is an individual target, not a general rule. Real-time monitoring via CGM allows for the tracking of glycemic variability and adjusting intake based on metabolic flux. These data permit the runner to match fuel supply to actual demand, securing metabolic availability without exceeding GI capacity [6,9].

5.2. Metabolic Pacing vs. Static Refueling Protocols

Traditional carbohydrate refueling relies on rigid, standardized dosages, ignoring real-time physiological fluctuations. Continuous glucose monitoring replaces this static model with metabolic pacing, allowing athletes to titrate intake based on immediate interstitial feedback. While CGM-informed feeding stabilizes glucose trajectories and minimizes variability, it does

not reduce RPE, blood lactate, or heart rate. This confirms its role as a strictly strategic tool for objective pacing rather than a source of acute sensory relief [4].

Performance data from the Lake Biwa 100 confirm that elite finishers maintain glucose within a tighter range than lower-ranked runners, whose unregulated fluctuations correlate directly with velocity decay [21]. Elite athletes do not chase glucose spikes; they prioritize homeostatic preservation through proactive titration. Maintaining a glucose floor prevents metabolic collapse. Endurance success depends on minimizing the amplitude between peaks and nadirs rather than merely maximizing acute carbohydrate availability [5,21].

Extreme distances degrade data reliability. Mileage itself dictates monitoring accuracy. As race duration extends, severe caloric deficits accumulate while interstitial glucose often remains within normal ranges. This physiological compensation creates metabolic drift. The decoupling of fuel supply and interstitial readings becomes highly unstable beyond 200 kilometers. Monitor stability can actively mask energy deficits, endocrine disruptions and muscle damage. Sole reliance on CGM during extreme ultramarathons poses a severe strategic risk [25].

5.3. Nocturnal Hypoglycemia: Metabolic Recovery and Diagnostic Artifacts

Nocturnal hypoglycemia is prevalent among elite endurance athletes. Interstitial readings often fall below 70 mg/dL during sleep. This state does not automatically signal pathology. As Schubert-Olesen et al. [11] explain, depleted skeletal muscles prioritize glycogen resynthesis following exhaustive exercise, extracting circulating glucose to restore intracellular stores during recovery. Yet, chronic overnight patterns demand scrutiny. Persistently low readings serve as an early biomarker for low energy availability (LEA) [9]. Excessive training loads overwhelm cellular homeostasis and lead to mitochondrial impairment, which deteriorates systemic glucose tolerance [31].

Analyzing nocturnal data requires separating metabolic reality from technical artifacts. Continuous glucose monitors are susceptible to physical pressure; sleeping on the device induces localized tissue compression. This force restricts interstitial fluid perfusion around the sensor filament and resulting occlusion produces rapid, false plummets known as compression lows. Practitioners must isolate true physiological hypoglycemia from postural mechanical failures before altering nutritional protocols [26].

5.4. Sex-Specific Physiology and Hormonal Fluctuations

Current sports nutrition and CGM guidelines rely disproportionately on male-only cohorts, producing flawed metabolic generalizations. With female athletes remaining underrepresented in endurance research, applying male-derived fueling protocols directly to female physiology ignores fundamental sex-based differences in carbohydrate metabolism [16].

Ovarian hormones dictate substrate partitioning and systemic glycemic control. By improving insulin action and prioritizing lipid oxidation, estradiol promotes intramuscular glycogen sparing, while progesterone antagonizes insulin sensitivity. Glucose tolerance and exogenous carbohydrate requirements fluctuate between the follicular and luteal phases [18,19]. These shifts complicate data interpretation in female runners. Depending on the menstrual phase, a stable glucose trace can reflect different states of energy availability. Without hormonal context, apparent stability masks critical fuel deficits, repositioning continuous monitoring from a simplistic fueling gauge into a diagnostic tool for Relative Energy Deficiency in Sport (RED-S) [9].

5.5. Metabolic Modulators: Adenosine Antagonism and Nutritional Adaptation

Caffeine alters the perception of systemic fatigue by targeting the central nervous system through adenosine receptor antagonism. Suppressing pain perception and lowering the rate of perceived exertion (RPE), this mechanism allows athletes to operate under an illusion of adequate fueling despite systemic depletion [29]. As this supplementation effectively decouples subjective fatigue from objective metabolic data, continuous glucose monitors may register a severe energetic deficit, while the athlete feels energized. Such metabolic mismatches demand rigorous contextual interpretation to prevent underfueling errors [30].

Background dietary interventions modulate baseline glycemic profiles. Examining fat-adapted athletes on low-carbohydrate, high-fat (LCHF) protocols, Flockhart and Larsen [6] reported distinct sensor traces characterized by restricted glycemic variability and a lower physiological glucose floor. In this population, low monitor values reflect a systemic adaptation prioritizing lipid oxidation rather than an acute metabolic crisis. Low-glycemic index (LGI) diets show a comparable effect, stabilizing the metabolic baseline to prevent severe acute fluctuations [26]. Continuous glucose monitors therefore never measure glucose as an isolated variable, as the recorded profile remains a complex product of baseline nutrition, external stimulants, and cellular adaptation.

6. Practical Limitations and Future Perspectives

Continuous glucose monitors act as a physiological proxy, measuring interstitial fluid rather than capillary blood or intramuscular glycogen. Rapid metabolic shifts expose an interstitial-to-blood lag of 5 to 15 minutes [4,27]. Environmental variables in ultra-endurance racing degrade data integrity. High sweat rates and mechanical friction compromise sensor adhesion, causing frequent device detachment [4]. Localized tissue compression from body position restricts interstitial perfusion, generating false hypoglycemic readings during sleep or rest [27]. Strict synchronization protocols demand frequent proximity to a receiver and exceeding these synchronization windows triggers data loss [9].

Unfiltered access to continuous telemetry introduces psychological risks. Athletes risk developing data obsession, emerging in clinical literature as glucorexia. By becoming over-sensitive to benign, nonphysiological interstitial fluctuations, runners alter behavioral responses. A fear of minor glucose drops dictates premature carbohydrate intake and the athlete becomes entirely decoupled from internal sensory inputs [9].

Future diagnostics will transition from single-analyte tracking to multiplexed biosensing. Hardware developers are integrating real-time sweat analysis to quantify electrolyte depletion, while microneedle arrays will measure interstitial lactate, ketones, and glucose. Artificial intelligence algorithms synthesize these multi-substrate inputs to construct personalized prediction models, forecasting metabolic risks prior to systemic failure [27].

7. Conclusions

Continuous interstitial glucose telemetry should be treated as an advisory metric rather than an absolute directive. The technology provides a view of metabolic trends, but it does not eliminate the necessity for proactive nutritional planning. Integration of CGM into ultra-endurance contexts represents a shift toward anticipatory metabolic management, yet its success depends on the user's ability to contextualize raw data.

Interstitial readings are highly susceptible to misinterpretation due to external factors, including caffeine-induced adenosine antagonism, temperature shifts, postural shifts, and baseline metabolic adaptations. Relying on uncontextualized data inevitably generates tactical fueling errors, as interstitial glucose may decouple from actual systemic energy status during extreme exertion.

Managing the 120 g/h carbohydrate protocol requires continuous surveillance to map individual absorption kinetics and prevent rebound hypoglycemia. Glycemic monitoring provides the

foundation for modern metabolic pacing, provided the athletes acknowledge structural hardware constraints and physiological lag times. CGM data should support, not replace, a well-planned nutritional strategy.

Disclosure

Author's contribution

Conceptualization: Dominika Dołęga-Mostowska;
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check: Julia Sochowska, Urszula Kacprzak; formal analysis: Julia Danieluk, Justyna Wójcik; investigation: Dominika Dołęga-Mostowska and Natalia Adamaszek; resources: Michalina Flejszman, Julia Sochowska; data curation:Agnieszka Buczkowska;
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