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editorial-office@sciformat.ca

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AMATEUR SPORT IN ADULTS WITH TYPE 1 DIABETES: GLYCEMIC CHALLENGES, INSULIN ADJUSTMENT STRATEGIES, AND THE ROLE OF MODERN GLUCOSE MONITORING TECHNOLOGIES

Weronika Cionżyńska (Corresponding Author, Email: weronikacionzynska@gmail.com)
Stefan Żeromski Specialist Hospital in Cracow; os. Na Skarpie 66, 31-913 Cracow, Poland
ORCID ID: 0009-0007-1464-4957

Karina Brzózka
Clinical Emergency Department, University Clinical Centre, ul. Dębinki 7, 80-952 Gdańsk, Poland
ORCID ID: 0009-0004-2338-1827

Zuzanna Pyrka
Gabriel Narutowicz Municipal Specialist Hospital, ul. Prądnicka 35-37, 31-202 Kraków, Poland
ORCID ID: 0000-0001-5688-1036

Adrianna Krupska
Non-Public Medical Clinic "Przychodnia", ul. Majętnego 14, 41-503 Chorzów, Poland
ORCID ID: 0009-0008-1710-1381

Alicja Pieluć
Medical University of Gdańsk, ul. Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk, Poland
ORCID ID: 0009-0003-1150-2241

Katarzyna Ledwożyw
Gabriel Narutowicz Municipal Specialist Hospital, ul. Prądnicka 35-37, 31-202 Kraków, Poland
ORCID ID: 0009-0000-6911-3001

ABSTRACT

Background: Regular physical activity in patients with type 1 diabetes (T1D) is associated with highly dynamic metabolic changes. Prolonged aerobic exercise carries the risk of rapid exercise-induced hypoglycemia, whereas anaerobic exercise and competitive stress can lead to paradoxical hyperglycemia. The growing population of adults with T1D engaging in amateur sports requires modern, evidence-based medical support.

Aim: This paper aims to review the current literature on physiological challenges, periexercise insulin therapy strategies, and the application of modern technologies in adult amateur athletes.

Material and methods: A review of the current literature was conducted using medical databases (PubMed, Scopus), focusing on the latest guidelines and clinical trials. The synthesis targeted adult patients with T1D, glycemic management during amateur sports, and modern diabetes technologies.

Results: Optimal glycemic management requires preemptive basal insulin reduction (planned 60–90 minutes in advance of exercise), modification of mealtime boluses, and targeted carbohydrate supplementation. Delayed late-onset hypoglycemia (LOH), particularly during the night, remains a critical challenge. The use of continuous glucose monitoring (CGM) systems allows for proactive responses to glucose fluctuations based on trend arrows. Furthermore, the implementation of advanced hybrid closed-loop (AHCL) systems, through exercise modes and nocturnal auto-suspension, not only improves metabolic safety but also drastically reduces the cognitive burden and the fear of hypoglycemia (FoH).

Conclusions: Safe and satisfying participation in amateur sports by adult patients with T1D is fully achievable by flexibly combining the latest therapeutic guidelines with advanced diabetes technology and thorough patient education.

KEYWORDS

Type 1 Diabetes, Amateur Sport, Exercise-Induced Hypoglycemia, Continuous Glucose Monitoring, Artificial Pancreas

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1. Introduction

It is well established that regular physical activity is a foundation of a healthy lifestyle for individuals without chronic conditions, as well as an indispensable component of comprehensive management for patients with type 1 diabetes (T1D) (Diabetes Poland, 2025). As unequivocally emphasized in the latest 2024 and 2025 Guidelines on the Management of Patients with Diabetes by Diabetes Poland, systematic physical exercise yields indisputable cardiovascular benefits, including a significant reduction in cardiovascular risk factors, which constitute the primary threat in this patient population. Furthermore, regular sports participation drastically improves tissue sensitivity to exogenous insulin, facilitating an increase in Time in Range (TIR) and the optimization of daily insulin requirements (American Diabetes Association, 2025). Leading global scientific societies, including the American Diabetes Association (ADA) and the International Society for Pediatric and Adolescent Diabetes (ISPAD), recommend incorporating at least 150 minutes of moderate-intensity physical activity per week. These recommendations are grounded in evidence demonstrating that sports are not only a viable method for improving metabolic parameters but also a crucial determinant of overall psychological well-being, preventing diabetes distress (therapeutic burnout) associated with chronic illness, and reducing stress levels (Diabetes Poland, 2025; American Diabetes Association, 2025).

Despite these undeniable benefits, it is crucial to highlight the metabolic challenges that engaging in physical activity poses for patients with T1D (American Diabetes Association, 2025). Recent Polish reviews emphasize that, while sport participation in patients with T1D yields substantial cardiovascular and metabolic benefits, it requires careful planning of insulin doses, carbohydrate intake, and glycemic monitoring (Błaszczak et al., 2024). The primary issue lies in the absence of a uniform physiological response to exercise. The impact of physical exertion is strictly dependent on the nature of the muscular work performed. Prolonged aerobic exercise, such as long-distance running, is associated with rapid and enhanced peripheral tissue glucose uptake, leading to a precipitous drop in blood glucose levels and a consequent risk of acute exercise-induced hypoglycemia (Riddell et al., 2017; Moser et al., 2020). Conversely, high-intensity anaerobic exercise, including sprinting or weightlifting, elicits a vastly different physiological response (Riddell et al., 2017). During such activities, as well as in stressful situations involving the release of catecholamines, particularly epinephrine (e.g., due to competitive stress affecting both professionals and amateurs), hepatic glucose production is heavily stimulated (Riddell et al., 2017; Moser et al., 2020). This mechanism can result in sudden, difficult-to-correct hyperglycemia (Moser et al., 2020). For individuals with T1D, the necessity to constantly balance insulin doses with carbohydrate intake, coupled with the unpredictability of glycemic responses, constitutes a significant burden (American Diabetes Association, 2025). Furthermore, the potential for the dangerous phenomenon of delayed, late-onset nocturnal hypoglycemia (LOH) acts as an additional anxiogenic factor (Colberg et al., 2016). The fear of hypoglycemia (FoH) represents the primary and most formidable psychological barrier preventing adults with T1D from regular sports participation (Brazeau et al., 2008).

Unlike professional athletes, adult amateurs typically do not invest in specialized dietary, physiological, or sports medicine supervision (Brennan et al., 2020). Consequently, patients must manage their glycemia independently in a highly dynamic environment. Amateurs frequently engage in physical activity after a workday - often sedentary office work compounded by stress, which significantly alters baseline insulin sensitivity (Riddell et al., 2017; Brennan et al., 2020). A positive development in this context is the paradigm shift from traditional self-monitoring of blood glucose (SMBG) via fingerstick and multiple daily injections (MDI) toward advanced technologies (American Diabetes Association, 2025). Currently, safe sports participation is facilitated by continuous glucose monitoring (CGM) systems (Moser et al., 2020). A genuine breakthrough has been the introduction of advanced hybrid closed-loop (AHCL) systems, comprising smart

insulin pumps integrated with CGM sensors and predictive algorithms (American Diabetes Association, 2025; Zaharieva et al., 2020). Despite this tremendous progress, these technologies do not entirely relieve the patient of the responsibility and decision-making required during exercise. Appropriate, often preemptive adjustments to basal insulin rates, along with the modification of periexercise mealtime boluses and carbohydrate intake, remain some of the most complex therapeutic and educational tasks for the amateur athlete (Moser et al., 2020; Zaharieva et al., 2020).

The current literature on physical activity in T1D is predominantly composed of publications focusing on pediatric populations or elite professional athletes (Brennan et al., 2020). There is a noticeable deficit of up-to-date, synthetic review articles specifically targeting adult amateur athletes (Brennan et al., 2020; Riddell & Burr, 2011). This demographic is steadily expanding, stimulated by a societal trend toward healthy lifestyles, an increasing number of amateur competitions, and the continuous development of sports infrastructure (Riddell et al., 2017; Colberg et al., 2016; Brennan et al., 2020). As noted by experts, the drive among adult T1D patients to engage in regular, often highly ambitious recreational sports has become a prevalent phenomenon. This necessitates a shift within the clinical community from outdated, conservative paradigms to modern, proactive training support. Furthermore, the extremely dynamic evolution of diabetes technology including the market introduction of next-generation CGM systems and increasingly sophisticated AHCL algorithms renders previous recommendations rapidly obsolete (Boughton & Hovorka, 2021; Sherr et al., 2023). This situation dictates an urgent, expert-recognized need for the systematic updating of clinical knowledge and the formulation of cohesive, accessible therapeutic guidelines (Sherr et al., 2023).

This review article aims to bridge the identified gap by summarizing the latest scientific reports and providing up-to-date, evidence-based medicine (EBM) guidelines. These insights are intended to assist clinicians in effectively supporting adult patients with T1D to safely and satisfyingly participate in amateur sports.

2. Material and Methods

A comprehensive review of the literature was conducted using primary medical and scientific databases, including PubMed, Scopus, and Google Scholar. The search strategy focused on peer-reviewed articles, clinical guidelines, and consensus statements published predominantly between 2015 and 2025, with a particular emphasis on the last five years to capture the rapid advancements in diabetes technology and shifting clinical paradigms. Additionally, seminal physiological studies and foundational papers from earlier years were included to provide essential mechanistic context. The literature search utilized combinations of the following Medical Subject Headings (MeSH) and free-text terms: "type 1 diabetes," "amateur sport," "exercise-induced hypoglycemia," "continuous glucose monitoring," "advanced hybrid closed-loop," and "artificial pancreas." The inclusion criteria were limited to studies involving adult human subjects and publications available in English or Polish. Articles focusing exclusively on pediatric populations or elite professional athletes, without direct translational relevance to adult amateur sports, were excluded from the primary synthesis.

3. Physiological Glycemic Challenges Depending on Exercise Modality

3.1 Aerobic Exercise – Mechanisms of Rapid Glycemic Decline

Prolonged aerobic exercise, such as long-distance running, cycling, or swimming, constitutes one of the most popular types of physical activity undertaken by adult amateur athletes. This type of muscular work is characterized by the increased oxidation of free fatty acids and blood glucose for ATP resynthesis. The primary mechanism responsible for the decrease in glycemia during aerobic exercise is the phenomenon of insulin-independent glucose uptake by working skeletal muscles (Riddell et al., 2017). Muscle fiber contraction activates AMP-activated protein kinase (AMPK), which leads to the translocation of glucose transporter type 4 (GLUT4) from intracellular vesicles to the sarcolemmal membrane. This causes a drastic increase in cell membrane permeability (via GLUT4) for circulating blood glucose (SyLOW et al., 2017).

In healthy individuals with normal endocrine pancreatic function, the onset of physical exercise triggers the suppression of endogenous insulin secretion by the beta cells of the islets of Langerhans and the release of glucagon by the alpha cells. This physiological response stimulates hepatic glycogenolysis and gluconeogenesis, which allows for the balancing of increased glucose consumption and the maintenance of euglycemia (Riddell et al., 2017; Colberg et al., 2016). A significant therapeutic challenge arises in patients with type 1 diabetes. The exogenously administered insulin (in the form of a basal rate from a pump or a long-

acting analog injection) is absorbed continuously from the subcutaneous tissue, regardless of the body's current metabolic demands. This leads to the phenomenon of relative hyperinsulinemia (Riddell et al., 2017). High levels of circulating insulin potentiate (synergistically with muscle contractions) glucose uptake into peripheral tissues and suppress hepatic glucose production. This effect essentially blocks the action of counter-regulatory hormones (glucagon and catecholamines) secreted during exercise (Colberg et al., 2016). As a result, during exercise in an adult patient with T1D, the rate of glucose utilization exceeds the rate of its hepatic release. The consequence is a rapid, linear decline in blood glucose levels, which, without adequately timed interventions (insulin dose reduction, exogenous carbohydrate supplementation prior to exercise), causes severe exercise-induced hypoglycemia (Colberg et al., 2016; Sylow et al., 2017). Environmental factors must also be considered, such as high ambient temperature, which further accelerates insulin absorption from subcutaneous tissue due to the vasodilation of the vascular bed (Gradel et al., 2018; Kenny et al., 2016).

3.2 Anaerobic and Resistance Exercise – The Paradox of Post-Exercise Hyperglycemia

A distinctly different physiological response is elicited by high-intensity exercise dominated by anaerobic metabolism and resistance training (e.g., CrossFit, weightlifting, sprinting, combat sports). During anaerobic activity, the glucose demand of working muscles is primarily met through muscle glycogenolysis. Consequently, lactate concentrations rise rapidly (Yardley et al., 2013). A key element characterizing anaerobic training is the activation of the sympathoadrenal system. High exercise intensity triggers a surge of counter-regulatory hormones, particularly catecholamines (epinephrine and norepinephrine), as well as glucagon and cortisol (Marliss & Vranic, 2002).

Glucagon and cortisol stimulate hepatic glucose production (HGP). High concentrations of catecholamines promote transient peripheral tissue insulin resistance, which limits glucose uptake by the muscles, independent of GLUT4 transporter activity (Yardley et al., 2013; Marliss & Vranic, 2002). In individuals without carbohydrate metabolism disorders, the anaerobic exercise-induced hepatic glucose surge and subsequent hyperglycemia are counterbalanced by a release of endogenous pancreatic insulin, allowing for the maintenance of euglycemia. In individuals with T1D, this mechanism is impaired. Exogenous insulin delivery does not occur automatically in response to the catecholamine surge. It is also significant that patients frequently reduce their basal insulin dose prior to training out of fear of hypoglycemia (Moser et al., 2020). Therefore, a phenomenon of insulin deficiency occurs relative to the increasing demand, which leads to paradoxical hyperglycemia during and immediately after the cessation of anaerobic exercise (Moser et al., 2020; Marliss & Vranic, 2002). It is important to acknowledge the so-called "competitive stress" accompanying athletes during competitions, which, independent of the muscular work performed, induces an epinephrine surge and stimulates hepatic glycogenolysis (Moser et al., 2020). It is also worth noting that hyperglycemia following resistance training is a transient phenomenon. Following the cessation of activity and the subsequent decline in stress hormone levels, tissues return to their baseline insulin sensitivity. Post-training, depleted muscle and liver glycogen stores are replenished, predisposing the athlete to delayed hypoglycemia, often occurring during nocturnal hours (Moser et al., 2020; Yardley et al., 2013).

3.3 Mixed (Interval) Exercise – Metabolic Chaos and Unpredictability

Activities such as football (soccer), basketball, tennis, or squash, which are gaining popularity and are frequent "after-work" activities, present an exceptional therapeutic difficulty. They are classified as mixed exercise (intermittent high-intensity exercise – IHE). They consist of a dynamic combination of an aerobic base (jogging, positioning) with sudden anaerobic bursts (sprinting, jumping, fast breaks). This specific training structure introduces the highest degree of unpredictability and risk of glycemic fluctuations in patients with T1D (Guelfi et al., 2005).

During mixed exercise, two opposing glucoregulatory mechanisms continuously overlap. Prolonged aerobic periods (e.g., easy running) open GLUT4 channels, facilitating greater glucose uptake by the muscles, thereby lowering blood glucose levels. Conversely, every intense, short-lasting anaerobic effort (e.g., a sprint) induces a sudden surge of catecholamines, stimulating the liver to synthesize glucose and temporarily inhibiting its peripheral utilization (Guelfi et al., 2005; Iscoe & Riddell, 2011). As a result of these phenomena, the athlete's blood glucose concentration fluctuates chaotically. Clinical studies indicate an adaptive mechanism to these changes: the incorporation of maximal sprints into continuous aerobic exercise in patients with T1D significantly attenuates the rate of glycemic decline, serving as a natural buffer against sudden exercise-induced hypoglycemia (due to hepatic glucose release) (Guelfi et al., 2005). Unfortunately, precise glycemic management remains a challenging task due to amateur athletes undertaking activities in the

afternoon or evening hours, central nervous system fatigue post-work, competition-related stress, and the continuous activity of a pre-training mealtime bolus (Iscoe & Riddell, 2011). Glucose drops may be masked or delayed by anaerobic activity during mixed exercise; however, this does not protect against the depletion of the body's energy reserves. The utilization of muscle glycogen during mixed activity, combined with increased tissue insulin sensitivity, leads to a high risk of severe, late-onset hypoglycemia (Guelfi et al., 2005; Iscoe & Riddell, 2011).

3.4 Late-Onset Post-Exercise and Nocturnal Hypoglycemia – A Critical Metabolic Challenge

The impact of physical exercise on glycemia in amateur athletes with type 1 diabetes does not cease upon the termination of activity. In addition to the risk of hypoglycemia during exertion, there is a threat of late-onset hypoglycemia (LOH), which can develop from 6 up to 24 hours post-exercise (Scott et al., 2021). The mechanism responsible for this state is the physiological necessity to rebuild depleted glycogen stores in the liver and skeletal muscles.

During the recovery period (the so-called post-exercise carbohydrate window), the process of glycogen resynthesis prioritizes glucose uptake from the bloodstream, placing a prolonged and significant burden on the body's carbohydrate balance (Scott et al., 2021; Kabadi, 2021). This is accompanied by a prolonged increase in the insulin sensitivity of peripheral tissues. The enhanced expression and relocation of GLUT4 transporters to cell membranes persist for many hours, while the sustained vasodilation of the muscle microcirculation facilitates the delivery of insulin and glucose to the myocytes (Kabadi, 2021). With a fully functioning pancreas, the body halts insulin secretion during the recovery phase. In T1D patients, externally administered basal insulin (long-acting analog, pump basal rate) operates independently of the body's post-training needs, resulting in a potentiated hypoglycemic effect under post-exercise conditions (Scott et al., 2021; Campbell et al., 2013).

A particularly dangerous manifestation of LOH is post-exercise nocturnal hypoglycemia, which constitutes a formidable psychological barrier for amateurs undertaking physical activity in the afternoon and evening hours (Campbell et al., 2013). During sleep, there is a physiological lowering of the sympathetic nervous system activation threshold and an attenuation of the counter-regulatory hormone response (epinephrine and glucagon) to falling glucose levels (Ertl & Davis, 2004). During deep sleep, a dangerous accumulation of additional hypoglycemic factors occurs: the peak "window" of glycogen resynthesis, and the active effect of an insulin bolus administered for a post-training meal or basal insulin. This leads to hypoglycemic episodes that pose a potential threat to life and undoubtedly deteriorate the patient's quality of life and sleep architecture (Kabadi, 2021; Ertl & Davis, 2004).

4. Therapy Management Strategies: Modification of Insulin Doses and Carbohydrate Supplementation

The foundation for implementing effective practical preventive strategies is understanding the complex physiological and endocrinological mechanisms occurring during the described forms of physical activity. In clinical practice and amateur sports, glycemic management in adults with type 1 diabetes relies on balancing two factors: the delivery of exogenous insulin and preventive as well as interventional carbohydrate supplementation (Franc et al., 2015). Below are practical, current, evidence-based medicine (EBM) guidelines regarding therapy modifications, categorized by periexercise phases.

4.1 Preparation for Exercise (Pre-training Phase)

A key element in preventing hypoglycemia during exercise (particularly aerobic) is minimizing the phenomenon of relative hyperinsulinemia prior to the onset of activity. The specific strategy depends on the model of insulin therapy utilized.

Modification of basal insulin in continuous subcutaneous insulin infusion (CSII) users: The most effective tool for preventing hypoglycemia is the utilization of the temporary basal rate (TBR) feature. A reduction in the basal rate by 50% to even 80% is recommended for planned moderate- to high-intensity aerobic exercise. The pharmacokinetics of rapid-acting insulin analogs must not be overlooked. For the basal reduction to yield the expected effect, the TBR modification must be programmed 60 to 90 minutes prior to the onset of physical activity (Zaharieva & Riddell, 2017; Franc et al., 2015). The most common mistake made by amateur athletes is completely suspending the pump or reducing the basal rate immediately before training.

This strategy does not protect against exercise-induced hypoglycemia, as insulin absorption has already commenced prior to the workout. Additionally, prolonged pump suspension drastically increases the risk of post-exercise hyperglycemia and ketonemia. In the case of anaerobic and resistance exercise, basal insulin reduction should be minimal or omitted to prevent the paradoxical hyperglycemia discussed earlier (Franc et al., 2015).

Modification of insulin in patients treated with multiple daily injections (MDI): Individuals utilizing insulin pens face a significantly more difficult task. Long-acting insulin analogs are characterized by a stable, multi-hour action profile that cannot be shortened for the duration of the training session (Zaharieva & Riddell, 2017). Guidelines permit a preemptive 10-20% reduction of the basal insulin dose on the day preceding a major, planned physical exertion (e.g., a marathon). However, daily, spontaneous activity does not fall under this strategy. These patients must rely primarily on the reduction of mealtime boluses and increased carbohydrate intake (Campbell et al., 2015).

Modification of the mealtime bolus and the issue of active insulin (IOB - Insulin on Board): If an athlete consumes a meal within a window of 1 to 3 hours prior to planned physical exertion, the mealtime bolus requires modification. The presence of a large amount of active insulin from a mealtime bolus at the onset of training is the strongest predictor of severe exercise-induced hypoglycemia (Franc et al., 2015; Campbell et al., 2015). Depending on the planned duration and intensity of the aerobic exercise, a bolus reduction is recommended: ranging from 25% for brief and light activity to as much as 75% for planned exertion exceeding 60 minutes (Zaharieva & Riddell, 2017; Campbell et al., 2015). Users of modern bolus calculators should be educated to manually modify the system-calculated dose, bearing in mind the synergistic, hypoglycemic effect of insulin and muscle contraction.

4.2 Glycemic Management During Exercise (Supplementation Rules and Safety Thresholds)

A planned reduction in basal insulin and mealtime boluses may prove insufficient to balance the rapid glucose uptake by working muscles. In such situations, a precise, targeted dose of exogenous carbohydrates becomes essential (Riddell & Milliken, 2011). The carbohydrate dosing strategy (supplementary feeding) during training must be individualized and adjusted based on current glucose concentrations, the dynamics of change (trend), and the level of active insulin (IOB).

Carbohydrates as a protective shield against hypoglycemia – dosing: According to sports diabetology guidelines, during prolonged aerobic exercise (lasting over 30-45 minutes), patients with T1D should routinely consume 15 to 30 grams of easily assimilable carbohydrates per hour of activity. During very high-volume exercise (e.g., a marathon, long-distance cycling) and with high levels of circulating insulin, the demand for simple carbohydrates can increase to up to 60 grams per hour (Riddell & Milliken, 2011; West et al., 2011). The selection of appropriate carbohydrates is crucial. Simple carbohydrates with a high glycemic index (GI), such as glucose in the form of energy gels, tablets, or isotonic drinks, are optimal in this scenario. Liquid and semi-liquid forms guarantee rapid gastrointestinal absorption, minimizing gastric burden during abdominal muscle contractions (West et al., 2011). During brief and intense anaerobic exercise (e.g., gym workouts), preemptive simple carbohydrate supplementation is mostly unnecessary and may even exacerbate the phenomenon of paradoxical post-exercise hyperglycemia (Moser et al., 2020; West et al., 2011).

Glycemic threshold values - absolute contraindications to exercise: A difficult yet vital skill for an amateur athlete is the ability to forgo training during states of acute homeostatic disruption. Experts have established safety frameworks based on glucose concentrations immediately prior to commencing activity (Colberg et al., 2015; Cakała et al., 2024).

Below 70 mg/dl (3.9 mmol/l) or 70-90 mg/dl (3.9-5.0 mmol/l) with a downward trend: The patient must absolutely refrain from initiating physical exertion. It is required to consume 15 grams of simple carbohydrates and wait 15 minutes (the so-called 15/15 rule) until the glucose concentration stabilizes at a safe level (>90 mg/dl).

Above 270 mg/dl (15.0 mmol/l): Such a high glucose level necessitates identifying its etiology. A measurement of ketone body concentrations, preferably from blood, is required. If the ketone body level exceeds 1.5 mmol/l (or urine ketone results are moderate/high), undertaking physical activity is absolutely contraindicated (Colberg et al., 2015). Consequently, physical exertion would lead to a surge of counter-regulatory hormones and exacerbate insulin deficiency, accelerating the development of life-threatening diabetic ketoacidosis (DKA) rather than lowering glycemia.

4.3 The Post-Exercise Window and Prevention of Nocturnal Hypoglycemia (Post-Training Phase)

Following the cessation of physical activity, the body enters a phase of heightened insulin sensitivity and the resynthesis of depleted muscle and liver glycogen reserves. To avoid late-onset hypoglycemia (LOH), particularly during the night, the implementation of specific therapeutic strategies is required.

Strategy for the post-workout (recovery) meal: Administering the standard, calculator-derived bolus dose for a post-workout meal carries a significant risk of hypoglycemia. This is due to the fact that consumed carbohydrates are taken up by the muscles to replenish reserves independently of insulin. According to guidelines, in the case of a meal consumed within a 1 to 2-hour window following moderate- or high-intensity training, the mealtime bolus dose should be reduced by approximately 50% (Campbell et al., 2015; Campbell et al., 2014). Such a strategy allows for the safe replenishment of energy reserves without the risk of post-exercise hyperinsulinemia.

Protection against nocturnal hypoglycemia: basal reduction and a prophylactic snack: Preventing LOH following afternoon or evening workouts requires both the modification of insulin doses and targeted dietary intervention before sleep.

Continuous Subcutaneous Insulin Infusion (CSII) users: The gold standard of management is programming a temporary basal rate (TBR) reduced by 20% for the duration of the night (typically for 6 to 8 hours during sleep) (Franc et al., 2015; Campbell et al., 2014).

Preventive nocturnal snack (bedtime snack): Regardless of the insulin delivery method utilized (CSII or MDI), checking glycemia immediately before sleep is imperative. In the event of a glucose concentration below 120-130 mg/dl after a day involving physical exertion, the patient should consume a small, prophylactic snack without administering an insulin bolus. This snack should consist of complex carbohydrates combined with protein and fat (e.g. a peanut butter sandwich, plain yogurt with nuts). Fats and proteins delay gastric emptying, ensuring a slow and stable release of glucose into the bloodstream. This provides a safeguard against sudden glycemic drops in the second half of the night, when the counter-regulatory hormone response in T1D patients is at its lowest (Campbell et al., 2014; Dubé et al., 2012).

5. The Role of Modern Technologies (CGM and AHCL) in the Monitoring and Therapy of Amateur Athletes

5.1 From Point-in-Time Glycemic Measurements to Prediction – The Paradigm Shift Brought by Continuous Glucose Monitoring (CGM) Systems

Historically, periexercise glycemic management relied on self-monitoring of blood glucose (SMBG) using glucometers. This method provides information solely regarding the current, point-in-time blood glucose concentration. In the dynamic environment of physical exertion, glycemic fluctuations in T1D patients can reach rates of 2 to even 4 mg/dl per minute, which creates a risk of making erroneous therapeutic decisions (Moser et al., 2020; Pettus & Edelman, 2017). The widespread clinical adoption of real-time continuous glucose monitoring (rtCGM) and intermittently scanned/flash glucose monitoring (isCGM/FGM) systems constitutes a revolution, transforming the patient's static metabolic picture into a continuous one.

Interestingly, the clinical advantage of CGM technology is not merely the frequency of measurements (typically every 5 minutes), but the provision of trend arrows. These indicate the direction and rate of change (ROC) of glucose concentrations (Pettus & Edelman, 2017). This enables amateur athletes to transition from reactive treatment (i.e. mitigating the effects of already active states) to proactive therapy. According to the latest consensus of the European Association for the Study of Diabetes (EASD), the interpretation of trend arrows fundamentally alters the approach to carbohydrate supplementation during exercise (Moser et al., 2020). For instance, a glucometer reading of 120 mg/dl does not indicate a necessity to consume simple carbohydrates. However, the exact same reading on a CGM sensor, accompanied by two downward trend arrows (indicating a drop of >2-3 mg/dl/min), signifies an imminent risk of severe hypoglycemia within 15 minutes. In such scenarios, guidelines recommend the immediate consumption of 15 to 20 grams of simple carbohydrates without interrupting the activity (Moser et al., 2020; Pettus & Edelman, 2017). Importantly, upward trend arrows prevent the phenomenon of "over-treatment" - unnecessary supplementary feeding that could result in subsequent rebound hyperglycemia (Moser et al., 2019). Furthermore, rtCGM systems offer the capability to set personalized, predictive auditory alarms that warn of an impending glycemic drop before the glucose level falls below the lower target threshold. This has profound psychological significance, reducing the fear of exercise and hypoglycemia (Moser et al., 2019).

5.2 Advanced Hybrid Closed-Loop (AHCL) Systems in the Dynamics of Exercise

Advanced Hybrid Closed-Loop (AHCL) systems alleviate the burden of continuous dose decision-making from the patient to the greatest extent. Predictive algorithms, such as the Minimed 780G, Tandem Control-IQ, or the CamAPS FX algorithm, are based on continuous communication between an rtCGM sensor and an insulin pump. The algorithm analyzes the current glucose concentration and trend arrows, autonomously increasing, decreasing, or completely suspending basal insulin delivery every few minutes (Moser et al., 2025). Crucial for athletes is the so-called Exercise Mode (or Activity Feature), which allows for a temporary alteration of the target glycemic threshold. The standard target glycemia to be maintained is approximately 100-110 mg/dl (5.5-6.1 mmol/l). Exercise mode typically raises this therapeutic target to a level of 140-150 mg/dl (7.8-8.3 mmol/l) (Moser et al., 2025; Breton & Kovatchev, 2021). This compels the algorithm to drastically reduce basal insulin delivery, establishing a safety buffer against a precipitous drop in glucose concentration during physical exertion. This facilitates the completion of moderate aerobic exercise with minimal risk of hypoglycemia and reduces the necessity for preemptive carbohydrate feeding (Breton & Kovatchev, 2021).

5.3 Technological Limitations and the Principle of "Outpacing the Algorithm"

Despite technological advancements, patients must be educated regarding their barriers. Notwithstanding the sophisticated algorithm, insulin absorption from subcutaneous tissue remains a slow and delayed process (Moser et al., 2025).

A fundamental error committed by amateur athletes utilizing closed-loop systems is activating the exercise mode immediately prior to commencing training. According to the latest consensus of the European Association for the Study of Diabetes (EASD) regarding physical activity in Automated Insulin Delivery (AID) systems, the exercise mode should be enabled 1 to 2 hours prior to planned physical activity. This ensures that the algorithm-generated basal reduction has sufficient time to physically lower the concentration of circulating active insulin (IOB) by the start of the workout (Moser et al., 2025). In the event of consuming a pre-training meal that causes an increase in glycemia, AHCL systems can trigger an automatic micro-correction bolus exactly at the onset of training, constituting a specific therapeutic trap. The combination of this bolus and intensified physical exertion can lead to rapid and inevitable hypoglycemia (Moser et al., 2025; Breton & Kovatchev, 2021). Therefore, strict control of mealtime boluses and manual intervention are recommended, despite the use of automated systems.

AHCL systems demonstrate undeniable efficacy in the context of eliminating the risk of delayed, late-onset nocturnal hypoglycemia (LOH). The algorithm detects a downward glycemic trend during sleep and suspends the basal insulin flow (the so-called auto-suspension feature), which protects the patient from a nocturnal incident and eliminates the need for them to wake up (Moser et al., 2025; Tauschmann et al., 2016). This alleviates patients' fear of undertaking physical activity in the evening hours, which could otherwise result in nocturnal hypoglycemia (Tauschmann et al., 2016).

A frequently marginalized aspect in endurance studies is the psychological dimension, which is inextricable, particularly for amateur athletes. Traditional treatment models require continuous carbohydrate calculation and precise dose modification during activity, generating an additional cognitive burden. Coupled with the fear of hypoglycemia (FoH) which has been identified by researchers for years as the primary barrier deterring patients with type 1 diabetes from regular sports participation (Brazeau et al., 2008), this leads to what is known as diabetes distress. The application of AHCL and rtCGM systems diametrically alters the patients' situation. Delegating the necessity of background insulin adjustments to the algorithm (including basal suspension in the face of hypoglycemia risk) significantly reduces FoH indices (Beato-Vibora et al., 2021). From a psychological standpoint, the athlete with T1D is no longer forced to concentrate their attention entirely on the prevention of acute complications; instead, they can genuinely focus on technique, tactics, and above all, the satisfaction derived from sports participation itself, thereby enhancing the patients' overall quality of life (QoL).

6. Conclusions

Physical activity in adult patients with type 1 diabetes is a vital component of therapy, although it can pose a significant medical challenge. Today, driven by advancements in technology and comprehensive patient education, it has become an integral and indispensable element of disease management and quality of life enhancement. As demonstrated in this review, amateur sports participation among adults with type 1 diabetes is associated with highly dynamic metabolic shifts. This necessitates that the patient not only comprehends the underlying physiological changes - ranging from precipitous glycemic drops during aerobic exercise to paradoxical hyperglycemia induced by stress and anaerobic exertion but also masters the proactive planning of physical activity.

Safe training hinges on the skillful balancing of insulin delivery and carbohydrate intake. The key is a timely reduction in basal insulin (initiated 60-90 minutes prior to exercise) coupled with aggressive reductions in mealtime boluses, which minimizes the risk of severe exercise-induced hypoglycemia. Equally critical is the recovery phase, during which appropriate therapeutic modifications protect against the phenomenon of delayed, late-onset nocturnal hypoglycemia.

The dynamic development of technology represents a true paradigm shift in sports diabetology. Continuous glucose monitoring (CGM) systems equipped with trend arrows enable preemptive responses to glycemic fluctuations, while advanced hybrid closed-loop (AHCL) systems featuring exercise modes alleviate a substantial portion of the decision-making burden from patients. It must be emphasized, however, that modern technology does not absolve the patient from the necessity of thorough diabetes education. Predictive algorithms still require an informed user capable of independently anticipating the consequences of the slow pharmacokinetics associated with subcutaneously administered insulin.

Today, the contemporary clinician should no longer discourage patients with T1D from participating in ambitious competitions or engaging in evening post-work training sessions. The role of the sports medicine physician and diabetologist is to provide the patient with evidence-based therapeutic tools and education, empowering them to pursue their athletic passions in a manner that is both entirely safe and highly satisfying.

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