endocrines

MDPI

Review

Exercise-Related Glycemic Fluctuations in Type 1 Diabetes: Mechanisms and Integrated Insulin–Carbohydrate Strategies in the Context of Diabetes Technologies

Filomena Mazzeo [1,2,†], Gabriele Ferrara [3,†], Fiorenzo Moscatelli [4,*], Antonietta Monda [5], Antonietta Messina [6], Maria Ruberto [4], Nicola Mancini [4], Raffaele Ivan Cincione [7], Gianluca Russo [3], Salvatore Allocca [3], Marco La Marra [3], Pasquale Perrone [4], Girolamo Di Maio [4], Maria Casillo [3], Giovanni Messina [3], Mario Ruggiero [2], Maria Giovanna Tafuri [8,‡] and Vincenzo Monda [9,‡]

1 Department of Law, Economics and Human Sciences (DiGiES), Mediterranea University of Reggio Calabria, Via dell'Università, 25, 89124 Reggio Calabria, Italy; filomena.mazzeo@uniparthenope.it
2 Department of Medical, Human Movement and Well-Being Sciences, University of Naples Parthenope, 80133 Naples, Italy; mario.ruggiero@phds.uniparthenope.it
3 Unit of Dietetics and Sports Medicine, Section of Human Physiology, Department of Experimental Medicine, University of Campania "Luigi Vanvitelli", 80138 Naples, Italy; gabriele.ferrara@unicampania.it (G.F.); gianluca.russo@unipegaso.it (G.R.); salvatore.allocca@unicampania.it (S.A.); marco.lamarra@unicampania.it (M.L.M.); maria.casillo@unicampania.it (M.C.); giovanni.messina@unicampania.it (G.M.)
4 Department of Education and Sport Sciences, Pegaso University, 80143 Naples, Italy; maria.ruberto@unipegaso.it (M.R.); nicola.mancini@unipegaso.it (N.M.); pasquale.perrone@unipegaso.it (P.P.); girolamo.dimaio@unipegaso.it (G.D.M.)
5 Department of Human Science and Quality of Life Promotion, San Raffaele Telematic University, 00166 Rome, Italy; antonietta.monda@uniroma5.it
6 Department of Precision Medicine, University of Campania "Luigi Vanvitelli", 80138 Naples, Italy; antonietta.messina@unicampania.it
7 Department of Clinical and Experimental Medicine, University of Foggia, 71122 Foggia, Italy; ivan.cincione@unifg.it
8 Department of Literary, Linguistic and Philosophical Studies, Pegaso University, 80143 Naples, Italy; mariagiovanna.tafuri@unipegaso.it
9 Department of Economics, Law, Cybersecurity and Sports Sciences, University of Naples Parthenope, 80131 Naples, Italy; vincenzo.monda@uniparthenope.it
* Correspondence: fiorenzo.moscatelli@unipegaso.it
† These authors contributed equally to this work.
‡ These authors also contributed equally to this work.

Abstract

Background/Objectives: Regular physical exercise is strongly recommended for individuals with type 1 diabetes mellitus (T1DM) because of its beneficial effects on cardiovascular fitness, insulin sensitivity, metabolic control, and overall health. Nevertheless, participation in physical activity remains limited, largely due to the fear of exercise-induced hypoglycemia and glycemic instability. Glycemic responses to exercise in T1DM are influenced by the interaction between exercise modality, circulating insulin levels, nutritional status, and diabetes technologies. Continuous aerobic exercise, resistance training, high-intensity interval exercise, and mixed intermittent activities elicit distinct metabolic and hormonal responses, resulting in heterogeneous glycemic trajectories. This narrative review aimed to provide a clinically oriented synthesis of the physiological mechanisms underlying exercise-related glycemic fluctuations in T1DM and to discuss integrated insulin- and carbohydrate-based strategies to support safer participation in physical activity in the context of modern diabetes technologies. **Methods**: A structured narrative review was conducted using PubMed/MEDLINE, Scopus, and complementary searches in Google Scholar to identify experimental studies, observational studies, systematic reviews, consensus statements, and clinical guidelines focused on exercise-related glycemic responses in individuals

Academic Editors: Marina Basina and Daniela Foti

Received: 14 March 2026
Revised: 15 May 2026
Accepted: 19 May 2026
Published: 21 May 2026

 https://doi.org/10.3390/endocrines7020022

with T1DM. Only articles published in English were considered. Evidence was selected and synthesized according to relevance to exercise modality, insulin therapy strategies, carbohydrate management, and diabetes technologies, including continuous glucose monitoring, continuous subcutaneous insulin infusion, and automated insulin delivery systems. The final narrative synthesis was based on 44 selected studies, reviews, consensus statements, and guidance documents considered most relevant to the objectives of this narrative review. **Results**: Available evidence indicates that continuous moderate-intensity aerobic exercise is most consistently associated with progressive glucose declines and increased risk of hypoglycemia, particularly when performed in the presence of elevated insulin on board. In contrast, resistance exercise and short-duration high-intensity or anaerobic exercise more frequently induce stable glycemia or transient hyperglycemia through adrenergic stimulation and increased hepatic glucose output. Mixed and intermittent exercise modalities often produce more variable responses depending on exercise sequencing, nutritional status, and insulin exposure. Across studies, integrated adjustment of basal and prandial insulin doses together with individualized carbohydrate supplementation emerged as the most effective strategy to reduce exercise-related glycemic instability. Continuous glucose monitoring and insulin pump technologies improved glucose trend awareness and management flexibility; however, physical exercise remains a challenging condition for current automated insulin delivery algorithms and still requires active user-driven decision-making. **Conclusions**: Exercise management in T1DM should be based on an individualized interpretation of exercise modality, glucose trends, insulin exposure, and nutritional context rather than on fixed glucose thresholds alone. Combining anticipatory insulin adjustments, tailored carbohydrate strategies, and appropriate use of diabetes technologies may substantially reduce glycemic variability and improve confidence toward physical activity participation. Structured education and individualized clinical guidance remain essential to translate physiological knowledge into effective real-world exercise management.

Keywords: type 1 diabetes; physical exercise; glycemic variability; insulin therapy; pharmacology; sport

1. Introduction

Type 1 diabetes mellitus (T1DM) is a chronic autoimmune disease characterized by pancreatic β-cell destruction and lifelong dependence on exogenous insulin therapy. Alongside the burden of daily glucose management, individuals with T1DM are exposed to an increased risk of cardiovascular and metabolic complications. Regular physical exercise is therefore strongly recommended because of its beneficial effects on cardiovascular fitness, insulin sensitivity, metabolic control, and overall health [1–5]. Despite these benefits, participation in structured physical activity remains lower than recommended in many individuals with T1DM, largely because exercise is frequently associated with unpredictable glycemic fluctuations and fear of hypoglycemia [6]. In individuals without diabetes, endogenous insulin secretion decreases rapidly at the onset of exercise, while glucagon and catecholamines increase to sustain glucose availability. In T1DM, this physiological regulation is often altered, since circulating insulin levels are determined by prior exogenous administration and cannot be rapidly adjusted in response to exercise demands [7].

Glycemic responses to exercise in T1DM are influenced by several interacting factors, including exercise modality, insulin on board, nutritional status, baseline glycemia, and individual fitness level [8]. Continuous aerobic exercise is generally associated with progressive glucose declines and increased risk of hypoglycemia, particularly when circulating insulin levels remain elevated. Conversely, high-intensity or anaerobic exercise may induce transient hyperglycemia through adrenergic stimulation and increased hepatic glucose output. These heterogeneous responses make exercise management particularly challenging in real-world settings [9–13].

Current clinical recommendations emphasize two main approaches to mitigate glycemic instability during exercise: adjustment of insulin therapy and appropriate carbohydrate intake [14]. Both strategies must be tailored to the characteristics of the planned or unplanned activity and to the individual's treatment regimen, whether based on multiple daily injections or continuous subcutaneous insulin infusion [15]. Nevertheless, the practical application of these recommendations in real-life settings remains challenging, particularly due to inter- and intra-individual variability and the complexity of decision-making surrounding insulin dosing and nutritional strategies [12].

In recent years, technological advances such as continuous glucose monitoring (CGM) and automated insulin delivery systems have provided new opportunities to support glucose management around exercise. While these tools have significantly improved overall glycemic control, physical activity continues to represent a critical scenario in which algorithm performance may be suboptimal, requiring user-driven adjustments and informed decision-making [16].

Fear of hypoglycemia, particularly delayed and nocturnal hypoglycemia after late-day exercise, remains one of the main psychological and practical barriers to regular physical activity in individuals with T1DM [17–19]. This concern is even more relevant in subjects with impaired hypoglycemia awareness or attenuated counterregulatory responses, emphasizing the need for individualized and clinically applicable preventive strategies [20].

The complex interaction between exercise modality, insulin exposure, carbohydrate intake, and diabetes technologies in determining glycemic variability during physical activity is summarized in Figure 1.

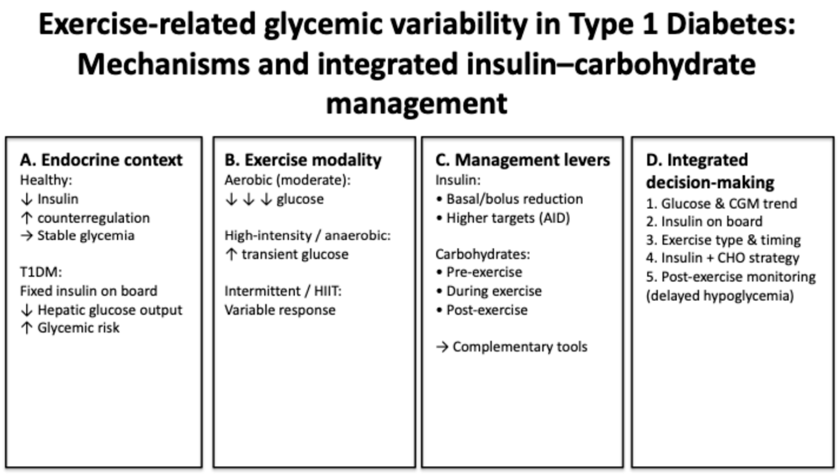

Figure 1. Conceptual overview of exercise-related glycemic variability in type 1 diabetes. Glycemic responses during physical activity are influenced by the interaction between exercise modality, circulating insulin levels, carbohydrate intake, and diabetes technologies. Continuous aerobic exercise is more commonly associated with glucose declines, whereas high-intensity or anaerobic exercise may induce transient hyperglycemia. Integrated insulin and nutritional strategies are required to support safer exercise participation.

2. Materials and Methods

2.1. Study Design

This study was conducted as a structured narrative review aimed at providing a clinically oriented synthesis of current evidence regarding exercise-related glycemic fluctu-

https://doi.org/10.3390/endocrines7020022

ations in individuals with T1DM. Particular attention was given to the interaction between exercise modality, insulin therapy, carbohydrate management, and diabetes technologies in real-world exercise settings. Because the objective of the review was conceptual and practical integration rather than quantitative comparison of outcomes, a narrative synthesis approach was adopted.

2.2. Information Sources and Search Strategy

A structured literature search was performed using major biomedical databases (e.g., PubMed/MEDLINE and Scopus), complemented by targeted searches in Google Scholar to retrieve relevant consensus statements, clinical practice guidelines, and position papers. Only articles published in English were considered. The literature search was updated until October 2025. The search strategy combined controlled terms and free-text keywords related to T1DM and exercise, including: “type 1 diabetes”, “exercise”, “physical activity”, “aerobic”, “resistance training”, “high-intensity”, “interval”, “hypoglycemia”, “post-exercise hypoglycemia”, “hyperglycemia”, “insulin on board”, “carbohydrate intake”, “nutrition”, “medical nutritional therapy”, “continuous glucose monitoring”, “insulin pump”, and “automated insulin delivery”. Boolean operators were used to refine the search (AND/OR), and reference lists of key articles were screened to identify additional pertinent studies (snowball approach).

2.3. Eligibility Criteria

Eligible records included (i) randomized and non-randomized controlled trials, (ii) observational studies, (iii) acute exercise physiology studies conducted in individuals with T1DM, (iv) systematic reviews and meta-analyses, and (v) consensus statements, guidelines, and expert position papers relevant to exercise management in T1DM. Articles were considered if they addressed at least one of the following domains: glycemic trajectories during exercise, determinants of glycemic variability (e.g., exercise intensity/duration, baseline glycemia, insulin on board), insulin adjustment strategies (basal/bolus modifications), carbohydrate dosing approaches, and the role of CGM/CSII/AID in the context of physical activity. Exclusion criteria were: studies not involving T1DM participants (unless used solely for mechanistic background), reports lacking exercise-related glycemic outcomes or practical management implications, case reports with limited generalizability, and non-peer-reviewed sources without sufficient methodological detail. When multiple publications reported overlapping data, the most complete or methodologically robust report was prioritized.

Preference was given to studies specifically addressing exercise-related glycemic responses in individuals with T1DM, including studies evaluating aerobic exercise, resistance exercise, high-intensity interval exercise, and mixed/intermittent exercise modalities. Articles lacking clinically relevant information on glucose responses, insulin adjustment, carbohydrate management, or exercise-related metabolic outcomes were excluded from the final synthesis.

2.4. Study Selection

Titles and abstracts were initially screened to assess relevance to the scope of the review. Full texts were then evaluated to confirm eligibility and to extract information aligned with the predefined domains (exercise modality, insulin regimen/technology, nutritional strategy, and glycemic outcomes). Particular attention was given to studies describing real-world decision variables (e.g., timing of exercise relative to meals, pre-exercise glucose levels, and insulin on board), as these elements directly inform clinical and practical recommendations. The literature selection process is summarized in Figure 2. Overall, 2741 records were identified through database searching, and 194 additional records were identified through reference lists, clinical guidelines, consensus statements, and position papers. After duplicate removal, 1986 records were screened by title and abstract. Of these, 1318 records were excluded because they were not focused on T1DM, were unrelated to exercise, did not report glycemic outcomes, or were not original research or guidance documents. A total of 668 full-text articles were assessed for eligibility, and 624 were excluded because of wrong population, lack of specific data on exercise-related glycemic responses, absence of information on insulin or carbohydrate management during exercise, or insufficiently relevant outcomes. The final narrative synthesis was based on 44 selected studies, reviews, consensus statements, and guidance documents considered most relevant to the objectives of this narrative review.

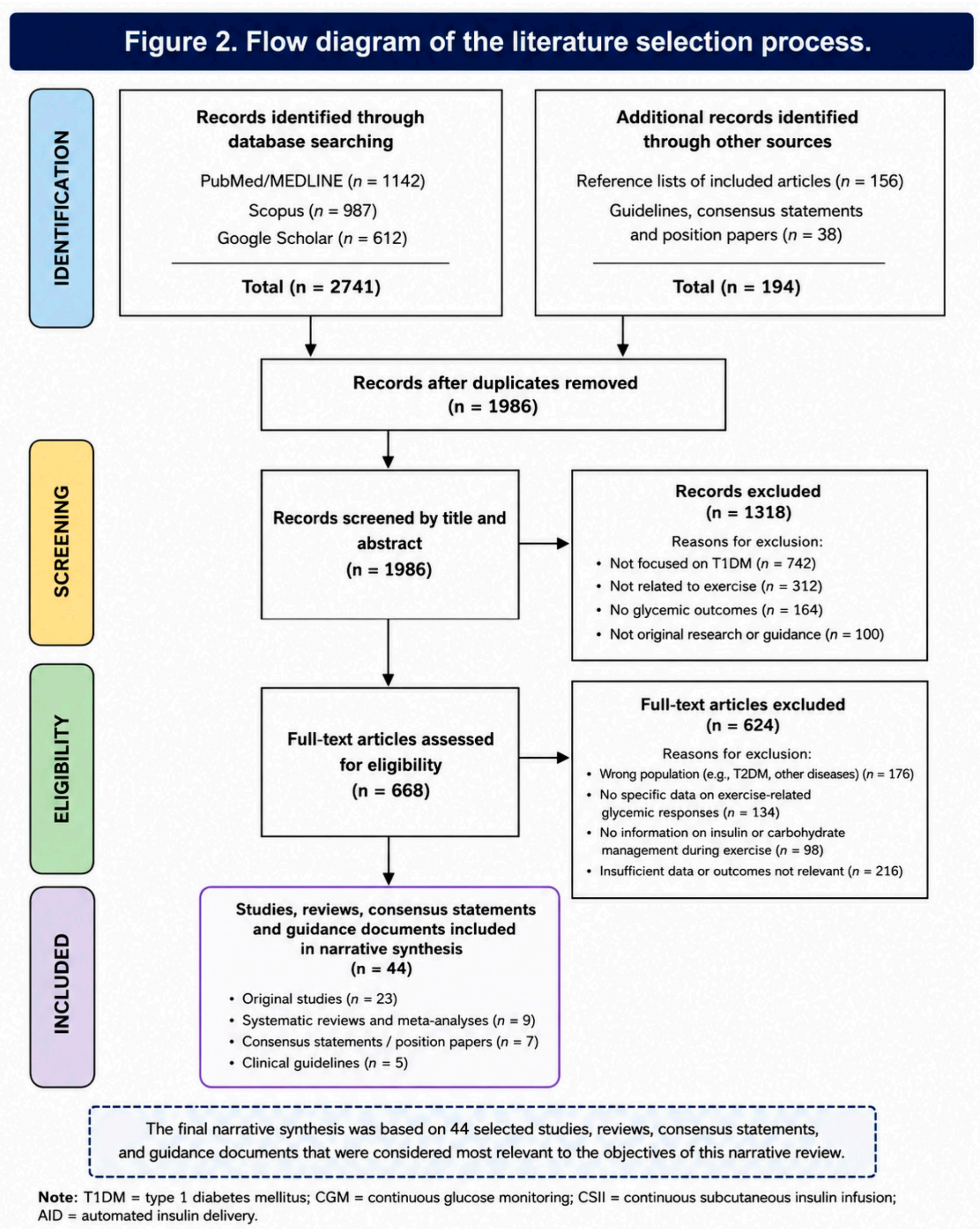

Figure 2. Flow diagram of the literature selection process. The diagram summarizes record identification, screening, eligibility assessment, and final inclusion of studies and documents considered relevant to exercise-related glycemic responses, insulin management, carbohydrate strategies, and diabetes technologies in individuals with type 1 diabetes mellitus.

2.5. Data Extraction and Synthesis Framework

For each included source, the following information was extracted when available: participant characteristics (age, sex, fitness level, diabetes duration), treatment regimen (MDI/CSII/AID), use of CGM and relevant device settings, exercise modality (aerobic, resistance, high-intensity/anaerobic, intermittent/mixed), exercise prescription details (intensity, duration, timing), carbohydrate intake protocols (dose, timing, type), insulin adjustments (basal reduction, bolus modification, temporary basal rates), and glycemic outcomes (magnitude and direction of glucose change, hypoglycemia/hyperglycemia incidence, post-exercise and nocturnal glycemia). Evidence was synthesized narratively. To enhance interpretability and clinical transferability, results were organized by (i) physiological mechanisms underlying exercise-induced glycemic changes, (ii) glycemic responses according to exercise modality and intensity, (iii) insulin and carbohydrate strategies (planned vs. unplanned exercise), and (iv) the role and limitations of diabetes technologies in supporting exercise safety. When findings were heterogeneous, differences were interpreted considering methodological factors (e.g., baseline glycemia, insulin on board, timing of exercise, and measurement approach).

2.6. Considerations on Methodological Quality

Given the narrative nature of the review and the heterogeneity of exercise protocols, outcome measures, and technological approaches across studies, a formal risk-of-bias assessment was not performed. Nevertheless, methodological quality was considered during evidence interpretation, particularly regarding exercise prescription clarity, insulin management standardization, carbohydrate intervention protocols, and reporting of glycemic outcomes.

3. Narrative Synthesis of Current Evidence

The available evidence consistently indicates that exercise-related glycemic responses in individuals with T1DM are strongly influenced by the interaction between exercise modality, insulin exposure, nutritional status, and diabetes technologies. Although predictable glycemic trends can be identified across different exercise modalities, substantial inter- and intra-individual variability remains a major clinical challenge.

3.1. Insulin Therapy and Exercise: Determinants of Glycemic Variability

In individuals with T1DM, exercise-related glycemic variability is largely determined by the amount of active insulin present at the time of physical activity. Unlike individuals without diabetes, people with T1DM cannot rapidly downregulate circulating insulin concentrations during exercise, and glycemic responses are therefore strongly influenced by prior basal delivery and recent prandial or correction boluses [21]. Consequently, the same exercise session may lead to markedly different glucose trajectories depending on whether it is performed in the fasting state, in the postprandial period, or several hours after insulin administration [22,23].

The metabolic impact of insulin exposure also differs according to exercise modality. During prolonged aerobic exercise, elevated insulin on board amplifies muscular glucose uptake and suppresses hepatic glucose production, substantially increasing the risk of hypoglycemia. In contrast, resistance exercise and short high-intensity efforts generally induce lower glucose utilization rates and greater adrenergic activation, often resulting in more stable glycemic responses or transient hyperglycemia. These modality-specific differences are clinically relevant because they require different anticipatory insulin and nutritional strategies.

Insulin pharmacokinetics and mode of delivery further shape exercise-related glycemic variability. With multiple daily injections, basal insulin cannot be rapidly adjusted around activity, and the residual action of long-acting formulations may contribute to late-onset hypoglycemia in the hours following exercise. With continuous subcutaneous insulin infusion (CSII), basal delivery can be modulated more flexibly; however, the timing of adjustments is critical because subcutaneous insulin kinetics introduce delays between a dose change and its metabolic effect. As a result, reductions implemented only at exercise onset may be insufficient to prevent early glycemic declines, particularly during moderate-intensity continuous activity. In addition, insulin absorption may also vary according to injection or infusion site and exercise-related changes in local blood flow [24].

Beyond the intra-exercise period, the post-exercise phase is a major contributor to overall glycemic instability in T1DM. Increased insulin sensitivity, enhanced muscular glucose uptake for glycogen restoration, and sustained metabolic adaptations can persist for several hours, creating a window of vulnerability to delayed hypoglycemia, including nocturnal events following afternoon or evening exercise. Overall, these findings emphasize that exercise-related glycemic responses in T1DM cannot be interpreted solely according to pre-exercise glucose values, but should instead be contextualized according to insulin exposure, exercise modality, and anticipated recovery effects. Moreover, management decisions aimed at correcting post-exercise hyperglycemia—more commonly observed after high-intensity or intermittent exercise—must be balanced against the increased risk of subsequent hypoglycemia due to the delayed and prolonged impact of exercise on insulin sensitivity [25] (Table 1).

Table 1. Key studies addressing insulin exposure and exercise-related glycemic variability in individuals with T1DM.

Authors, Year	Population/Sample	Methodology	Main Findings
Riddell et al., 2017 [7]	Individuals with T1DM	Consensus statement	Elevated insulin on board substantially increases hypoglycemia risk during aerobic exercise
Kastrati et al., 2025 [21]	Individuals with T1DM	Systematic review and meta-analysis	Exercise-related glucose variability is strongly influenced by circulating insulin concentrations
Riddell et al., 2023 [16]	Adults and adolescents with T1DM	Real-world structured exercise study	Glycemic responses differed according to exercise modality and insulin exposure
Zaharieva et al., 2023 [26]	Individuals using AID systems	Practical review	Automated insulin delivery systems improve exercise management but still require user-driven adjustments

3.2. Hormonal and Metabolic Responses: Physiological Versus T1DM Pattern

In healthy individuals, exercise induces rapid endocrine adaptations that maintain glucose homeostasis through coordinated modulation of insulin secretion and counterregulatory hormones. In T1DM, this physiological flexibility is impaired because circulating insulin levels depend on exogenous administration and cannot be rapidly adjusted according to exercise demands.

In addition to impaired insulin modulation, counterregulatory hormone responses are frequently attenuated in T1DM. Several studies have documented a reduced glucagon response to declining glucose levels, as well as blunted adrenergic activation, especially in individuals with a history of recurrent hypoglycemia or impaired hypoglycemia awareness [27,28].

The metabolic consequences of these altered hormonal patterns extend beyond the exercise bout itself. Following physical activity, particularly aerobic exercise, skeletal muscle exhibits enhanced insulin sensitivity and increased glucose uptake to replenish glycogen stores. In individuals with T1DM, this prolonged increase in insulin sensitivity may persist for several hours, amplifying the glucose-lowering effect of previously administered insulin. When combined with residual basal insulin action, this mechanism substantially increases the risk of delayed hypoglycemia, including nocturnal hypoglycemia after late-day exercise. This delayed effect often occurs despite apparently stable glycemia immediately after exercise, underscoring the importance of considering post-exercise metabolic dynamics in addition to intra-exercise responses [29].

Conversely, during high-intensity or anaerobic exercise, the acute stress response may partially compensate for inappropriate insulin levels. Intense muscular contractions stimulate a marked release of catecholamines, which enhances hepatic glucose production and inhibits peripheral glucose uptake, potentially resulting in stable glycemia or transient hyperglycemia. Lactate accumulation during high-intensity efforts further contributes to hepatic gluconeogenesis in the recovery phase, reinforcing post-exercise glucose elevations. However, these hyperglycemic responses are often short-lived and may be followed by delayed glucose declines as catecholamine levels normalize and insulin sensitivity increases [30].

Overall, the hormonal and metabolic response to exercise in T1DM is characterized by a loss of physiological flexibility, with impaired insulin downregulation and variable counterregulatory activation. This altered response underlies much of the glycemic instability observed during and after physical activity and highlights the need for individualized management strategies that account not only for exercise characteristics but also for the underlying endocrine context in which exercise is performed (Table 2).

Table 2. Hormonal and metabolic responses to exercise in individuals with T1DM.

Authors, Year	Population/Sample	Methodology	Main Findings
Colberg et al., 2016 [9]	Individuals with diabetes	Position statement	Exercise induces modality-specific hormonal and metabolic responses
Riddell et al., 2017 [7]	Individuals with T1DM	Consensus statement	Impaired insulin modulation and altered counterregulatory responses contribute to glycemic instability
McClure et al., 2023 [31]	Adults with T1DM	Systematic review and meta-analysis	High-intensity interval exercise may induce transient hyperglycemia and reduce immediate hypoglycemia risk
Eckstein et al., 2023 [32]	Individuals with T1DM	Systematic review and meta-analysis	Continuous aerobic exercise is consistently associated with progressive glucose reductions

3.3. Glycemic Responses According to Exercise Modality and Intensity in T1DM

3.3.1. Continuous Aerobic Exercise

Continuous moderate-intensity aerobic exercise is the exercise modality most consistently associated with progressive glucose declines in individuals with T1DM. Activities such as running, cycling, swimming, or prolonged brisk walking increase skeletal muscle glucose uptake through both insulin-dependent and insulin-independent pathways, leading to a progressive reduction in circulating glucose concentrations during exercise [31,33]. The magnitude of this decline is strongly influenced by the amount of active insulin present at the time of exercise. When aerobic activity is performed within a few hours after a meal bolus or under conditions of elevated basal insulin exposure, the risk of hypoglycemia increases substantially because hepatic glucose production is often unable to adequately compensate for increased peripheral glucose utilization [20,31].

Exercise duration also plays a major role in determining glycemic responses. Longer aerobic sessions are generally associated with greater glucose reductions and a higher likelihood of both intra-exercise and delayed post-exercise hypoglycemia. This phenomenon is further amplified by the prolonged increase in insulin sensitivity that follows aerobic activity, particularly during the recovery period and overnight hours after afternoon or evening exercise [25,33]. Importantly, even when glucose values appear stable immediately after exercise termination, delayed declines may still occur due to glycogen replenishment and sustained muscular glucose uptake.

Although continuous aerobic exercise is typically associated with hypoglycemia risk, considerable inter-individual variability remains. Baseline glycemia, nutritional status, physical conditioning, stress levels, previous exercise exposure, and diabetes technologies all contribute to the heterogeneity of glycemic responses observed in real-world settings [16]. Consequently, safe participation in aerobic exercise generally requires individualized insulin adjustments and/or carbohydrate supplementation tailored to exercise duration, intensity, and glucose trends.

3.3.2. Resistance Exercise

Resistance exercise generally produces more stable glycemic responses compared with prolonged aerobic exercise. Activities such as weightlifting, resistance circuit training, and body-weight strength exercises are characterized by short-duration muscular contractions interspersed with recovery periods, resulting in lower overall glucose utilization during exercise and greater activation of counterregulatory hormonal responses [31,34].

Several studies suggest that resistance exercise may attenuate the rapid glucose declines typically observed during aerobic activity, particularly when performed alone or before aerobic exercise within the same training session [31–34]. The more stable glycemic profile observed during resistance exercise is thought to reflect a combination of lower sustained oxidative glucose demand and increased catecholamine-mediated hepatic glucose output. Nevertheless, resistance exercise does not completely eliminate the risk of glycemic disturbances. Delayed glucose reductions may still occur during recovery because exercise-induced improvements in insulin sensitivity can persist for several hours after training [25].

Exercise sequencing also appears clinically relevant. Evidence indicates that performing resistance exercise before aerobic exercise may reduce the magnitude of subsequent glucose declines compared with initiating the session with continuous aerobic activity [32,34]. This observation has practical implications for exercise programming in individuals with T1DM, particularly in those who experience recurrent hypoglycemia during prolonged aerobic sessions.

Despite generally more stable intra-exercise glycemic responses, resistance exercise management still requires individualized interpretation of insulin on board, glucose trends, and recovery-related glucose dynamics. Excessive insulin correction following transient post-exercise hyperglycemia may predispose individuals to delayed hypoglycemia later in the recovery period.

3.3.3. High-Intensity and Anaerobic Exercise

Short-duration high-intensity or anaerobic exercise elicits glycemic responses that differ substantially from those typically observed during continuous aerobic activity. Sprinting, repeated maximal efforts, high-intensity interval exercise, and explosive anaerobic activities stimulate marked sympathetic nervous system activation, leading to increased catecholamine release and enhanced hepatic glucose production [31].

In many individuals with T1DM, these hormonal responses partially counterbalance muscular glucose uptake during exercise and may induce stable glycemia or transient hyperglycemia during and immediately after activity. Increased hepatic glycogenolysis and gluconeogenesis, together with reduced insulin-mediated glucose disposal, contribute to these acute glycemic elevations [30,31]. Consequently, high-intensity exercise is often perceived as metabolically "safer" than prolonged aerobic exercise in terms of immediate hypoglycemia risk.

However, these hyperglycemic responses are usually temporary and should not be interpreted as complete protection against later glucose declines. As catecholamine concentrations normalize during recovery and insulin sensitivity progressively increases, delayed reductions in glucose levels may still occur several hours after exercise [25]. This delayed effect may become clinically relevant when post-exercise hyperglycemia is aggressively corrected with additional insulin administration, potentially increasing the risk of subsequent hypoglycemia.

Importantly, glycemic responses to high-intensity exercise remain heterogeneous and may vary according to baseline glucose levels, insulin exposure, exercise duration, fitness level, and previous physical activity. Therefore, individualized interpretation of glucose trends and careful post-exercise monitoring remain essential even when exercise initially produces stable glycemia or transient hyperglycemia.

3.3.4. Mixed and Intermittent Exercise Modalities

Mixed and intermittent exercise modalities, including many team and field sports, often produce more heterogeneous and less predictable glycemic responses than purely aerobic or anaerobic exercise. Activities such as soccer, basketball, tennis, volleyball, and high-intensity interval training combine repeated bursts of high-intensity effort with periods of lower-intensity aerobic activity or passive recovery [31,32].

Because aerobic and anaerobic metabolic demands alternate continuously during these activities, glucose trajectories may fluctuate considerably both within and between exercise sessions. In some cases, intermittent activation of counterregulatory hormones during high-intensity efforts partially offsets the glucose-lowering effects of aerobic components, resulting in more stable glycemia compared with continuous aerobic exercise [31]. However, this apparent stability may vary substantially according to exercise structure, duration, sequencing of efforts, nutritional status, and insulin on board.

The unpredictable nature of mixed exercise modalities represents a major challenge for glucose management in real-world settings. Two sessions involving the same sport may produce markedly different glycemic responses depending on contextual factors such as recent meals, stress, environmental conditions, pre-exercise glucose trends, and prior physical activity [16]. Consequently, exercise management during intermittent activities frequently requires dynamic adjustments based on real-time glucose monitoring and individualized experience.

Continuous glucose monitoring systems have become particularly valuable in this context because they provide trend-based information that supports rapid decision-making during rapidly changing physiological conditions. Nevertheless, even with advanced diabetes technologies, mixed and intermittent exercise remains one of the most difficult scenarios for automated insulin delivery systems because rapid fluctuations in glucose kinetics may exceed current algorithm responsiveness [12,17].

Overall, mixed and intermittent exercise modalities remain among the most difficult exercise scenarios for glucose management in individuals with T1DM because glycemic responses may fluctuate rapidly according to exercise structure, insulin exposure, and nutritional context (Table 3).

Table 3. Glycemic responses according to exercise modality in individuals with T1DM.

Exercise Modality	Key References	Typical Glycemic Response	Main Clinical Implication
Continuous aerobic exercise	Riddell et al., 2023 [16]; Eckstein et al., 2023 [32]	Progressive glucose decline	Increased risk of intra- and post-exercise hypoglycemia
Resistance exercise	Riddell et al., 2017 [7]; Riddell et al., 2023 [16]	More stable glycemia	Lower immediate hypoglycemia risk compared with aerobic exercise
High-intensity/anaerobic exercise	McClure et al., 2023 [31]	Transient hyperglycemia or stable glycemia	Careful interpretation of post-exercise correction insulin is required
Mixed/intermittent exercise	Jaggers et al., 2023 [33]; Riddell et al., 2023 [16]	Highly variable glycemic responses	Real-time CGM interpretation is particularly important

3.4. Integrated Management Around Exercise: Insulin and Carbohydrate as Complementary Levers

Across the available literature, effective exercise management in individuals with T1DM consistently emerges as a dynamic and individualized process in which insulin modulation and carbohydrate supplementation should be considered complementary rather than independent strategies. Exercise-related glycemic responses are strongly influenced by the interaction between circulating insulin concentrations, muscular glucose utilization, hepatic glucose production, and nutritional status [35].

Carbohydrate supplementation represents the most immediate and flexible intervention to counteract exercise-induced glucose declines, particularly during prolonged aerobic exercise or when physical activity is unplanned. The amount of carbohydrates required during exercise depends largely on the rate of muscular glucose uptake and on the level of active insulin present at the time of activity. When exercise is performed shortly after a meal bolus or under conditions of elevated insulin exposure, carbohydrate requirements increase substantially because hepatic glucose production is more strongly suppressed and cannot adequately compensate for peripheral glucose disposal [21,26]. Conversely, when exercise is performed several hours after insulin administration or under relatively low-insulin conditions, smaller carbohydrate amounts may be sufficient to maintain glycemic stability.

Although carbohydrate supplementation is highly effective in reducing immediate hypoglycemia risk, excessive reliance on carbohydrates alone may contribute to large glycemic fluctuations, unnecessary caloric intake, and post-exercise hyperglycemia in some individuals. For this reason, anticipatory insulin dose adjustment remains a key component of planned exercise management. Reductions in prandial insulin doses before exercise aim to decrease circulating insulin levels during activity and thereby preserve hepatic glucose production while reducing excessive muscular glucose uptake. In individuals using continuous subcutaneous insulin infusion (CSII), temporary basal rate reductions may provide additional flexibility, although their effectiveness depends strongly on timing because subcutaneous insulin pharmacokinetics delay the metabolic impact of insulin delivery modifications [25,26].

Exercise timing relative to meals also substantially influences management strategies. Physical activity performed within the postprandial period generally carries a greater risk of hypoglycemia because of elevated circulating insulin concentrations, whereas exercise performed several hours after meals or under fasting conditions may require less aggressive carbohydrate supplementation. However, fasting exercise is not universally protective against glycemic instability, as prolonged activity may still induce progressive glucose reductions, particularly during continuous aerobic exercise [33].

The distinction between planned and unplanned exercise is particularly important in real-world diabetes management. When exercise can be anticipated, insulin adjustments implemented before activity may substantially reduce glycemic variability and decrease the need for large carbohydrate supplementation during exercise. Conversely, during spontaneous or unplanned physical activity, rapid carbohydrate intake combined with close glucose monitoring becomes the primary compensatory strategy because insulin exposure cannot be modified in time [26,35].

Importantly, the post-exercise period deserves as much attention as exercise itself. Increased insulin sensitivity and glycogen replenishment may persist for several hours after physical activity, creating a prolonged window of vulnerability to delayed hypoglycemia, particularly overnight following afternoon or evening exercise sessions [25]. Consequently, integrated management should extend beyond the exercise bout itself and include post-exercise glucose monitoring, individualized recovery nutrition, and careful interpretation of correction insulin needs.

Overall, the available evidence supports a clinically oriented approach in which insulin therapy, carbohydrate supplementation, exercise modality, and glucose monitoring are interpreted together rather than separately. Flexible and individualized decision-making, supported by structured education and accumulated personal experience, remains essential to reduce exercise-related glycemic variability and promote safer long-term participation in physical activity in individuals with T1DM (Table 4).

Table 4. Integrated insulin and carbohydrate management strategies around exercise in T1DM.

Authors, Year	Population/Sample	Methodology	Main Findings
Riddell et al., 2017 [7]	Individuals with T1DM	Consensus statement	Insulin modulation and carbohydrate supplementation should be integrated rather than considered separately
Zaharieva et al., 2023 [26]	Individuals using AID systems	Practical review	Carbohydrate intake remains essential despite advanced insulin delivery technologies
Moser et al., 2025 [12]	Individuals using AID systems	Position statement	Basal insulin reduction strategies may improve exercise safety
Cavallo et al., 2024 [36]	Athletes with T1DM	Narrative review	Meal timing and nutritional composition significantly influence exercise-related glycemic responses

3.5. Pre-Exercise Glucose Assessment: Pragmatic Interpretation and Clinical Context

Assessment of glycemic status before initiating physical activity represents a fundamental component of exercise safety in individuals with T1DM. However, pre-exercise glucose evaluation should not be interpreted as a simple verification of whether glucose values fall within predefined thresholds. Instead, effective decision-making requires integration of multiple contextual factors, including glucose trends, insulin on board, recent carbohydrate intake, exercise modality, anticipated exercise duration, and individual history of glycemic responses during physical activity [11,12].

Low pre-exercise glucose values or rapidly declining glucose trends are generally associated with an increased likelihood of hypoglycemia during exercise, particularly during prolonged aerobic activity. This risk becomes even more pronounced when significant circulating insulin remains active from recent prandial boluses or elevated basal insulin delivery. Under these conditions, pre-emptive carbohydrate supplementation is often necessary to restore an adequate metabolic safety margin before initiating exercise [26]. Importantly, the amount of carbohydrates required cannot be standardized across all situations, as carbohydrate needs vary substantially according to exercise intensity, duration, insulin exposure, and individual insulin sensitivity.

Conversely, glucose values within or slightly above the target range do not necessarily guarantee metabolic stability during exercise. Continuous aerobic exercise may still induce progressive glucose declines despite apparently "safe" starting glucose concentrations, especially when exercise is prolonged or initiated in the postprandial period [31,33]. Therefore, exercise-related risk should be interpreted according to the broader metabolic context rather than absolute glucose values alone. Mild to moderate pre-exercise hyperglycemia in the absence of ketones does not automatically contraindicate physical activity and may occasionally provide a temporary protective buffer against exercise-induced glucose reductions, particularly during moderate-intensity aerobic exercise. However, marked hyperglycemia associated with insulin deficiency or ketone production requires greater caution. Under these conditions, exercise—especially high-intensity activity—may further increase catecholamine-mediated hepatic glucose output and exacerbate metabolic dysregulation [17]. Consequently, correction of insulin deficiency and stabilization of metabolic status should generally take priority over immediate exercise initiation.

Continuous glucose monitoring (CGM) systems have substantially improved pre-exercise assessment by allowing interpretation of glucose direction and rate of change rather than reliance on isolated glucose measurements alone [12,17]. Trend arrows and real-time glucose trajectories may help identify whether glycemia is stable, rising, or rapidly declining before exercise, thereby supporting more informed and timely decision-making.

Importantly, pre-exercise glucose assessment should not be viewed as a rigid rule-based process based exclusively on fixed numerical thresholds. Excessive dependence on strict cut-offs risks oversimplifying a highly dynamic physiological condition characterized by substantial inter- and intra-individual variability. Many individuals with T1DM may experience markedly different glycemic responses during apparently similar exercise sessions because of variations in stress, sleep quality, nutritional intake, previous physical activity, hydration status, environmental conditions, and insulin exposure [16]. Therefore, practical exercise management should prioritize individualized contextual interpretation rather than strict adherence to universal glucose thresholds alone.

Structured education combined with real-time glucose monitoring may improve confidence and support more effective individualized exercise-related decision-making in individuals with T1DM (Table 5).

https://doi.org/10.3390/endocrines7020022

Table 5. Key considerations for pre-exercise glucose assessment in individuals with T1DM.

Authors, Year	Population/Sample	Methodology	Main Findings
Adolfsson et al., 2022 [11]	Children and adolescents with T1DM	ISPAD consensus guidelines	Pre-exercise glucose assessment should include glucose trends and insulin on board
Zaharieva et al., 2023 [26]	Individuals using AID systems	Practical review	CGM trend arrows improve exercise-related decision-making
Moser et al., 2025 [12]	Individuals using AID systems	Position statement	Exercise planning should integrate glucose trajectories and anticipated insulin activity
Riddell et al., 2023 [16]	Adults and adolescents with T1DM	Real-world exercise study	Similar exercise sessions may produce highly variable glucose responses

3.6. Insulin, Carbohydrate and Nutritional Strategies Around Exercise

Effective exercise management in individuals with T1DM requires coordinated interpretation of insulin exposure, carbohydrate availability, exercise modality, and glucose trends within the broader clinical context of physical activity [17,35]. Because glycemic responses to exercise are highly variable, insulin and nutritional strategies should be adapted according to exercise intensity and duration, meal timing, baseline glucose levels, and anticipated recovery demands.

Carbohydrate supplementation remains the most immediate strategy to prevent or correct exercise-induced hypoglycemia, particularly during prolonged aerobic exercise or when physical activity occurs in the presence of elevated circulating insulin levels [26,31]. However, carbohydrate requirements vary substantially between individuals and across exercise sessions, limiting the usefulness of rigid standardized recommendations in real-world settings.

The timing of exercise relative to insulin administration is one of the major determinants of glycemic variability during physical activity. Exercise performed during the postprandial period is generally associated with a greater risk of hypoglycemia because circulating insulin concentrations remain elevated [21]. Under these conditions, anticipatory reduction in prandial insulin doses may help attenuate excessive glucose declines and reduce dependence on large carbohydrate supplementation during exercise. In individuals using continuous subcutaneous insulin infusion (CSII), temporary basal rate reductions initiated sufficiently before exercise may further improve glycemic stability, although their effectiveness depends on the delayed kinetics of subcutaneous insulin absorption [12,25].

Exercise modality also substantially influences management strategies. Continuous aerobic exercise is more frequently associated with progressive glucose declines, whereas resistance exercise and short high-intensity efforts may induce more stable glycemia or transient hyperglycemia because of greater adrenergic activation and increased hepatic glucose output [31–34]. Consequently, insulin and nutritional adjustments should be tailored according to the metabolic demands of the planned activity.

Nutritional management extends beyond carbohydrate intake alone. Meal composition and meal timing may substantially influence post-exercise glucose responses, particularly during the recovery period. Meals rich in fat and protein may delay postprandial hyperglycemia and complicate overnight glucose management, especially following late-day exercise sessions [36–38].

Continuous glucose monitoring systems have significantly improved exercise-related decision-making by providing real-time information regarding glucose trends and rate of change [12,17]. Nevertheless, despite advances in automated insulin delivery technologies, exercise remains a challenging physiological condition for current glucose-responsive algorithms because rapid shifts in glucose kinetics and energy expenditure are difficult to fully predict. As a result, individualized interpretation of glucose trends and user-driven adjustments remain essential even in technologically advanced treatment settings.

Particular attention should also be given to the post-exercise recovery phase, during which increased insulin sensitivity may persist for several hours and contribute to delayed hypoglycemia, especially overnight after afternoon or evening exercise [25]. In this context, individualized recovery nutrition, overnight glucose monitoring, and cautious correction insulin strategies may help reduce late glycemic disturbances.

Overall, current evidence supports a flexible and clinically integrated approach combining insulin modulation, carbohydrate management, nutritional planning, and real-time glucose interpretation to support safer participation in physical activity among individuals with T1DM (Table 6).

Table 6. Evidence-based insulin, carbohydrate, and nutritional strategies around exercise in individuals with T1DM.

Authors, Year	Exercise Context	Study Characteristics	Main Management Strategy/Results
Riddell et al., 2017 [7]	Aerobic exercise	Consensus statement	Pre-exercise insulin reduction combined with carbohydrate supplementation reduces hypoglycemia risk
McClure et al., 2023 [31]	High-intensity interval exercise	Systematic review and meta-analysis	High-intensity exercise frequently produces more stable glycemia than continuous aerobic exercise
Zaharieva et al., 2023 [26]	Automated insulin delivery systems	Practical review	Real-time CGM interpretation improves exercise-related decision-making
Cavallo et al., 2024 [36]	Athletes with T1DM	Narrative review	Meal composition and timing influence post-exercise glycemic responses

4. Clinical Implications and Future Perspectives

The evidence synthesized in this review highlights that exercise management in individuals with type 1 diabetes mellitus (T1DM) should be interpreted as a dynamic and individualized process rather than as the application of rigid glucose thresholds or standardized algorithms. Although regular physical activity remains a fundamental component of diabetes care because of its beneficial effects on cardiovascular health, insulin sensitivity, body composition, and overall metabolic well-being, fear of exercise-related hypoglycemia and unpredictable glucose fluctuations continues to represent a major barrier to sustained exercise participation in many individuals with T1DM [7,11,16]. The available literature consistently demonstrates that glycemic responses to exercise are strongly influenced by the interaction between exercise modality, insulin exposure, nutritional status, and individual metabolic variability, making real-world exercise management inherently complex.

One of the most clinically relevant observations emerging from current evidence is that different exercise modalities produce distinct glycemic trajectories. Continuous moderate-intensity aerobic exercise remains the modality most consistently associated with progressive glucose declines and increased risk of hypoglycemia, particularly when exercise is performed in the presence of elevated circulating insulin levels or shortly after prandial insulin administration [31,33,39]. In contrast, resistance exercise and short high-intensity efforts often induce more stable glycemic responses or transient hyperglycemia because of greater adrenergic activation and increased hepatic glucose production [31–34]. Mixed and intermittent exercise modalities, including many team and field sports, frequently produce more heterogeneous responses because aerobic and anaerobic metabolic demands continuously alternate during activity. These modality-specific patterns have important practical implications because they require different anticipatory insulin and nutritional strategies according to exercise type, timing, and individual metabolic context.

Across the available evidence, insulin dose modulation and carbohydrate supplementation consistently emerge as complementary strategies rather than isolated interventions. Carbohydrate intake represents the most immediate tool to counteract exercise-induced glucose declines, particularly during prolonged aerobic exercise or unplanned physical activity when insulin exposure cannot be modified sufficiently in advance [26,35,40]. However, excessive reliance on carbohydrate supplementation alone may contribute to wide glycemic fluctuations and post-exercise hyperglycemia in some individuals. Conversely, when exercise is planned, anticipatory reduction in prandial insulin doses and, where feasible, basal insulin adjustments may reduce active insulin exposure during activity, preserve hepatic glucose production, and decrease dependence on large carbohydrate loads. Nevertheless, the feasibility and effectiveness of insulin adjustments differ according to the insulin regimen. In individuals treated with multiple daily injections, basal insulin cannot be rapidly titrated around exercise because of the prolonged pharmacokinetics of long-acting insulin formulations. In contrast, insulin pump therapy allows for more flexible basal rate modifications, although reductions must generally be implemented sufficiently in advance to exert meaningful metabolic effects because of delays in subcutaneous insulin absorption kinetics [12,25,40].

Another clinically important aspect highlighted by the current literature is the relevance of the post-exercise recovery period. Increased insulin sensitivity and glycogen replenishment may persist for several hours after exercise, thereby extending vulnerability to delayed hypoglycemia, particularly overnight following afternoon or evening physical activity sessions [25,40]. This phenomenon may occur even when glucose levels appear stable immediately after exercise termination, emphasizing the importance of continued glucose monitoring and individualized post-exercise nutritional and insulin-related strategies during recovery.

The growing adoption of continuous glucose monitoring (CGM), insulin pump therapy, and automated insulin delivery (AID) systems has substantially improved exercise-related glucose management in individuals with T1DM. These technologies provide real-time information regarding glucose values and glucose trends while also allowing more flexible insulin delivery adjustments [12,17,41]. CGM trend arrows and alerts facilitate earlier recognition of rapid glucose fluctuations and support more timely carbohydrate interventions during exercise. Similarly, insulin pump therapy may facilitate temporary basal reductions and individualized exercise-specific settings. However, despite these technological advances, physical activity remains one of the most difficult physiological conditions for current AID algorithms because rapid changes in glucose kinetics, energy expenditure, and counterregulatory responses are difficult to fully predict algorithmically. Consequently, user-driven interpretation of glucose trends and individualized decision-making remain essential even in technologically advanced treatment settings.

Taken together, these findings support a clinically meaningful perspective in which exercise management in T1DM should be framed as a contextual decision-making process integrating exercise modality, glucose trends, insulin on board, nutritional status, and anticipated recovery demands rather than reliance on isolated glucose values alone [36,42]. This integrated approach also aligns with the practical distinction between planned and unplanned exercise. When exercise can be anticipated, insulin modulation strategies may reduce excessive insulin exposure during activity and limit glycemic instability. Conversely, during spontaneous or unplanned physical activity, rapid carbohydrate supplementation combined with close glucose monitoring often becomes the primary strategy to reduce hypoglycemia risk. Across both scenarios, structured education and professional guidance

https://doi.org/10.3390/endocrines7020022

remain fundamental to support safer exercise participation in individuals with T1DM. Several limitations within the current evidence base should also be acknowledged. Many studies evaluating exercise-related glycemic responses in T1DM have been conducted under controlled experimental conditions that may not fully reflect the complexity of real-world exercise management, including variability in meal composition, stress, sleep, hydration status, environmental conditions, and adherence to technological settings [43,44]. Considerable heterogeneity also exists across exercise protocols, insulin adjustment strategies, carbohydrate supplementation approaches, and outcome reporting, limiting direct comparison between studies. Furthermore, the rapidly evolving landscape of CGM and AID technologies requires continuous updating of practical recommendations as new algorithms, sensors, and exercise-related device features become available.

Future research should prioritize pragmatic and real-world studies specifically evaluating exercise modality, insulin exposure, nutritional strategies, and diabetes technologies under everyday conditions. Greater attention should also be dedicated to pediatric and older populations, sex-related differences, nocturnal glycemic responses, and individualized predictors of exercise-related glycemic variability. Improved integration between physiological knowledge, technological innovation, and patient-centered education may further enhance exercise safety and support long-term participation in physical activity among individuals with T1DM.

5. Conclusions

Physical exercise remains a fundamental component of care in individuals with type 1 diabetes mellitus (T1DM) because of its well-established benefits on cardiovascular health, insulin sensitivity, metabolic control, and overall well-being [44]. However, exercise-related glycemic variability continues to represent one of the main barriers to regular physical activity participation in this population. The evidence discussed in this review highlights that glycemic responses to exercise are strongly influenced by the interaction between exercise modality, insulin exposure, nutritional status, and individual metabolic variability.

Current evidence supports the need for a flexible and individualized approach to exercise management in T1DM rather than reliance on rigid glucose thresholds or standardized rules alone. Effective prevention of exercise-related glycemic disturbances requires integrated interpretation of glucose trends, insulin on board, exercise characteristics, and nutritional strategies within real-world clinical contexts. In this regard, insulin modulation and carbohydrate supplementation should be considered complementary components of a unified management strategy adapted to the specific metabolic demands of physical activity.

Continuous glucose monitoring, insulin pump therapy, and automated insulin delivery systems have substantially improved exercise-related glucose management and expanded opportunities for safer participation in physical activity. Nevertheless, exercise remains a challenging physiological condition for current technologies, and patient education and individualized exercise planning continue to play a central role. Overall, promoting safe and sustainable exercise participation in individuals with T1DM requires an integrated and education-centered approach combining physiological understanding, individualized insulin and nutritional strategies, and appropriate use of diabetes technologies. Such an approach may help reduce fear of exercise-related glycemic disturbances and support long-term engagement in physical activity as part of comprehensive diabetes management.

Author Contributions: Conceptualization, F.M. (Filomena Mazzeo), G.F. and F.M. (Fiorenzo Moscatelli); methodology, M.R. (Maria Ruberto) and M.R. (Mario Ruggiero); software, N.M.; validation, G.D.M., A.M. (Antonietta Messina) and S.A.; investigation, A.M. (Antonietta Monda) and A.M. (Antonietta Messina); resources, M.G.T. and V.M.; data curation, N.M., R.I.C. and S.A.; writing—original draft preparation, F.M. (Filomena Mazzeo) and G.F.; writing—review and editing, F.M. (Fiorenzo Moscatelli); visualization, M.L.M., P.P., G.D.M., G.R., M.R. (Mario Ruggiero) and M.C.; supervision, G.M., M.G.T. and V.M.; project administration, F.M. (Filomena Mazzeo) and G.F. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: No new data were created or analyzed in this study. Data sharing is not applicable to this article.

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. Durá-Travé, T.; Gallinas-Victoriano, F. Type 1 Diabetes Mellitus and Vitamin D. *Int. J. Mol. Sci.* **2025**, *26*, 4593. [CrossRef] [PubMed]
2. Chen, X.; Zhang, L.; Chen, W. Global, Regional, and National Burdens of Type 1 and Type 2 Diabetes Mellitus in Adolescents from 1990 to 2021, with Forecasts to 2030: A Systematic Analysis of the Global Burden of Disease Study 2021. *BMC Med.* **2025**, *23*, 48. [CrossRef] [PubMed]
3. Grattoni, A.; Korbutt, G.; Tomei, A.A.; García, A.J.; Pepper, A.R.; Stabler, C.; Brehm, M.; Papas, K.; Citro, A.; Shirwan, H.; et al. Harnessing Cellular Therapeutics for Type 1 Diabetes Mellitus: Progress, Challenges, and the Road Ahead. *Nat. Rev. Endocrinol.* **2025**, *21*, 14–30. [CrossRef]
4. Kamitani, F.; Nishioka, Y.; Koizumi, M.; Nakajima, H.; Kurematsu, Y.; Okada, S.; Kubo, S.; Myojin, T.; Noda, T.; Imamura, T.; et al. Immune Checkpoint Inhibitor-related Type 1 Diabetes Incidence, Risk, and Survival Association. *J. Diabetes Investig.* **2025**, *16*, 334–342. [CrossRef]
5. Jacobsen, L.M.; Felton, J.L.; Nathan, B.M.; Speake, C.; Krischer, J.; Herold, K.C. Type 1 Diabetes TrialNet: Leading the Charge in Disease Prediction, Prevention, and Immunotherapeutic Mechanistic Understanding. *Diabetes Care* **2025**, *48*, 1112–1124. [CrossRef]
6. Arce, N.N.; Vu, M.H.; Barnett, B.; Black, D.S. Continuous Glucose Monitoring-Derived Glycemic Profiles and Correlates in Young Adults Ages 18 to 26 Years. *J. Diabetes Sci. Technol.* **2025**, *20*, 627–638. [CrossRef]
7. Riddell, M.C.; Gallen, I.W.; Smart, C.E.; Taplin, C.E.; Adolfsson, P.; Lumb, A.N.; Kowalski, A.; Rabasa-Lhoret, R.; McCrimmon, R.J.; Hume, C.; et al. Exercise Management in Type 1 Diabetes: A Consensus Statement. *Lancet Diabetes Endocrinol.* **2017**, *5*, 377–390. [CrossRef]
8. Corrado, A.; Scidà, G.; Abuqwider, J.; Rainone, C.; Lupoli, R.; Masulli, M.; Bozzetto, L.; Annuzzi, G. Management of Prolonged Aerobic Exercise in People With Type 1 Diabetes on Automated Insulin Delivery Systems: A Randomized Controlled Study. *Diabetes Care* **2024**, *47*, e76–e77. [CrossRef]
9. Colberg, S.R.; Sigal, R.J.; Yardley, J.E.; Riddell, M.C.; Dunstan, D.W.; Dempsey, P.C.; Horton, E.S.; Castorino, K.; Tate, D.F. Physical Activity/Exercise and Diabetes: A Position Statement of the American Diabetes Association. *Diabetes Care* **2016**, *39*, 2065–2079. [CrossRef] [PubMed]
10. Pemberton, J.; Li, Z.; Gal, R.L.; Turner, L.V.; Bergford, S.; Calhoun, P.; Riddell, M.C. Duration of Physical Activity Required to Ameliorate Hyperglycemia without Causing Hypoglycemia in Type 1 Diabetes: A T1DEXI Adults and Pediatric Cohort Analyses. *Diabetes Res. Clin. Pract.* **2025**, *220*, 111981. [CrossRef]
11. Adolfsson, P.; Taplin, C.E.; Zaharieva, D.P.; Pemberton, J.; Davis, E.A.; Riddell, M.C.; McGavock, J.; Moser, O.; Szadkowska, A.; Lopez, P.; et al. ISPAD Clinical Practice Consensus Guidelines 2022: Exercise in Children and Adolescents with Diabetes. *Pediatr. Diabetes* **2022**, *23*, 1341–1372. [CrossRef]
12. Moser, O.; Zaharieva, D.P.; Adolfsson, P.; Battelino, T.; Bracken, R.M.; Buckingham, B.A.; Danne, T.; Davis, E.A.; Dovč, K.; Forlenza, G.P.; et al. The Use of Automated Insulin Delivery around Physical Activity and Exercise in Type 1 Diabetes: A Position Statement of the European Association for the Study of Diabetes (EASD) and the International Society for Pediatric and Adolescent Diabetes (ISPAD). *Diabetologia* **2025**, *68*, 255–280. [CrossRef]

13. Dhaliwal, M.; Tang, K.; Aiello, E.M.; Zaharieva, D.P.; Lal, R.A.; Summers, C.; Arbiter, B.; Watson, K.; Connolly, M.J.; Figg, L.E.; et al. Variation in Hypoglycemia Risk During Real-World Physical Activity in Adults with Type 1 Diabetes: Insights from the Type 1 Diabetes Exercise Initiative. *Diabetes Technol. Ther.* **2025**, *28*, 428–443. [CrossRef] [PubMed]
14. Ogle, G.D.; Wang, F.; Haynes, A.; Gregory, G.A.; King, T.W.; Deng, K.; Dabelea, D.; James, S.; Jenkins, A.J.; Li, X.; et al. Global Type 1 Diabetes Prevalence, Incidence, and Mortality Estimates 2025: Results from the International Diabetes Federation Atlas, 11th Edition, and the T1D Index Version 3.0. *Diabetes Res. Clin. Pract.* **2025**, *225*, 112277. [CrossRef]
15. Codella, R.; Terruzzi, I.; Luzi, L. Why Should People with Type 1 Diabetes Exercise Regularly? *Acta Diabetol.* **2017**, *54*, 615–630. [CrossRef]
16. Riddell, M.C.; Li, Z.; Gal, R.L.; Calhoun, P.; Jacobs, P.G.; Clements, M.A.; Martin, C.K.; Doyle, F.J., III; Patton, S.R.; Castle, J.R.; et al. Examining the Acute Glycemic Effects of Different Types of Structured Exercise Sessions in Type 1 Diabetes in a Real-World Setting: The Type 1 Diabetes and Exercise Initiative (T1DEXI). *Diabetes Care* **2023**, *46*, 704–713. [CrossRef]
17. Fan, W.; Deng, C.; Xu, R.; Liu, Z.; Leslie, R.D.; Zhou, Z.; Li, X. Efficacy and Safety of Automated Insulin Delivery Systems in Patients with Type 1 Diabetes Mellitus: A Systematic Review and Meta-Analysis. *Diabetes Metab. J.* **2025**, *49*, 235–251. [CrossRef]
18. Rizos, E.C.; Markozannes, G.; Charitakis, N.; Filis, P.; Stoimeni, A.E.; Nørgaard, K.; Ntzani, E.E.; Tsilidis, K.K. Continuous Glucose Monitoring in Type 1 Diabetes, Type 2 Diabetes, and Diabetes During Pregnancy: A Systematic Review with Meta-Analysis of Randomized Controlled Trials. *Diabetes Technol. Ther.* **2025**, *27*, 537–552. [CrossRef] [PubMed]
19. Jacobs, P.G.; Levy, C.J.; Brown, S.A.; Riddell, M.C.; Cinar, A.; Boughton, C.K.; Breton, M.D.; Dassau, E.; Forlenza, G.; Henderson, R.J.; et al. Research Gaps, Challenges, and Opportunities in Automated Insulin Delivery Systems. *Diabetes Technol. Ther.* **2025**, *27*, S60–S71. [CrossRef]
20. Qu, X.; Liu, C.; Sun, L.; Sheng, Z. Global Burden of Type 1 Diabetes Mellitus Related Chronic Kidney Disease among Adolescents and Young Adults, and Projections to 2035: Results from the Global Burden of Disease Study 2021. *Diabetes Metab. J.* **2025**, *49*, 812–825. [CrossRef] [PubMed]
21. Kastrati, L.; Alvarez-Martinez, M.; Thomas, A.; Thevis, M.; Muka, T.; Stettler, C.; Herzig, D.; Glisic, M.; Bally, L. Effect of Exercise on Plasma Insulin Levels in Individuals with Type 1 Diabetes: A Systematic Review and Meta-analysis. *Diabetes Obes. Metab.* **2025**, *27*, 876–884. [CrossRef]
22. Feng, M.; Gu, L.; Zeng, Y.; Gao, W.; Cai, C.; Chen, Y.; Guo, X. The Efficacy of Resistance Exercise Training on Metabolic Health, Body Composition, and Muscle Strength in Older Adults with Type 2 Diabetes: A Systematic Review and Meta-Analysis. *Diabetes Res. Clin. Pract.* **2025**, *222*, 112079. [CrossRef]
23. Pan, Y.; Wang, P.; Yue, C.; Liu, C. Effect of Nine Different Exercise Interventions on Insulin Sensitivity in Diabetic Patients: A Systematic Review and Mesh Meta-Analysis. *Front. Endocrinol.* **2025**, *16*, 1409474. [CrossRef]
24. Kazeminasab, F.; Mohebinejad, M.; Mahboobi, M.H.; Nojoumi, M.; Belyani, S.; Bagheri, R.; Dutheil, F. Combined Exercise Training and Dietary Interventions versus Independent Effect of Exercise on Ectopic Fat in Individuals with Overweight and Obesity: A Systematic Review, Meta-analysis, and Meta-Regression. *J. Int. Soc. Sports Nutr.* **2025**, *22*, 2528534. [CrossRef]
25. Rakab, M.S.; Rateb, R.M.; Elsalakawi, B.H.; Al Zoubi, B.M.; Nazir, A.; Abu-Laila, M.M.; Ghanem, A.S.; Maamoun, A.; Mattar, M.; Ataallah, B.; et al. Ultra-Rapid Lispro or Fast-Acting Aspart Compared to Standard Insulin Lispro and Aspart Using Closed-Loop Insulin Therapy: A Systematic Review and Meta-Analysis of Randomized Control Trials. *Front. Endocrinol.* **2025**, *16*, 1600157. [CrossRef] [PubMed]
26. Zaharieva, D.P.; Morrison, D.; Paldus, B.; Lal, R.A.; Buckingham, B.A.; O'Neal, D.N. Practical Aspects and Exercise Safety Benefits of Automated Insulin Delivery Systems in Type 1 Diabetes. *Diabetes Spectr.* **2023**, *36*, 127–136. [CrossRef]
27. Hidayat, K.; Zou, S.; Shi, B. The Influence of Maternal Body Mass Index, Maternal Diabetes Mellitus, and Maternal Smoking during Pregnancy on the Risk of Childhood-onset Type 1 Diabetes Mellitus in the Offspring: Systematic Review and Meta-analysis of Observational Studies. *Obes. Rev.* **2019**, *20*, 1106–1120. [CrossRef]
28. Dean, Y.E.; Motawea, K.R.; Aslam, M.; Pintado, J.J.L.; Popoola-Samuel, H.A.O.; Salam, M.; Dundi, P.O.R.; Donaldy, W.; Aledani, E.M.; Alqiqie, Z.; et al. Association Between Type 1 Diabetes Mellitus and Eating Disorders: A Systematic Review and Meta-Analysis. *Endocrinol. Diabetes Metab.* **2024**, *7*, e473. [CrossRef] [PubMed]
29. Broadley, M.M.; White, M.J.; Andrew, B. A Systematic Review and Meta-Analysis of Executive Function Performance in Type 1 Diabetes Mellitus. *Psychosom. Med.* **2017**, *79*, 684–696. [CrossRef]
30. Pańkowska, E.; Błazik, M.; Dziechciarz, P.; Szypowska, A.; Szajewska, H. Continuous Subcutaneous Insulin Infusion vs. Multiple Daily Injections in Children with Type 1 Diabetes: A Systematic Review and Meta-Analysis of Randomized Control Trials. *Pediatr. Diabetes* **2009**, *10*, 52–58. [CrossRef] [PubMed]
31. McClure, R.D.; Alcántara-Cordero, F.J.; Weseen, E.; Maldaner, M.; Hart, S.; Nitz, C.; Boulé, N.G.; Yardley, J.E. Systematic Review and Meta-Analysis of Blood Glucose Response to High-Intensity Interval Exercise in Adults With Type 1 Diabetes. *Can. J. Diabetes* **2023**, *47*, 171–179. [CrossRef] [PubMed]

32. Eckstein, M.L.; Aziz, F.; Aberer, F.; Böckel, S.; Zimmer, R.T.; Erlmann, M.P.; Sourij, H.; Moser, O. Blood Glucose Response to Running or Cycling in Individuals with Type 1 Diabetes: A Systematic Review and Meta-analysis. *Diabet. Med.* **2023**, *40*, e14981. [CrossRef]
33. Jaggers, J.R.; King, K.M.; McKay, T.; Dyess, R.J.; Thrasher, B.J.; Wintergerst, K.A. Association between Intensity Levels of Physical Activity and Glucose Variability among Children and Adolescents with Type 1 Diabetes. *Int. J. Environ. Res. Public Health* **2023**, *20*, 1623. [CrossRef]
34. Lei, M.; Ling, P.; Ni, Y.; Chen, D.; Wang, C.; Yang, D.; Yang, X.; Xu, W.; Yan, J. The efficacy of glucose-responsive insulin and glucagon delivery on exercise-induced hypoglycaemia among adults with type 1 diabetes mellitus: A meta-analysis of randomized controlled trials. *Diabetes Obes. Metab.* **2024**, *26*, 1524–1528. [CrossRef]
35. Gera, S.; Iyer, J.; Meighan, S.; March, C.A.; Marks, B.E. A Review of Real-World Evidence About the Use of Automated Insulin Delivery Systems in People With Type 1 Diabetes. *Endocr. Pract.* **2025**, *31*, 1150–1161. [CrossRef]
36. Cavallo, M.; De Fano, M.; Barana, L.; Dozzani, I.; Bianchini, E.; Pellegrino, M.; Cisternino, L.; Migliarelli, S.; Giulietti, C.; Pippi, R.; et al. Nutritional Management of Athletes with Type 1 Diabetes: A Narrative Review. *Nutrients* **2024**, *16*, 907. [CrossRef]
37. Boxall, L.R.; Eskandari, F.; Wallis, J.; Bielat, A.D.; Appleton, K.M. The Effects of Aspartame on Glucose, Insulin, and Appetite-Regulating Hormone Responses in Humans: Systematic Review and Meta-Analyses. *Adv. Nutr.* **2025**, *16*, 100449. [CrossRef]
38. Martin-Payo, R.; Fernandez-Alvarez, M.d.M.; García-García, R.; Pérez-Varela, Á.; Surendran, S.; Riaño-Galán, I. Effectiveness of a Hybrid Closed-Loop System for Children and Adolescents with Type 1 Diabetes during Physical Exercise: A Cross-Sectional Study in Real Life. *An. Pediatr. (Engl. Ed.)* **2024**, *101*, 183–189. [CrossRef] [PubMed]
39. Vasco, P.; Allocca, S.; Casella, C.; Colecchia, F.P.; Ruberto, M.; Mancini, N.; Casillo, M.; Messina, A.; Monda, M.; Messina, G.; et al. Nutrition and Physical Activity in the University Population: A Scoping Review of Combined Impacts on Psychological Well-Being, Cognitive Performance, and Quality of Life. *J. Funct. Morphol. Kinesiol.* **2025**, *10*, 374. [CrossRef] [PubMed]
40. Soltani, P.; Almeida, F.M.; Melo, H.C.d.C.; Ferreira, G.B.S.; dos Santos, L.M.; de Brito Gomes, J.L. Body-Weight Functional Exercise Promotes Greater and Safer Blood Glucose Reduction Compared to Aerobic and Strength Exercises in Type 1 Diabetics: A Randomised Crossover Study. *J. Diabetes Complicat.* **2025**, *39*, 109043. [CrossRef]
41. Hohendorff, J.; Grzelka-Wozniak, A.; Wrobel, M.; Kania, M.; Lapinska, L.; Rokicka, D.; Stoltny, D.; Kowalska, I.; Strojek, K.; Zozulinska-Ziolkiewicz, D.; et al. Impact of the Initiation of IsCGM Soon after Type 1 Diabetes Mellitus Diagnosis in Adults on Glycemic Indices and Fear of Hypoglycemia: A Randomized Controlled Trial. *Front. Endocrinol.* **2025**, *15*, 1503891. [CrossRef] [PubMed]
42. Martín-Ávila, J.; Rodríguez-Jiménez, E.; Valero-Moreno, S.; Gil-Gómez, J.A.; Montoya-Castilla, I.; Pérez-Marín, M. The Impact of the EmoTICare Program on Socioemotional Adjustment and Psychological Well-Being in Adolescents with Type 1 Diabetes Mellitus. *Front. Endocrinol.* **2025**, *16*, 1668398. [CrossRef]
43. Dymińska, M.; Kowalczyk-Korcz, E.; Piechowiak, K.; Szypowska, A. The Impact of Two Different Insulin Dose Calculation Methods on Postprandial Glycemia After a Mixed Meal in Children with Type 1 Diabetes: A Randomized Study. *Nutrients* **2025**, *17*, 3287. [CrossRef] [PubMed]
44. Mazzeo, F. Current concept of obesity. *Sport Sci.* **2016**, *9*, 42–48.