

Recovery phase nutrition and insulin strategies for a female collegiate distance runner with type
1 diabetes mellitus: a case study

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B.A., The Master's University, 2004

A REPORT

submitted in partial fulfillment of the requirements for the degree

MASTER OF SCIENCE

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KANSAS STATE UNIVERSITY
Manhattan, Kansas

2022

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Abstract

Background

There is scant published research regarding nutrition and insulin strategies for athletic performance in collegiate distance runners with Type 1 Diabetes Mellitus (CDRT1). Acute carbohydrate supplementation (CHOs_{sup}) and insulin reduction used to minimize hypoglycemia during exercise may result in deteriorated glycemic control post-exercise in CDRT1. The present case study of a CDRT1 investigated the effectiveness of a moderate-carbohydrate (ModCHO) diet and 24 hr insulin adjustment during the recovery phase for improved glycemic control and reduced use of acute strategies.

Methods

During an 8-day period, a female CDRT1 followed a ModCHO (~4 g/kg/day) nutrition program. Recovery phase adjustments to insulin doses were made using an equation developed to estimate reduced insulin needs post-exercise, as a function of exercise intensity and duration. Daily training was performed in the fasted-state at 6:00 a.m. and included additional exercise strategies to reduce glycemic variability when needed. Daily blood glucose time in range (TIR) and use of CHOs_{sup} were assessed. Additionally, athlete well-being was determined using the Student-Athlete Well-Being Score survey at baseline, and days 1, 3, and 7.

Results

Throughout the 8-day case study, the athlete's mean TIR increased (77% versus < 50%) and the magnitude of glycemic excursions decreased (~3.8–15 versus ~3.0–26 mmol/L) relative to a prior comparison period. Minimal pre-exercise CHOs_{sup} was employed and CHOs_{sup} during exercise was not required. The athlete achieved a new lifetime best in the 5,000 m run and maintained positive well-being during the 8-day period.

Conclusion

The present case study provides examples of recovery phase strategies (i.e., ModCHO diet and 24-hour insulin adjustments) that may support glycemic control and athletic performance in CDRT1 and provides potential starting points for nutrition and insulin strategies for use by athletes and coaches.

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Introduction

Type 1 Diabetes Mellitus (T1DM) is an autoimmune disease characterized by an uncontrolled inflammatory response whereby pancreatic β cells are damaged or destroyed by the immune system, causing insulin levels to be severely limited or absent, with resultant hyperglycemia. Disease progression necessitates exogenous insulin administration via multiple daily injections (MDI) or continuous subcutaneous insulin infusion (CSII, i.e., pump therapy) (Ozougwu et al., 2013; Zaccardi et al., 2015). According to a recent systematic review and meta-analysis, the worldwide prevalence of T1DM is 9.5 per 10,000 people ($\sim 1\%$), while prevalence in the United States is slightly higher (12.2 per 10,000 people) (Mobasseri et al., 2020). Although many people with T1DM compete in sporting events, with some reaching elite or professional levels, the prevalence of T1DM among competitive athletes is currently unknown.

There is a large body of published research regarding metabolic and hormonal responses to exercise in people with T1DM. However, there is a lack of research providing evidence-based sport-specific strategies to support athletic performance in athletes with T1DM across the spectrum of sporting events. Though there are many types of athletes absent within published research, the present case study highlights a collegiate distance runner with T1DM (CDRT1) as a unique case from the larger endurance athlete population. For the current report, we define “collegiate distance runners” as highly trained college athletes competing in 5,000 and 10,000 m running events. We distinguish CDRT1 as a distinct sub-group within the broad category of “endurance athlete” because most studies investigating performance strategies for endurance athletes with T1DM involve recreational athletes, marathon runners, or professional cyclists

training and competing at intensities and durations that may be different from what 5,000 and 10,000 m collegiate runners would typically encounter (Table 10).

Moreover, published research pertaining to athletic performance in T1DM has been predominantly focused on acute (i.e., pre- and during-exercise) strategies such as carbohydrate supplementation (CHOs_{sup}) and/or insulin reduction for mitigating hypoglycemia during exercise, with little attention given to the recovery phase (i.e., period between workout bouts) (Scott et al., 2021). Although the acute strategies mentioned can be effective for preventing exercise-induced hypoglycemia (Grimm et al., 2004; Rabasa-Lhoret et al., 2001), applying commonly used strategies to CDRT1 may lead to deteriorated glycemic control following exercise (Yardley et al., 2013), potentially compromising recovery and glycogen repletion (Hwang et al., 1995; O'Neill et al., 2018), glycemic stability during subsequent training bouts (Galassetti et al., 2003), and body composition goals (Brown et al., 2011). For the current case study, we used an approach that emphasized the recovery phase, hypothesizing that this strategy may be more effective for improving glycemia and athletic performance in a female CDRT1 than acute strategies alone. Therefore, the aim of the present case study was to evaluate the effectiveness of recovery phase strategies (i.e., moderate-carbohydrate diet and 24 hr insulin dose adjustment) and early morning exercise for improving overall glycemia, with reduced reliance on acute strategies, to support athletic performance in a female CDRT1. The current case study also comprises a highlight of current knowledge gaps regarding CDRT1 and an overview of the published research addressing nutrition and insulin strategies for athletic performance in T1DM that are most closely related to the current case.

Review of the Literature

In order to identify available published research addressing nutrition and insulin strategies for athletic performance in T1DM, records were identified through a primary search of PubMed for articles. Search terms included the following: “type 1 diabetes mellitus” (MeSH) AND “athlete” (MeSH); Additional searches were performed including “type 1 diabetes mellitus” (MeSH) AND one or more of the additional terms: “runner”, “sports”, “athlete”, “endurance athlete”, “nutrition”, “management”, “strategies”, “basal rate”, “insulin”.

Strategies for Hypoglycemia During Exercise

At rest, fatty acids are the predominant fuel source in skeletal muscle; however, during exercise, reliance on carbohydrate (CHO) for fuel increases with exercise intensity (Romijn et al., 1993). During exercise, CHO needs in skeletal muscle are primarily supplied by skeletal muscle glycogen (Coyle et al., 1986), with small amounts of blood glucose (BG) uptake augmenting energy demands as exercise continues. To maintain euglycemia (i.e., BG level between 3.9–10 mmol/L) during exercise, naturally decreased insulin and increased glucagon secretion (i.e., decreased insulin-glucagon ratio) promote increased hepatic glucose production (HGP) to match BG uptake by skeletal muscle (Wahren & Ekberg, 2007). However, in T1DM, exogenously administered insulin cannot be naturally decreased following injection, rendering the athlete incapable of dynamic internal coordination of metabolic and hormonal responses during exercise. Moreover, portal absorption of insulin present in subcutaneous tissue may persist at an elevated rate during exercise due to increased blood perfusion at the injection site (Pitt et al., 2020). When the circulating insulin-glucagon ratio does not decrease, “hyperinsulinemia” suppresses HGP while exercise accelerates skeletal muscle BG uptake; when

HGP is insufficient to keep pace with BG uptake by skeletal muscle, hypoglycemia ensues (Felig & Wahren, 1975).

Hypoglycemia is defined as BG < 3.9 mmol/L; “severe hypoglycemia” refers to hypoglycemic events requiring assistance from another person to treat. While it has been estimated that > 10% of adults with T1DM experience one severe hypoglycemic event per year (Weinstock et al., 2013), the true prevalence of hypoglycemia during and around exercise is unknown and no association linking severe hypoglycemia to physical activity has been established (Bohn et al., 2015; Cockcroft et al., 2020). Regardless of prevalence, the implications of hypoglycemia in athletic performance are evident, including fear of participation (Kennedy et al., 2018) and reduced training and competition performance (Kelly et al., 2010). While exercise induced hypoglycemia and hyperglycemia can both negatively affect exercise capacity over the long term, hypoglycemia may be a more immediate detriment during athletic performance (Kelly et al., 2010; Stettler et al., 2006). Accordingly, among strategies for optimizing athletic performance in T1DM, those aimed at mitigating hypoglycemia during exercise are the most prevalent in published research. Common approaches include acute (i.e., pre- and during-exercise) CHO supplementation (CHOs_{up}) and/or acute insulin (i.e., bolus and/or basal) dose reduction (Cockcroft et al., 2020). These approaches are outlined in the subsequent paragraphs.

CHO Supplementation

The amount of CHOs_{up} needed to avoid hypoglycemia during exercise depends on many factors, including insulin “on board”, timing and macronutrient composition of a recent meal, glycogen stores, exercise duration and intensity, antecedent hypoglycemia, and exercise timing (Galassetti et al., 2013; Lindmeyer et al., 2020; McKewen et al., 1999; Rabasa-Lhoret et al., 2001; West et al., 2011). Amid such complexity of factors influencing CHOs_{up}, Table 1

provides a synthesis of results from a study by Grimm and colleagues (2004), indicating the amount of CHO intake necessary to prevent hypoglycemia during exercise of varying intensities and durations. The study involved 67 patients with T1DM separated into four treatment groups (i.e., with or without acute CHOsup and with or without acute insulin reduction), investigating nine subgroups of exercise intensities and durations (i.e., < 20, 20–60, and > 60 min, each conducted at < 45%, 45–60%, and > 60% VO₂max). Notably, pre-exercise insulin dose reductions are recommended in addition to CHOsup during higher exercise intensities and longer exercise durations.

Table 1. Carbohydrate Required to Prevent Hypoglycemia at Various Intensities and Durations of Exercise

Exercise Intensity	< 20 min	20–60 min	> 60 min
~45% VO ₂ max		15 g	30 g/hr
~45–60% VO ₂ max	15 g	30 g	75 g/hr, - 20% insulin dose
> ~60% VO ₂ max	30 g	75 g, -20% insulin dose	100 g/hr, - 30% insulin dose

Adapted from Grimm et al., 2004; VO₂max estimates according to Liguori et al., 2022. **Bold** indicates intensities and durations of training and competition typically encountered by CDRT1.

Because of the complex factors affecting CHOsup needs, athletes with T1DM typically rely on pre-exercise BG as a guide. Many athletes with T1DM find continuous glucose monitoring (CGM) technology advantageous in this context, as most CGM systems display arrows indicating the directional trend of current BG status, providing valuable information for decision making (Zaharieva & Riddell, 2015). While athletes may use their own empirically

developed strategies to determine CHOsup needs, Table 2 provides recommendations based on pre-exercise BG. These values were synthesized based on recommendations provided from a collection of case studies, reviews, and the American Diabetes Association (ADA) position statement regarding physical activity and exercise in patients with T1DM (Colberg et al., 2016; Zaharieva & Riddell, 2015).

Table 2. Carbohydrate Intake Recommendations According to Pre-Exercise Blood Glucose

Pre-Exercise Blood Glucose	Carbohydrate Intake and Other Recommendations
< 5.0 mmol/L	Ingest 15–30 g fast-acting CHO pre-exercise (may be unnecessary for brief and/or very high-intensity exercise); prolonged activity may require additional CHO intake during exercise (0.5–1.0 g/kg/hr)
5.0–8.3 mmol/L	Consume 0.5–1.0 g/kg/hr during exercise (depending on exercise intensity and duration and insulin on board)
8.3–13.9 mmol/L	Delay CHO intake during exercise until BG < 8.3 mmol/L
13.9–19.4 mmol/L	Test for ketones; initiate mild-moderate exercise only if ketones are negative; delay intense exercise until BG < 13.9 mmol/L
≥ 19.4 mmol/L	Test for ketones; consider 50% insulin correction dose; initiate mild-moderate exercise only if ketones are negative; delay intense exercise until BG < 13.9 mmol/L

Adapted from Colberg et al. (2016) and Zaharieva & Riddell (2015).
Abbreviations: BG = blood glucose, CHO = carbohydrate.

While the values indicated by tables 1 and 2 provide guidance for CHOsup to perform training, such large quantities of CHO intake may result in post-exercise hyperglycemia,

(McKewen et al., 1999; Murillo et al., 2015; Yardley et al., 2013). Additionally, CHOsup may promote weight gain (Zaharieva & Riddell, 2015), especially when additional insulin is required to mitigate post-exercise hyperglycemia (Brown et al., 2011), making such an approach problematic in athletes desiring to maintain a low body mass. Moreover, high CHO intake before and during exercise may be impractical for some athletes due to the possibility of gastrointestinal distress. Therefore, pre-exercise reduction of bolus insulin, basal insulin, or both, have been suggested as complimentary or alternative solutions for preventing hypoglycemia during exercise.

Bolus Insulin Reduction

Bolus insulin refers to rapid-acting insulin dosed to “cover” prandial (i.e., mealtime) CHO or to correct hyperglycemia (Cornell et al., 2021). Bolus insulin administered to correct hyperglycemia is typically referred to as a “correction bolus”, which is dosed using a correction factor (i.e., the amount BG is lowered by 1 unit of insulin). The most common method for calculating prandial bolus insulin dose is using an “insulin-carbohydrate” ratio (ICR), though other novel methods have been investigated (Bozzetto et al., 2015). The patient’s ICR represents how many grams of CHO are covered by 1 unit of insulin and is typically titrated by a Certified Diabetes Care and Education Specialist (Cornell et al., 2021). Among insulin adjustment strategies, reduction of pre-exercise mealtime bolus insulin is the most common in published research, presumably due to feasibility in MDI and pump therapy users alike.

Table 3 shows the amount by which pre-exercise bolus insulin should be reduced to mitigate hypoglycemia during various exercise intensities and durations; notably, 60 min of exercise performed at > 50% VO₂max is not reflected here, and to our knowledge, has not been addressed elsewhere in published research (Rabasa-Lhoret et al., 2001; Riddell et al., 2017). As a

point of reference, the athlete in the present case study generally trains 50–100 min per day at intensities > 50% VO₂max (Table 10).

Table 3. Pre-Exercise Bolus Insulin Reduction to Mitigate Hypoglycemia at Various Exercise Intensities and Durations

Exercise Intensity	Pre-Exercise Bolus Insulin Reduction	
	30 min exercise	60 min exercise
25% VO ₂ max	-25%	-50%
50% VO ₂ max	-50%	-75%
75% VO ₂ max	-75%	unknown

Adapted from Rabasa-Lhoret et al., 2001. Bolus insulin administered with meal containing 75 g carbohydrate.

West and colleagues (2011) examined the impact of timing of exercise relative to mealtime on the effect of prandial bolus insulin reduction by comparing glycemic responses when exercise was initiated 30, 60, 90, and 120 min after a pre-exercise meal containing 75 g CHO with concurrent 75% bolus insulin reduction. Each exercise bout consisted of 45 min of running at 71% peak VO₂. The best preservation of BG was observed when the bolus insulin dose was administered 30 min before initiation of exercise, which may be explained by the pharmacodynamics of rapid-acting insulin (Table 4). When administered 30 min beforehand, exercise may be near completion before the peak activity of bolus insulin is reached. Importantly, it is unknown whether a 75% bolus reduction 30 min pre-exercise would preserve BG during running at high intensities or long durations. Moreover, consuming a meal within 30 min of exercise may be undesirable to some athletes due to the possibility of gastrointestinal distress.

Table 4. Pharmacodynamics of Various Types of Insulin

Insulin product	Action	Basal/Bolus Use	Time to Onset	Peak Effect	Duration
Humalog® (lispro)	Rapid ^a	Bolus in MDI	5–15 min	45–75 min	3–5 hr
NovoLog® (aspart)	Rapid	Basal and Bolus in insulin pump	5–15 min	45–75 min	3–5 hr
Apidra® (glulisine)	Rapid	Basal and Bolus in insulin pump	5–15 min	45–75 min	3–5 hr
Lantus® (glargine)	Long ^b	Basal in MDI	1.5–2 hr	Flat ^c	18–24 hr
Levemir® (detemir)	Long	Basal in MDI	1.5–2 hr	Flat	18–24 hr

Adapted from Yurkewicz et al., 2016.

^a Rapid = quick onset and short-duration activity.

^b Long = relatively slow onset and long-duration activity.

^c Flat peak effect = consistent action with little/no peak.

While most distance runners prefer their meals > 2 hr before exercise, reducing insulin this far in advance is likely to cause hyperglycemia (Zaharieva et al., 2019). Therefore, published research generally indicates initiation of exercise < 2 hr after mealtime bolus insulin reduction (Riddell et al., 2017). However, because the duration of rapid-acting insulin activity can extend up to 5 hr post injection (Table 4), bolus insulin delivered < 2 hr prior to exercise will still be active during exercise. Unfortunately, bolus insulin activity is highly variable across athletes and exercise intensities and durations, potentially making glycemic responses to exercise difficult to predict. Furthermore, bolus insulin on board during exercise durations > 30 min typically requires acute CHOsup to avoid hypoglycemia during exercise (Grimm et al., 2004). Therefore, athletes wishing to avoid CHOsup may consider adopting a fasted-state morning exercise regimen, as this practice has been shown to reduce risk of hypoglycemia during and after

exercise, potentially reducing or eliminating the need for additional CHO intake (Gomez et al., 2015). While the mechanism favoring morning versus afternoon exercise in reducing risk of exercise-induced hypoglycemia is not clearly understood, the absence of circulating mealtime bolus insulin may contribute to observed glycemic improvement accompanying morning exercise (Gomez et al., 2015; Scott et al., 2018). Alternatively, some have speculated that improved glycemic stability during morning exercise may be related to the circadian rhythm of cortisol, the primary counterregulatory hormone to insulin, typically present in higher concentrations in the morning than in the afternoon (Gomez et al., 2015). However, more research is needed to confirm this effect.

Basal Insulin Reduction

Basal insulin, sometimes called “background” insulin, refers to insulin administered to regulate fluctuations in BG from glucose produced endogenously during the fasted-state. While bolus insulin is always rapid-acting, basal insulin can be either rapid-acting or long-acting depending on the mode of delivery. Multiple daily injection (MDI) insulin therapy requires long-acting insulin that is manually administered once or twice daily, whereas pump therapy (i.e., CSII) automatically delivers small amounts of rapid-acting insulin continuously throughout the day. A “basal rate” refers to the amount of basal insulin delivered per hour using an insulin pump (Cornell et al., 2021).

Perhaps due to low flexibility, suggestions for pre-exercise basal insulin reduction are less prevalent in published research than the other strategies already discussed. While CHOsup and bolus insulin reduction may be initiated as late as 30 min prior to exercise, basal insulin reduction requires greater forethought and may be impractical for those using MDI. However, pre-exercise basal rate reduction may be a feasible strategy for attenuating hypoglycemia during

exercise in athletes using an insulin pump, albeit advanced planning is needed as several studies have indicated questionable efficacy of basal rate reductions implemented < 1 hr before exercise. McAuley and colleagues (2016) showed that 50% basal rate reduction 1 hr pre-exercise did not preserve BG during 30 min of moderate-intensity exercise. Likewise, 80% basal rate reduction 40 min pre-exercise appears insufficient to minimize exercise-induced hypoglycemia (Roy-Fleming et al., 2019). Because the peak effect of rapid-acting insulin occurs 45–75 min post injection (Table 4), basal rate reduction > 75 min before exercise may be required to improve BG preservation during exercise. To this point, Zaharieva and colleagues (2019) demonstrated that 50–80% basal rate reduction 90 min before exercise can be effective for avoiding hypoglycemia around exercise.

Alternatively, some researchers have suggested basal rate suspension during exercise as a means of mitigating hypoglycemia (Tsalikian et al., 2006). However, though basal rate suspension during exercise is safe and even preferred by many athletes, suspension alone is ineffective for preventing hypoglycemia, and may result in post-exercise hyperglycemia when suspended longer than 75 min (Tsalikian et al., 2006; Zaharieva et al., 2019).

Strategies for Post-Exercise Hypoglycemia

Hypoglycemia that occurs long after the conclusion of exercise (i.e., “late”; 6–12 hr after exercise) and nocturnal hypoglycemia are also concerns in T1DM, as BG can fall markedly > 6 hr after exercise (Campbell et al., 2015) due to increased insulin sensitivity that may last up to or longer than 24 hr post-exercise (Bird & Hawley, 2017). During the exercise recovery phase, diminished insulin requirements may result in hypoglycemia if appropriate adjustments to insulin doses or CHO intake are not made (Campbell et al., 2015). While avoiding hypoglycemia during exercise is important, mitigating post-exercise hypoglycemia should not be overlooked. Though

prevalence of post-exercise hypoglycemia in T1DM is unclear (Cockcroft et al., 2020), studies suggest that late and nocturnal hypoglycemia are not uncommon, especially among children and those performing exercise in the afternoon (Campbell et al., 2014; Tsalikian et al., 2005, 2006). Moreover, it is important to note that antecedent hypoglycemia can blunt counterregulatory defenses against hypoglycemia during subsequent exercise; likewise, antecedent exercise can blunt counterregulatory responses to subsequent hypoglycemia (Briscoe et al., 2007; Schneider et al., 1991).

Despite the reality of post-exercise hypoglycemia and the potential for increased risk with antecedent hypoglycemic episodes diminishing counterregulatory responses, there is little published research indicating specific strategies for mitigating hypoglycemia during the exercise recovery phase, and a “trial and error” approach is generally recommended. Research-based suggestions include reducing bolus insulin by 20–50% with the first meal post-exercise, and a similar reduction of basal rate during the first 6–12 hr post-exercise (Campbell et al., 2015; Riddell et al., 2020; Scott et al., 2021; Taplin et al., 2010). The broad range of insulin reductions suggested by current research may reflect the heterogeneity of post-exercise insulin sensitivity among athletes and begs the question of whether greater specificity can be established in order to avoid a purely “trial and error” approach. According to a recent survey, lack of clarity regarding variability in glycemic responses to exercise is a chief concern of patients with T1DM, caregivers, and healthcare providers (Klaprat et al., 2020). Along these lines, Moser and colleagues (2020) recently endeavored to assess glycemic responses to various exercise intensities during an observational study that followed seven professional cyclists with T1DM competing in a 5-day road-cycling race. The study demonstrated a significant negative association between the previous day’s exercise intensity and bolus insulin requirement,

highlighting the need and opportunity for future research investigating systematic strategies for calculating recovery-phase insulin requirement according to prior exercise intensity.

Interestingly, while exercise increases insulin sensitivity, exercise-induced inflammation resultant from muscle damage can cause transient insulin resistance in skeletal muscle tissue; however, more research is needed to determine the extent to which this mechanism may influence post-exercise insulin requirements in athletes with T1DM (Belli et al., 2017).

While most athletes are aware of increased insulin sensitivity following exercise, the lack of practical methods for estimating appropriate bolus and basal insulin adjustment, may lead athletes to consume additional CHO as a means of mitigating post-exercise hypoglycemia; a strategy that may conflict with body composition goals for CDRT1.

Strategies for Post-Exercise Hyperglycemia

Alternatively, high-to-maximal intensity exercise may result in post-exercise hyperglycemia in T1DM. During activity performed at $> 85\%$ VO_{2max} , catecholamine levels are elevated > 12 -fold beyond typical resting levels, resulting in a > 7 -fold increase in HGP (Sigal et al., 1996). While insulin levels rise rapidly post-exercise in people without diabetes to regulate transient increases in glycemia caused by excess HGP, in T1DM, lack of automatic endogenous insulin secretion can quickly lead to hyperglycemia if untreated (Marliss & Vranic, 2002; Purdon et al., 1993). Catecholamine response during high-to-maximal intensity exercise appears to be independent of concurrent insulin-glucagon ratio (Sigal et al., 1996). Thus, post-exercise hyperglycemia may still be experienced in the presence of relative hyperinsulinemia in T1DM.

Potentially compounding post-exercise hyperglycemia, the common strategies for mitigating hypoglycemia during exercise, such as acute (i.e., pre- and during-exercise) CHOsup

and/or insulin dose reduction, may also promote post-exercise hyperglycemia (Riddell et al., 2020). Though less frequently addressed in published research, post-exercise hyperglycemia may be a more common occurrence in athletes with T1DM, perhaps because the attention paid to hypoglycemia avoidance may lead athletes to err on the side of hyperglycemia (Colberg, 2020). The effects of post-exercise hyperglycemia include delayed recovery and inflammation, reduced glycogen repletion, decreased heart rate variability, reduced anaerobic threshold, and reduced 24 hr glycemic control (Campbell et al., 2014; Komatsu et al., 2010; Lespagnol et al., 2020; McKewen et al., 1999; O'Neill et al., 2018).

Delivery of an insulin correction dose is the most prevalent strategy indicated in published research for reducing post-exercise hyperglycemia in T1DM. Correction doses are calculated by dividing the amount of BG elevation above target, by the individual's correction factor (i.e., the amount BG is lowered by 1 unit of insulin). Administration of a 50% insulin correction dose has been demonstrated to reduce post-exercise hyperglycemia without causing subsequent hypoglycemia (Turner et al., 2015). However, while 50% insulin correction dose may be a good starting point, variability of post-exercise insulin sensitivity may challenge accurate dosing.

CHO Availability

The importance of adequate CHO availability in competitive athletes for optimal exercise economy during intense endurance events is well-understood (Burke et al., 2011; Thomas et al., 2016). However, optimizing CHO availability may create an additional challenge and add complexity for CDRT1. Unfortunately, CHO guidelines have not been established for athletes with T1DM and current recommendations (Tables 5 and 6) for athletes without diabetes may promote deteriorated glycemic control in T1DM (McKewen et al., 1999). Importantly,

glycemic excursions have been demonstrated to suppress hepatic and skeletal muscle glycogen synthesis, while long-term near normal glycemia has been demonstrated to promote normal glycogen synthesis in T1DM. (Bischof et al., 2001, 2002; Hwang et al., 1995). Therefore, insisting athletes with T1DM adhere to CHO intake and acute fueling recommendations for athletes without diabetes at the expense of glycemic control, may unfortunately result in reduced glycogen synthesis. Additionally, poor glycemic control has been shown to reduce cardiopulmonary response (i.e., cardiac output, stroke volume, arterial blood pressure, vascular resistance, and airway pressure) to exercise in endurance athletes (Baldi et al., 2010). Clearly CHO availability in T1DM should be considered in the context of glycemic management. More research is needed to establish appropriate CHO intake guidelines for athletes with T1DM, especially those competing in events where performance benefits are derived from high CHO availability.

Table 5. Daily Carbohydrate Intake Recommendations for Athletes

Exercise Intensity	Description	Carbohydrate Intake
Light	Low-intensity or skill-based activities	3–5 g/kg/day
Moderate	Moderate exercise program (e.g., ~1 hr/day)	5–7 g/kg/day
High	Endurance program (e.g., 1–3 hr/day moderate to high-intensity exercise)	6–10 g/kg/day
Very High	Extreme commitment (e.g., 4–5 hr/day moderate to high-intensity exercise)	8–12 g/kg/day

Adapted from Thomas et al., 2016.

Table 6. Acute Carbohydrate Fueling Recommendations

Strategy	Timing	Carbohydrate Intake
Carbohydrate loading	Preparation for events > 90 min of sustained/intermittent exercise	36–48 hr of 10–12 g/kg/24 hr
Speedy refueling	< 8 hr recovery between two fuel-demanding sessions	1–1.2 g/kg/hr for the first 4 hr then resume daily fuel needs
Pre-event fueling	Before exercise > 60 min	1–4 g/kg consumed 1–4 hr before exercise
During brief exercise	< 45 min	Not needed
During sustained high intensity exercise	45–75 min	Small amounts, including mouth rinse
During endurance exercise including "stop and start" sports	1–2.5 hr	30–60 g/hr

Adapted from Thomas et al., 2016.

Briefly, low-carbohydrate diets (LCD; < 130 g/day) have been employed since the pre-insulin era as a means of managing glycemia in T1DM, though after the discovery of insulin in 1922, increasingly greater flexibility of diet composition has been observed (Scott et al., 2019a). However, lack of glycemic control among 79% of adults with T1DM and recent rise in prevalence of obesity in T1DM, have led to reevaluation of the usefulness of LCDs for glycemic control and weight management (Corbin et al., 2018; Foster et al., 2019). Additionally, the relatively new concept of “train low, compete high” in terms of CHO availability, has prompted consideration of the feasibility of LCDs for glycemic management and performance in athletes with T1DM (Scott et al., 2019a). While potentially beneficial physiological adaptations of CHO restricted training have been noted in endurance athletes without T1DM (Bartlett et al., 2014; Impey et al., 2018), overall improvement of athletic performance in elite endurance athletes using the “train low, compete high” method is questionable, as adaptations may blunt CHO

oxidizing capacity during competition (Bartlett et al., 2014; Burke et al., 2017). Moreover, to our knowledge, such practices have not been tested in athletes with T1DM (Scott et al., 2019a). Further research is needed to investigate the efficacy of LCDs for athletic performance in collegiate distance runners (i.e., 5,000–10,000 m) with T1DM.

Summary of Knowledge Gaps

Published research aimed at maximizing athletic performance in T1DM predominantly consists of acute nutrition and insulin strategies to allay hypoglycemia during exercise, with some attention given to mitigation of post-exercise late and nocturnal hypoglycemia, and to a lesser extent post-exercise hyperglycemia. However, few extant strategies encompass exercise intensities and durations frequently encountered by CDRT1, necessitating a synthesis of strategies extrapolated from a combination of studies involving runners without diabetes and non-runners with diabetes. Additionally, published research addressing recovery phase strategies for optimizing performance in T1DM is scarce, requiring athletes to take a “trial and error” approach to insulin reduction, and to follow glycogen repletion strategies designed for athletes without diabetes. Finally, it is important to acknowledge the lack representation of female athletes with T1DM in the available published research. The aim of the present case study is to address several of the aforementioned knowledge gaps while demonstrating the need and opportunity for future research.

Athlete Background

A female collegiate distance runner (i.e., 5,000 and 10,000 m), age 22 years, with a 3-year history of competitive running, diagnosed with T1DM at age 15 years, noted extreme glycemic excursions and chronic pain during her freshman and sophomore years of college running. While CHOsup before and during exercise allowed the athlete to avoid severe hypoglycemia during training and competition, the time between workouts was often marked by a cycle of hypo- and hyper-glycemic excursions.

The athlete and coach questioned whether acute strategies for mitigating hypoglycemia during exercise may be the cause of poor glycemic control throughout the day. Our hypothesis was that prioritizing glycemic control and glycogen repletion during the recovery phase would reduce reliance on acute strategies for hypoglycemia during exercise, and thereby improve daily glycemic control, and ultimately athletic performance. During the athlete's junior year, athlete and coach partnered to develop recovery phase strategies for daily nutrition, insulin adjustments, and exercise timing, aimed at reversing the cycle of glycemic excursions toward better glucose control and predictability. The following case study is the culmination of their efforts.

Methods

During an 8-day period, while maintaining her prescheduled training regimen (Table 10), the athlete followed a predetermined daily nutrition program and insulin adjustment strategy developed in partnership with her coach. The athlete's training regimen and detailed description of her nutrition program and insulin adjustment strategy, including rationale, are provided below.

Nutrition Program

Although a variety of eating patterns can support general health and glycemic management in T1DM (American Diabetes Association, 2018), the athlete followed a moderate-carbohydrate (ModCHO) diet (Table 7), consisting of CHO intake of ~ 4 g/kg/day (~ 225 g/day) throughout the eight days. While demonstrated to support performance in endurance athletes without diabetes (Burke et al., 2011), our experience and available published research indicate that adherence to a high CHO diet (Table 5 and 7) may promote deterioration of glycemic control in T1DM (McKewen et al., 1999; Murillo et al., 2015; Yardley et al., 2013). As an alternative, some have considered very low to low CHO diets as a possible strategy for athletic performance and glycemic management in T1DM (Scott et al., 2019a); however, CHO intakes < 50 g/day may impair exercise economy at higher intensities (Burke et al., 2017). Notably, data from a recent survey indicate that $> 60\%$ of active individuals with T1DM choose very low to moderate CHO diets (Colberg, 2020), shedding light on current practices. With these observations in mind, a ModCHO diet was chosen as a “happy medium” intended to provide the greatest amount of CHO intake feasible without compromising glycemic control. Additionally, consideration was given to choosing a diet that was sustainable long-term and would not interfere with social interaction for the athlete.

Table 7. Carbohydrate Diet Definitions

Diet	Definition
Very low carbohydrate diet	20–50 g/day or < 10% caloric intake or < 1 g/kg/day
Low carbohydrate diet	< 130 g/day or < 26% total energy intake or < 3 g/kg/day
Moderate carbohydrate diet	26–45% of total energy intake or 3–6 g/kg/day
High carbohydrate diet	> 45% of total energy intake or 7–8 g/kg/day
ADA guidelines	45–60% total energy intake from carbohydrate

Adapted from Scott et al. (2019).

Daily meals were prepared in advance, and macronutrient content was estimated using cronometer.com (Revelstoke, British Columbia, Canada). Three meals were consumed per day (Table 8) at the time of day preferred by the athlete. While a variety of meals (chosen by the athlete) were consumed during the case study, total daily CHO intake was evenly distributed between meals (~70 g/meal) to minimize variability in prandial glycemic response and to simplify glycemic management (American Diabetes Association, 2018). Per instruction provided by her diabetes clinic and in keeping with her usual practice, the athlete subtracted total fiber content from CHO each meal to calculate her bolus insulin dose. However, because this practice is not supported by current research (Cornell et al., 2021), the fiber content of each meal was consistent for all meals throughout the study to eliminate any potential variation caused by subtracting total fiber from CHO. Protein and fat intake followed current guidelines for athletes (Thomas et al., 2016) and were spread throughout the day to promote greater predictability of glycemic response (Evert et al., 2013). Moreover, consistent macronutrient distribution allowed for protein and fat insulin requirements to be built-in to the athlete's insulin-carbohydrate ratio (ICR). Additionally, dual-wave boluses (i.e., a combination of an immediate normal bolus and a bolus delivered over a prolonged specified amount of time, referred to as a “square wave”) were employed to account for delayed gastric emptying from fat intake and amino acid

gluconeogenesis (Paterson et al., 2015). An additional consideration was the potential for augmented glycogen synthesis during sub-optimal CHO intake with co-ingestion of small amounts protein (Cermak & van Loon, 2013). Daily CHO intake was generally low-glycemic load (GL) with high fiber content given the evidence supporting general health benefits and glycemic management in T1DM (Evert et al., 2013), and possible potentiation of insulin activity (Colberg, 2020).

Table 8. Moderate-Carbohydrate Nutrition Program

	Carbohydrate ^a	Protein ^b	Fat ^c
Macronutrient Servings	1/3 cup oats, dry	1 ounce poultry or beef	2 TBSP nuts/seeds
	1/3 cup brown rice, cooked	1 ounce fish	1 TBSP nut/seed butter
	1/2 cup legumes, cooked	1 large egg	1 TBSP butter
	1/2 cup quinoa, cooked	1 cup milk, 1%	1 TBSP olive oil
	1 cup milk, 1%	1 cup chocolate milk, 1%	1/2 avocado, medium
	1/2 cup chocolate milk, 1%	1/3 cup cottage cheese, 1%	
	1/2 potato, medium	1/3 cup Greek yogurt, 1%	
	1/2 sweet potato, medium	1/4 cup feta crumbles	
	1/4 cup granola, low-fat	1/4 cup shredded cheese	
	1 cup berries	1 ounce sliced cheese	
	1/2 piece of fruit		
	2 TBSP dried fruit		
	1 medjool date		
	1 TBSP honey or maple syrup		
	Non-starchy Vegetables		
	2 cups leafy greens		
	1 cup raw vegetables		

Daily intake included 3 meals/day according to number of macronutrient servings specified below with 1 serving of non-starchy vegetables/meal; 1 low-carbohydrate (i.e., 10 g) high-protein (i.e., 20 g) bedtime snack/day.

^a 5 servings/meal (~70 g).

^b 4 servings/meal (~35 g).

^c ~1 serving/meal (~10 g).

In addition to daily meals, a low-CHO high-protein bedtime snack was consumed once per evening. Although evidence supporting the efficacy of nutritional strategies in preventing nocturnal hypoglycemia is lacking in published research (Campbell et al., 2014; Desjardins et al., 2013), this strategy was effective for reducing nocturnal hypoglycemia in the athlete observed in the present case study. When needed, CHOsup was implemented in response to present or imminent hypoglycemia, or when pre-exercise blood glucose (BG) was < 8 mmol/L. Medjool dates (~10 g CHO per date) were ingested when pre-exercise CHOsup was needed (i.e., pre-exercise BG < 8). Athlete and coach found dates favorable because of personal preference for taste, portability, easy dosing of CHO, and high glucose-fructose content (Al-Farsi & Lee, 2008). Though published research is limited regarding the practice of pre-exercise glucose-fructose intake by individuals with T1DM, several studies indicate this may be an effective strategy for hypoglycemia prevention (Bally et al., 2017; Kosinski et al., 2020). Results from a clinical trial involving 15 men with T1DM indicated that pre-exercise glucose-fructose co-ingestion has a BG preserving effect with lower incidence of post-exercise hyperglycemia than pre-exercise glucose intake alone during moderate-intensity exercise (i.e., 90 min of cycling at 50% VO₂max) (Bally et al., 2017). Bally and colleagues (2017) suggested that this result was due to increased fat oxidation and the glycogen sparing effect of fructose.

Insulin Dose Adjustment

Throughout the study, in alignment with usual practices, the athlete used her own Medtronic MiniMed 770G System (Medtronic Diabetes, 2022) for blood glucose monitoring (i.e., CGM) and insulin administration (i.e., CSII, pump therapy). It is important to note that although the 770G System has “auto mode” capability, allowing for automatic real-time adjustment of basal rate based on the patient’s sensor glucose readings (i.e., “closed-loop

system”), the athlete does not normally use auto mode, nor was it enabled during the days of the current study.

As a means to predict reduced recovery phase insulin needs, with greater specificity as compared to a broad range (i.e., 20–50%) currently provided by published research (Scott et al., 2021), an Insulin Equation (Table 9) was developed by the athlete and coach with consideration given to research indicating that insulin requirements during the recovery phase may correspond to daily exercise intensity (Moser et al., 2020; Scott et al., 2020). Development of the equation began with observation of 24 hr trends in the athlete’s glycemic response to various exercise intensities and durations, followed by the creation of an equation designed to calculate a recovery phase insulin dose that matched insulin needs as indicated by observed trends in the athlete’s glycemic response to different exercise intensities and durations. During a 6-month period, the equation was tested and modified until calculated daily insulin adjustments produced > 70% BG time in range (TIR; i.e., the amount of time someone with diabetes spends with BG between 3.9–10 mmol/L during a 24 hr period) (Agiostatidou et al., 2017) during the recovery phase. The resultant Insulin Equation determines the percent reduction of insulin total daily dose (-%TDD) as a function of exercise intensity (% VO₂max) and duration, and then uses -%TDD to calculate daily adjustments to temporary basal rate (TBR), insulin-carbohydrate ratio (ICR), and correction factor (CF). Importantly, the constant 0.75 was empirically discovered to appropriately reduce the impact of the exercise duration component in the calculation of -%TDD as trial and error indicated inputting 100% of exercise duration resulted in over or under dosing of insulin at longer and shorter durations respectively. During the present case study, daily adjustments to TBR, ICR, and CF were manually programed into the athlete’s pump

immediately following completion of daily exercise, for the subsequent 24 hr period until the next training bout.

Table 9. Insulin Equation

	Calculation	Example ^a
-%TDD	$[\%VO_{2max} \times (0.75^b \times ED)] \div 100 = -\%TDD$	$[0.65 \times (0.75 \times 60)] \div 100 = 0.29$ (i.e., -29%)
TBR	$1 - (-\%TDD) = TBR$	$1 - 0.29 = 0.71$ (i.e., 71%)
ICR	$450^c \div (TDD_B \times TBR) = ICR$ consequent ^d	$450 \div (50 \times 0.71) = 12.67$ (i.e., 1:13)
CF	$94^e \div (TDD_B \times TBR) = CF$	$94 \div (50 \times 0.71) = 2.65$ (i.e., 2.7)

The insulin equation expresses daily insulin adjustments (**bold**) in the form in which they are programed into the 770G System insulin pump; TBR (percent), ICR (ratio), and CF (factor).

Clinical constants derived from Cornell et al. (2021) and King & Armstrong (2007).

Abbreviations: ED = exercise duration, -%TDD = percent reduction of total daily dose, TDD_B = baseline (i.e., without exercise) total daily dose, TBR = temporary basal rate, ICR = insulin-carbohydrate ratio, CF = correction factor.

^a Example of calculations for 60 min run at 65% VO₂max; TDD_B = 50.

^b 0.75 = empirically established constant.

^c 450 = clinical constant used to calculate ICR (i.e., $450 \div TDD_B = ICR$ consequent).

^d Where ratio is a:b, consequent = b.

^e 94 = clinical constant used to calculate CF (i.e., $94 \div TDD_B = CF$).

It should be noted that the athlete's %VO₂max during exercise was not measured in this case study, rather estimates of %VO₂max at various training paces were determined according to Jack Daniels' training pace calculator and the American College of Sports Medicine guidelines for aerobic exercise testing and prescription (Daniels, 2022; Liguori et al., 2022). Additionally, although we recognize expressing relative exercise intensity as % VO₂max has been criticized for its failure to account for individual differences in metabolic stress caused by level of fitness and other physiological factors (Mann et al., 2013), for the purpose of the present case study, we

have chosen to define exercise intensity as %VO₂max for ease of comparison with related studies.

No pre-exercise reductions to bolus doses were implemented, as all exercise was performed during the fasted-state throughout the 8-day data collection period (see “Exercise Timing” section). Likewise, no planned reductions of pre-exercise basal insulin were implemented, as this practice had been previously observed by athlete and coach to result in post-exercise hyperglycemia in the athlete. Basal rate suspension during exercise was always employed per the athlete’s preference to be “free” from her pump while running. To account for basal insulin “missed” while running, an equivalent bolus insulin dose was administered when pump therapy resumed immediately following exercise. Notably, post-exercise bolus insulin applied for missed basal insulin during exercise was delivered as a “square wave” (i.e., single bolus delivered evenly over an extended amount of time as specified by the user) over a period equal to the number of minutes basal rate was suspended during exercise. To our knowledge, this is a novel strategy, determined solely by athlete and coach.

While it is common for people with T1DM to lower their ICR in the morning to account for the apparent increase in insulin requirement for breakfast compared with lunch or dinner (i.e., “meal phenomenon”) (Blackard et al., 1989), throughout the 8-day data collection period, the athlete’s ICR was not lowered for breakfast as it was reported by the athlete that CGM readings prior to the case study indicated that lowering her ICR in the morning resulted in BG trending low around noon. However, it should be noted that early (i.e., 45 min pre-meal) compared with standard (i.e., 20 min pre-meal) administration of breakfast bolus insulin was found by the athlete to control prandial glycemic response most effectively. Therefore, early delivery of breakfast bolus insulin was practiced throughout the duration of the present case study.

Regarding baseline insulin titration, it is important to address the critical nature of establishing appropriate insulin total daily dose (TDD) and correct allocation of basal and bolus insulin before attempting insulin reduction strategies for glycemic management around exercise. Such strategies added on the foundation of poorly dosed baseline insulin, will be ineffective at best. Additionally, it has been noted that proper dose and timing of insulin administration can support glycemic management without weight gain (Brown et al., 2011). Though beyond the scope of the present case study, we hypothesize that programing the correct basal rate may be the key component underpinning effective pump therapy and insulin reduction strategies for exercise. In our experience, when basal insulin rate is poorly matched to HGP, glycemic excursions typically lead to compensatory prandial bolus doses, making an accurate ICR difficult to determine. When ICR is mismatched, glycemia may become a pendulum of excursions swinging between highs and lows. Therefore, we suggest the establishment of the athlete's baseline basal rate should be prioritized, followed by ICR titration, and then utilization of insulin reduction strategies for exercise. Several studies support basal rate programing according to circadian rhythms and demonstrate the validity of the so-called "dawn phenomenon" (Lindmeyer et al., 2020; Nauck et al., 2019). The present case study adds support for such an approach, observing a marked increase in the athlete's insulin requirement between 3:00–7:00 a.m. Though less prevalent, "dusk phenomenon" (Nauck et al., 2019), a secondary less prominent peak in insulin requirement at dusk (i.e., 4:00–7:00 p.m.), has also been noted by the athlete in the present case study. Notably, changes in fitness and body weight can influence the athlete's baseline insulin requirements. Therefore, periodic reevaluation of estimated baseline insulin requirement is encouraged (Breton et al., 2018). Research aimed at the development of expert advisory systems designed to reduce glycemic variation (GV) by providing automated titration

and dose of insulin is currently underway (Breton et al., 2018). Various methods for self-evaluating individual basal insulin requirements have been suggested, including 24 hr fasted tests targeted at detecting periods of inappropriate insulin infusion (Nauck et al., 2019). Athletes should be encouraged to work with their Certified Diabetes Care and Education Specialist to determine personalized TDD, basal rate, and ICR.

Exercise Timing and Strategies

Table 10 represents the athlete's training regimen during the case study. Per usual schedule, the athlete's training was conducted at 6:00 a.m. every day with no planned afternoon exercise, in alignment with previous studies indicating greater glycemic stability, including reduced late onset hypoglycemia, following morning exercise during the fasted-state as compared with post-prandial or afternoon exercise (Gomez et al., 2015; Scott et al., 2019b).

Table 10. Athlete Training Regimen During Case Study

Day	ED (min)	EI (%VO ₂ max)	Description
1	100	60%	long run
2	60	65%	easy run
3	60	60%	easy run
4	75	55%, 75%	tempo run ^a
5	60	55%, 85%	light pre-race workout ^b
6	105	60%, 45%, 85%	morning easy run, afternoon race ^c
7	50	50%	recovery jog
8	100	> 60%	long run

VO₂max estimated according to minutes per mile running pace recorded by the athlete's GPS running watch. Abbreviations: ED = exercise duration, EI = exercise intensity.

^a 20 min warm-up/cool down (55% VO₂max), 55 min tempo run (75% VO₂max).

^b 50 min run (55% VO₂max), strides (85% VO₂max).

^c 40 min morning run (60% VO₂max), 45 min warm-up/cool-down (45% VO₂max), ~20 min race (85% VO₂max).

Because the athlete was not connected to her pump during exercise, the athlete's self-perception of glycemia while running was relied upon to determine if action steps were required to prevent hypoglycemia during exercise. During the 6-month period prior to the present case study, the athlete self-reported greater tendency towards hypoglycemia during running > 30 min performed at < 60% VO₂max. To prevent hypoglycemia during lower intensity exercise (i.e., < 60% VO₂max), athlete and coach developed a "sprint protocol" in which four 60 m sprints (near-maximal effort), with a 20 s recovery jog between sprints, are employed when the athlete sensed her BG was trending low during exercise. This strategy was drawn from published research indicating that intermittent sprints before or after moderate-intensity aerobic exercise may attenuate hypoglycemia post-exercise due to elevated catecholamine levels resultant from sprint activity (Bussau et al., 2006, 2007). Additionally, a study conducted by Iscoe and Riddell (2011) involving 11 trained athletes with T1DM demonstrated that using exercise of a similar duration, continuous moderate-intensity exercise resulted in higher prevalence of nocturnal hypoglycemia than did continuous moderate-intensity exercise with intermittent high-intensity work (i.e., > 80% VO₂max). The authors suggested that this effect may be modulated by catecholamine response to high-intensity exercise to promote increased liver glycogenolysis (Iscoe & Riddell, 2011).

During the present case study, in the event of post-exercise hyperglycemia, the athlete was prescribed a prolonged cool-down; if BG was not within range after the prolonged cool-down, an insulin correction dose was considered depending on the severity of hyperglycemia and BG directional trend as indicated by the athlete's CGM. In keeping with published research regarding reductions in correction boluses post-exercise, correction insulin needs during the study were dosed according to the CF calculated by the Insulin Equation (Table 9).

Data Collection and Analysis

Electronic copies of the athlete's continuous glucose monitor (CGM) graphs, an 8-day food record, and training schedule during the 8-day case study were obtained from the athlete and her coach. In addition, CGM graphs from an 8-day period 6 months before the case study, as well as BG time in range (TIR; i.e., 3.9–10 mmol/L) values from two 14-day periods during the 18 months prior to the case study, were obtained for comparison. Glycemic control, measured as a percent of TIR, was used to assess the effects of the holistic strategies employed during the case study. Additionally, upon obtaining written and oral informed consent, and on days 1, 3, and 7 of the case study, the athlete completed a Student-Athlete Well-Being Scale (SAWS)TM survey (Table 13) (Curvey et al., 2019) to assess general well-being throughout the duration of the study. Though sometimes overlooked, we maintain the athlete's general well-being should never be compromised to obtain short-term performance goals. Although variables influencing glycemia can be tightly regulated to achieve target BG range (i.e., 3.9–10 mmol/L), the impact of restricted autonomy on general well-being should be considered (Fisher et al., 2009; Wisting et al., 2018). Additionally, a diet maintaining “perfect” glycemic control may not be sustainable if the athlete's well-being is negatively impacted. While methods used for optimizing glycemic control in this case study were intended to maintain individuality, choice, freedom, and flexibility within structure, the SAWS survey was implemented to ensure the athlete's well-being was not compromised by the strategies employed in the case study.

Finally, it should be acknowledged that the coach working in partnership with the athlete to develop the strategies observed here is the graduate student-researcher involved with the present case study. No coercion was placed on the athlete to participate in the case study or to complete the study. The athlete was engaging in typical training practices and engagement with

the coach. Per the athlete's usual routine, in-person athlete–coach check-ins were conducted daily throughout the duration of the 8-day data collection, but these meetings were not a part of the case study. Additionally, the athlete's RN Certified Diabetes Care and Education Specialist was available throughout the study in the event the athlete had any questions or concerns.

Results and Discussion

The present case study sought to investigate the effectiveness of recovery phase strategies in improving glycemic stability by evaluating the athlete's daily time in range (TIR) and dependence on acute strategies during the case study. We hypothesized that recovery phase strategies including a ModCHO diet and 24 hr insulin adjustment, and early morning exercise, would improve overall glycemic control and reduce reliance on acute (i.e., pre- and during-exercise) hypoglycemia strategies (i.e., CHOsup and insulin reduction), thereby promoting a positive feedback loop of improved glycemic stability (Figure 1).

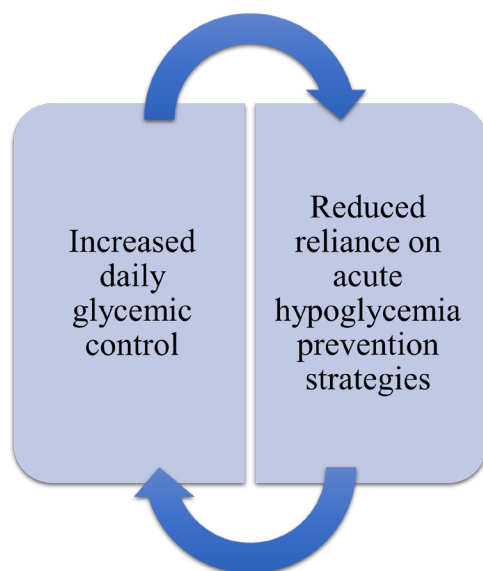


Figure 1. Glycemic Stability Positive Feedback Loop.

Depiction of the potential cyclic relationship between daily glycemic control (i.e., blood glucose time in range and magnitude of glycemic excursions) and acute (i.e., pre- and during exercise) strategies (i.e., carbohydrate supplementation and insulin reduction) for hypoglycemia prevention during exercise.

Daily Glycemic Control

Figures 2 and 3 provide a comparison of glycemic control during the present case study (i.e., January 24–31, 2022) and a prior 8-day period (i.e., July 19–26, 2021). Because the athlete used a different insulin pump during July 2021 than the pump she currently uses, the appearance and details included in the graphs are somewhat different. Nevertheless, the figures reveal increased mean TIR (i.e., 77% versus < 50%) and lower magnitude of glycemic excursions (i.e., ~3.8–15 versus ~3.0–26 mmol/L) during the current case study compared to the previous 8-day period. Notably, the athlete's mean TIR during the current 8-day case study was 77%, which exceeds current ADA guidelines for TIR (i.e., > 70% TIR) (American Diabetes Association, 2021). Moreover, as can be seen by figures 2 and 3, in addition to increased TIR, the extent of hyperglycemic excursions was markedly reduced during the case study, with BG never rising above 15 mmol/L during the case study compared with multiple departures between 15–26 mmol/L observed daily during the prior July 2021 period.

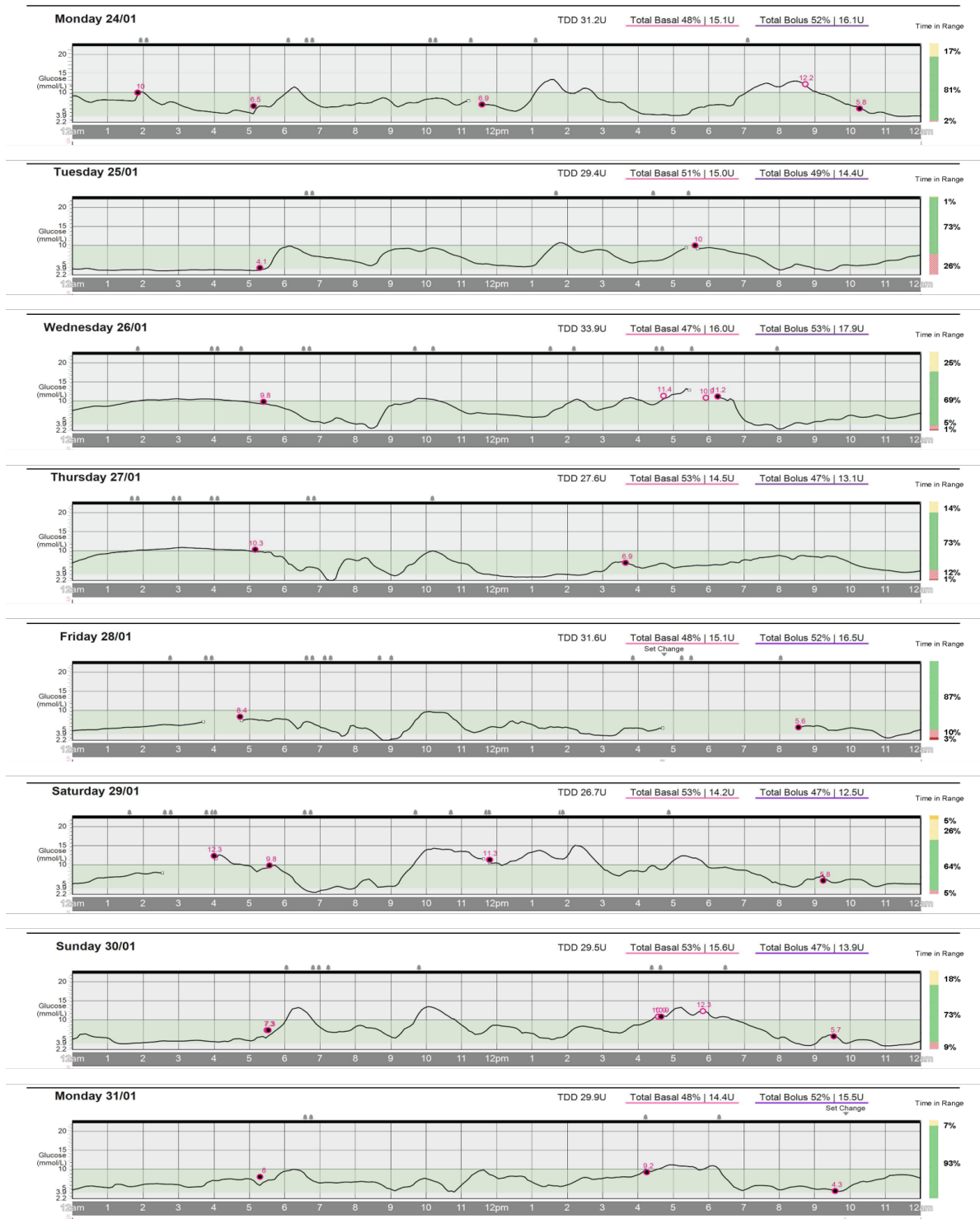


Figure 2. Daily Blood Glucose Data January 24–31, 2022.

Graphs depict data captured by the athlete's continuous blood glucose monitor (CGM) reflecting the athlete's daily blood glucose January 24–31, 2022; the light grey band represents target blood glucose range (i.e., 3.9–10 mmol/L). Abbreviations: TDD = total daily dose, U = units of insulin, ● = GGM sensor calibration.

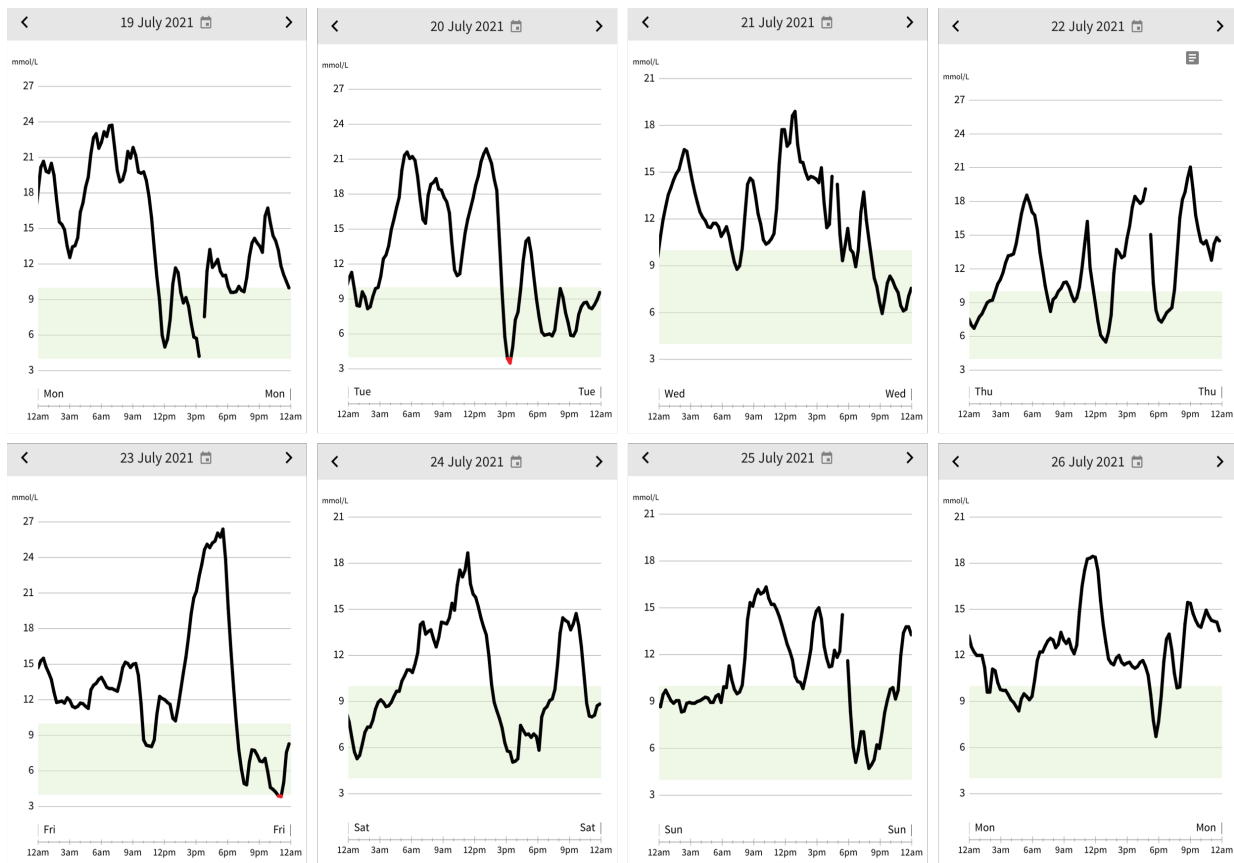


Figure 3. Daily Blood Glucose Data July 19–26, 2021.

Graphs depict data captured by the athlete’s continuous blood glucose monitor reflecting the athlete’s daily blood glucose July 19–26, 2021; the light grey band represents target blood glucose range (i.e., 3.9–10 mmol/L).

Additionally, Table 11 reflects the athlete’s time in range throughout two 14-day periods recorded during the 18 months prior to the case study. As can be seen, the athlete’s time in range improved by > 20% during the current 8-day case study compared with each of the 14-day comparison periods.

Table 11. Blood Glucose Time in Range During Case Study Compared with Prior Period

Data Collection Date	18-Month Period		
	July 17–30, 2020	October 23–November 5, 2020	January 24–31, 2022
Time in Range	56%	51%	77%

It should be noted that on Day 2 of the case study, glucose levels hovered just below 3.9 mmol/L between 12:00–5:00 a.m. While this may have been caused by poorly dosed insulin adjustments (i.e., 55% TBR, 1:16 ICR, 3.4 CF) on Day 1 or other uncontrolled variables, we suspect overcompensating for high (i.e., ~12 mmol/L) BG between 7:30–9:00 p.m. on Day 1 (Figure 2) may have resulted in hypoglycemia during the early hours of Day 2. In our experience, and as may have been the case here, fear of hyper- or hypoglycemia may elicit over-compensatory responses to glycemic variation (GV) that lead to further deterioration of glycemic control. Nonetheless, the evening of Day 1 provides an example of the numerous decisions required of athletes with T1DM, and illustrates the need for more research aimed at developing guidance and systematic approaches for decision-making regarding responses to GV, with consideration given to the potential for increased insulin sensitivity post-exercise. While more research is needed, we believe the development of data-informed decision algorithms may promote reduced decision-making burden and alleviate fear-induced over-compensatory responses.

Concerning the use of TIR to assess the effects of the strategies used during the current case study, while hemoglobin A1c (HbA1c, i.e., glycated hemoglobin) is considered the gold-standard for assessing diabetes disease development and progression, HbA1c lacks information regarding acute glycemic excursions and daily trends (Battelino et al., 2019). Alternatively, TIR

provides details about daily glycemic variation and may be a better indicator of overall glycemic control than HbA1c (Beck et al., 2018, 2019). While current ADA guidelines recommend aiming for > 70% TIR (American Diabetes Association, 2021), a recent survey of 1,026 patients with T1DM revealed that fewer than 20% of respondents perceived their current insulin therapy to be “very successful” in achieving target TIR (Runge et al., 2018). Although we were unable to identify published research reporting mean TIR among athletes with T1DM, we suspect perceived “success” in meeting ADA TIR guidelines may be similar or < 20% among CDRT1 due to the absence of established strategies for mitigating exercise-induced glycemic excursions in this population. Nevertheless, the results of the present case study demonstrate that it is possible to perform high-intensity aerobic exercise for > 60 min per day while maintaining > 70% TIR (Table 12). Moreover, results suggest holistic strategies including a ModCHO diet and 24 hr insulin adjustments, and morning exercise, may support meeting TIR guidelines in CDRT1.

Table 12. Case Study Results, Compiled Data

Day	Carbohydrate Intake (g)		Exercise		Insulin Adjustments				
	Daily	Pre-Exercise	During-Exercise	Duration (min)	% VO2max	TBR	ICR	CF	%TIR
1	240	30	-	100	60	55	1:16	3.4	81
2	240	30	-	60	65	65	1:13	2.7	73
3	225	-	-	60	60	70	1:13	2.6	69
4	175	-	-	75	55, 75	55	1:15	3.2	73
5	230	-	-	60	55, 85 60, 45,	70	1:12	2.6	87
6	210	-	-	105	85	60, 55	1:16	3.4	64
7	240	20	-	50	50	70, 80	1:13	2.7	73
8	240	20	-	100	> 60	55	1:16	3.4	93
Mean	225	12.5	0	~75	> 60				~77

VO₂max estimated according to minutes per mile running pace recorded by the athlete's GPS running watch. TBR, ICR, and CF reflect estimates calculated by Insulin Equation (Table 9); TBR is rounded to the nearest 5% and ICR consequent^a is rounded to the nearest whole number per 770G insulin pump TBR and ICR setting requirements. Days 6 and 7 each used two different TBR to account for afternoon competition on Day 6 (lowest TBR was used to calculate ICR and CF each day). Abbreviations: CF = correction factor, CHO = carbohydrate, ICR = insulin-carbohydrate ratio, TBR = temporary basal rate, TIR = time in range.

^a Where ratio is a:b, consequent = b.

Reliance on Acute Hypoglycemia Prevention Strategies

While existent strategies for athletic performance in T1DM are primarily focused on acute (i.e., pre- and during-exercise) CHOsup and insulin dose reduction to prevent hypoglycemia during exercise (Cockcroft et al., 2020), acute strategies may contribute to later deterioration of glycemic control in the athlete. Therefore, we hypothesized that daily strategies aimed at improving TIR during the recovery phase may increase glycemic stability during exercise and reduce dependence on acute strategies for hypoglycemia prevention.

Acute Carbohydrate Supplementation

Remarkably, CHOsup during exercise was never used during the case study, including two separate bouts of 100 min of running at > 60% VO₂max (Table 12; i.e., Days 1 and 8). This can be contrasted with published research suggesting intake of 75–100 g/hr, to support exercise > 60 min performed at > 60% VO₂max (Table 1). Moreover, pre-exercise CHOsup was only implemented when pre-exercise BG < 8 mmol/L (i.e., Days 1, 2, 7, and 8), and was ingested in relatively small amounts (Table 12; i.e., < 30 g). Notably, during the long runs completed on Days 1 and 8 of the case study, < 30 g CHO was ingested pre-exercise (i.e., ~5:00 a.m.), no CHO was consumed while running, and no acute insulin reductions were made. Importantly, the athlete's BG remained stable and in range (i.e., 3.9–10 mmol/L) for the duration of both long runs, and late (i.e., 6–12 hr post-exercise) hypoglycemia was not observed on either day (i.e.,

Days 1 and 8). These results may be compared with published research (Iscoe & Riddell, 2011) indicating the greatest risk for exercise-related hypoglycemia may be during or after continuous moderate-intensity ($> 50\%$ VO₂max) exercise, indicating the potential benefits of the holistic strategies applied during the case study in promoting glycemic stability during and after moderate-intensity long-duration exercise.

Acute Insulin Reduction

Additionally, no pre-exercise insulin dose reduction (i.e., < 2 hr pre-exercise) was employed throughout the 8-day data collection period, except before competition on Day 6 per the athlete's preference to further augment hypoglycemia avoidance prior to racing. However, it should be noted that pre-competition insulin reduction may have contributed to pre- and post-competition hyperglycemia, though other factors, such as competition-related stress, may have influenced the athlete's BG before and after racing (Jimenez et al., 2007). Nonetheless, preservation of the athlete's BG during all exercise performed throughout the case study in the absence of acute pre-exercise insulin reduction indicate the potential BG preserving effects of the strategies employed during the present case study. We suspect insulin adjustments made according to exercise duration and intensity facilitated insulin delivery that closely matched post-exercise insulin needs during the recovery phase, providing enough insulin to prevent hyperglycemia but not in excess requiring acute reduction to avoid hypoglycemia during subsequent exercise. Additionally, a ModCHO diet may have supported glycogen repletion without promoting hyperglycemia, thereby facilitating CHO availability and reducing need for pre-exercise insulin reduction. Furthermore, in agreement with published research, fasted-state morning exercise may have contributed to greater glycemic stability during training bouts due to

the absence of residual bolus insulin on board, eliminating the need for acute insulin reduction before morning exercise.

Although hypoglycemia can be avoided using acute (i.e., pre- and during-exercise) CHOsup and insulin reduction strategies, when these strategies are extended to apply to exercise durations and intensities commonly encountered by CDRT1, post-exercise hyperglycemia may occur, presumably because relatively high amounts of CHO intake and insulin reduction are recommended to preserve BG during the durations and intensities typical in college-level running (Grimm et al., 2004). Alternatively, the above results support our hypothesis that recovery phase strategies for glycemic control (i.e., ModCHO diet and 24 hr insulin adjustments) and morning exercise, may reduce reliance on acute hypoglycemia prevention strategies, promoting a positive feedback loop of improved glycemic stability (Figure 1), as evidenced by the athlete's mean TIR (i.e., 77%) during the case study.

Athletic Performance

While many factors may have contributed to the athlete's overall athletic improvement, we suggest the holistic strategies employed during the present case study may have promoted improved training and competition performance by supporting increased TIR and reduced reliance on acute hypoglycemia strategies. Notably, on Days 1 and 8 of the case study, the athlete completed the highest mileage per duration long run of her running career to date. Additionally, on Day 6 of the current case study, the athlete recorded a new lifetime best for 5,000 m on the track. New personal records during training runs and competition indicate the athlete's current fitness likely exceeds previous levels of fitness experienced by the athlete.

Well-Being

Results of the Student-Athlete Well-Being Score TM (Table 13) confirm that the athlete's well-being was not compromised during the case study. Additionally, the athlete reported feeling happier and more satisfied with her athletic performance during the 8-day case study than during the previous 6 months. While many uncontrolled factors may have influenced the athlete's well-being during the case study, improved TIR and reduced magnitude of glycemic excursions requiring treatment may have alleviated stress associated with glycemic variations. Moreover, we suspect utilization of the daily nutrition program and Insulin Equation may have eased the burden of diabetes-related decision-making, further promoting the athlete's well-being during the case study.

Table 13. Student-Athlete Well-Being Score Survey Results

	Measures	Student Athlete Response			
		Baseline	Day 1	Day 3	Day 7
Well-Being	1 I effectively manage my academic stress.	often	often	often	almost always
	2 I effectively manage my academic and athletic time demands.	often	almost always	often	almost always
	3 I effectively manage pressure related to my athletic performance.	almost always	often	often	almost always
	4 I am satisfied with my physical health.	often	almost always	almost always	almost always
	5 I am satisfied with my body.	often	almost always	almost always	almost always
	6 I have satisfying relationships with my family and other close relationships.	almost always	almost always	almost always	almost always
	7 I enjoy my sport.	almost always	almost always	almost always	almost always
	8 I am satisfied with my athletic performance.	almost always	almost always	almost always	almost always
	9 I feel happy.	almost always	almost always	almost always	almost always

			Student Athlete Response			
		Measures	Baseline	Day 1	Day 3	Day 7
Distress	10	I feel stressed.	sometimes	sometimes	sometimes	sometimes
	11	I feel worried.	sometimes	sometimes	sometimes	never

Adapted from Curvey et al., 2019; designed as a screening and outcome measure of student-athlete well-being using a four-point Likert scale ranging from never–sometimes–often–almost always.

Recovery Phase Strategies

In addition to daily glycemic control, reliance on acute hypoglycemia strategies, athletic performance, and well-being, the recovery phase and exercise strategies (i.e., daily nutrition program, 24 hr insulin adjustments, and exercise timing and strategies) themselves are also part of the overall results.

Daily Nutrition Program

With consideration given to published research, feasibility, sustainability, and the athlete’s preference and experience, a low-GL ModCHO diet was chosen. Table 14 reflects the athlete’s food diary from the 8-day collection period and includes macronutrient intake estimates calculated using cronometer.com (Revelstoke, British Columbia, Canada). Notably, the athlete’s mean daily CHO intake during the current case study was 225 g/day, which may be contrasted with intake < 130 g/day prior (i.e., > 6 months) to the case study according to athlete nutrition recall. The athlete’s limited use of acute strategies for exercise-induced hypoglycemia and 77% mean TIR during the case study suggest a low-GL ModCHO diet can support daily glycemic control and reduce reliance on acute strategies for hypoglycemia prevention. Moreover, we suspect that moderate (i.e., 4 g/kg/day) compared with low (i.e., < 3 g/kg/day, or < 130 g/day) CHO intake may have improved glycogen repletion, potentially improving CHO availability

during exercise and reducing reliance on acute hypoglycemia strategies. Additionally, a ModCHO diet compared with recommended daily intake of CHO for endurance athletes without diabetes (i.e., 6–10 g/kg/day) may have supported sufficient glycogen repletion without compromising glycemic control. These findings are consistent with published research indicating high CHO intake may promote deteriorated glycemic control in people with T1DM.

Table 14. Athlete Food Diary

Day	Meal				
	Pre-Run	Breakfast	Lunch	Dinner	Bedtime Snack
24-Jan	3 medjool dates (30 g CHO)	1 cup homemade granola (60 g CHO, 10 g PRO, 15 g fat)	pesto-chicken & roasted vegetable whole wheat calzone (70 g CHO, 40 g PRO, 15 g fat)	pesto-chicken & roasted vegetable whole wheat calzone with 1/4 cup cottage cheese (70 g CHO; 45 g PRO, 15 g fat)	~1 cup low-fat cottage cheese (10 g CHO, 20 g PRO, 4 g fat)
25-Jan	3 medjool dates (30 g CHO)	1 cup homemade granola (60 g CHO, 10 g PRO, 15 g fat)	BBQ chicken, roasted sweet potato, & kale power bowl (70 g CHO, 40 g PRO, 10 g fat)	BBQ chicken, roasted sweet potato, & kale power bowl (70 g CHO, 40 g PRO, 10 g fat)	~1 cup low-fat cottage cheese (10 g CHO, 20 g PRO, 4 g fat)
26-Jan	none	1 cup homemade granola with 1 cup whole milk (70 g CHO, 20 g PRO, 20 g fat)	whole wheat pocket filled with chicken, broccoli, & cheese (70 g CHO, 50 g PRO, 10 g fat) ^a	whole wheat pocket filled with chicken, broccoli, & cheese, 4 oz baby carrots (75 g CHO, 50 g PRO, 10 g fat)	~1 cup low-fat cottage cheese (10 g CHO, 20 g PRO, 4 g fat)

Day	Meal				
	Pre-Run	Breakfast	Lunch	Dinner	Bedtime Snack
27-Jan	none	1 cup homemade granola (60 g CHO, 10 g PRO, 15 g fat)	chicken, feta, & roasted vegetable whole wheat penne pasta salad (70 g CHO, 30 g PRO, 15 g fat)	1/2 chicken, feta, & roasted vegetable pasta salad with 1 cup cottage cheese (45 g CHO, 35 g PRO, 20 g fat)	none
28-Jan	none	1 cup homemade granola with 1 cup whole milk (70 g CHO, 20 g PRO, 20 g fat)	chicken, feta, & roasted vegetable whole wheat penne pasta salad (70 g CHO, 30 g PRO, 15 g fat)	chicken, feta, & roasted vegetable whole wheat penne pasta salad (70 g CHO, 30 g PRO, 15 g fat)	~2 cups low-fat cottage cheese (20 g CHO, 40 g PRO, 8 g fat)
29-Jan	none	1 cup homemade granola with 1 cup whole milk (70 g CHO, 20 g PRO, 20 g fat)	whole wheat pocket filled with chicken, broccoli, & cheese (70 g CHO, 50 g PRO, 10 g fat)	whole wheat pocket filled with chicken, broccoli, & cheese (70 g CHO, 50 g PRO, 10 g fat)	none
30-Jan	2 medjool dates (20 g CHO)	1 cup homemade granola with 1 cup whole milk (70 g CHO, 20 g PRO, 20 g fat)	whole wheat pocket filled with chicken, broccoli, & cheese (70 g CHO, 50 g PRO, 10 g fat)	1 cup homemade granola with 1 cup low-fat cottage cheese (80 g CHO, 30 g PRO, 20 g fat)	none
31-Jan	2 medjool dates (20 g CHO)	1 cup homemade granola with 1 cup whole milk (70 g CHO, 20 g PRO, 20 g fat)	chicken, feta, & roasted vegetable whole wheat penne pasta salad (70 g CHO, 30 g PRO, 15 g fat)	chicken, feta, & roasted vegetable whole wheat penne pasta salad (70 g CHO, 30 g PRO, 15 g fat)	~1 cup low-fat cottage cheese (10 g CHO, 20 g PRO, 4 g fat)

Day	Meal				
	Pre-Run	Breakfast	Lunch	Dinner	Bedtime Snack

Macronutrient content estimated using cronometer.com (Revelstoke, British Columbia, Canada). Abbreviations: CHO = carbohydrate, PRO = protein.

24 hr Insulin Adjustments

We hypothesized that calculating daily adjustments in insulin TDD as a function of exercise duration and intensity would provide more accurate estimates of daily insulin requirements than the broad range (i.e., 20–50%) currently provided by published research (Scott et al., 2021). To that end, exercise duration and intensity were used as variables in an equation (Table 9) developed by athlete and coach to predict appropriate reductions in the athlete’s insulin TDD, which was then used to calculate daily TBR, ICR, and CF. Table 12 reflects the daily TBR, ICR, and CF calculated by the Insulin Equation and programed into the athlete’s insulin pump during each day of the 8-day collection period, along with corresponding daily TIR.

As indicated in Table 12, daily TIR exceeded ADA guidelines (i.e., > 70%) for all but 2 days (i.e., 69% Day 3, and 64% Day 6) during the case study, with overall mean TIR above (i.e., 77%) ADA guidelines, indicating insulin adjustments according to daily exercise intensity and duration can support glycemic control during training for college-level distance running. Notably, Day 6 of the case study, the athlete completed her first race since April 2021, competing in the 5,000 m run at 1:30 p.m. Although the lowest %TIR (i.e., 64%) recorded during the 8-day data collection period, glycemic control was markedly better on Day 6 than previous competition days (i.e., < 40% TIR) according to athlete self-report, suggesting insulin adjustments according to daily exercise intensity and duration may also support improved glycemic stability on race days. While pre- and post-race hyperglycemia occurred, possibly due

to competition-related stress, remarkably, no post-exercise hypoglycemia was observed during the recovery phase on Day 6, indicating the potential for insulin adjustments based on exercise intensity and duration to mitigate late- and nocturnal hypoglycemia in CDRT1 following afternoon competition.

Exercise Timing and Strategies

Each day of the 8-day collection period the athlete initiated daily exercise at 6:00 a.m., including on the day of competition (i.e., Day 6) in which a 40 min easy run (i.e., 60% VO₂max) was employed to control for potential GV that may have occurred without her usual morning exercise. In agreement with published research and the results of the current case study (i.e., 77% mean TIR and minimal use of acute strategies), we maintain morning exercise during the fasted-state may support glycemic stability during and after exercise. When morning exercise is not possible, initiating pre-exercise mealtime bolus insulin > 5 hr before exercise may minimize the effects of lingering bolus insulin during exercise. This strategy was implemented on Day 6 of the case study in which the athlete's breakfast bolus was delivered at 7:00 a.m., > 5 hr before starting her pre-race warm-up (i.e., 1:00 p.m.). Additionally, to account for post-competition hyperglycemia, possibly resultant from a combination of excessive acute insulin reduction and hormonal response to competition (i.e., elevated catecholamine activity) (Marliss & Vranic, 2002), a prolonged cool-down (i.e., 35 min) was successfully implemented to bring the athlete's BG back into range.

Clinical Implications

As with any case study, there are clear limitations regarding generalizability of any of the outcomes. Nonetheless, the strategies provided herein, and the equations developed during the course of the case study may benefit other collegiate endurance athletes and coaches by

providing potential starting points for daily nutrition programming, insulin TDD reduction calculations, and strategies for exercise timing. While these strategies will require further research in order to determine generalizability, Figure 4 highlights several possible implications that may be drawn from the present case study. Perhaps most notably, our experience and limited published research indicate that adjustments to insulin TDD based on intensity and duration of exercise may support improved glycemic control and recovery in athletes with T1DM. While more research is needed to establish the relationship between various intensities and durations of exercise and reduced insulin requirements post-exercise, the present case study provides a promising starting point for future research.

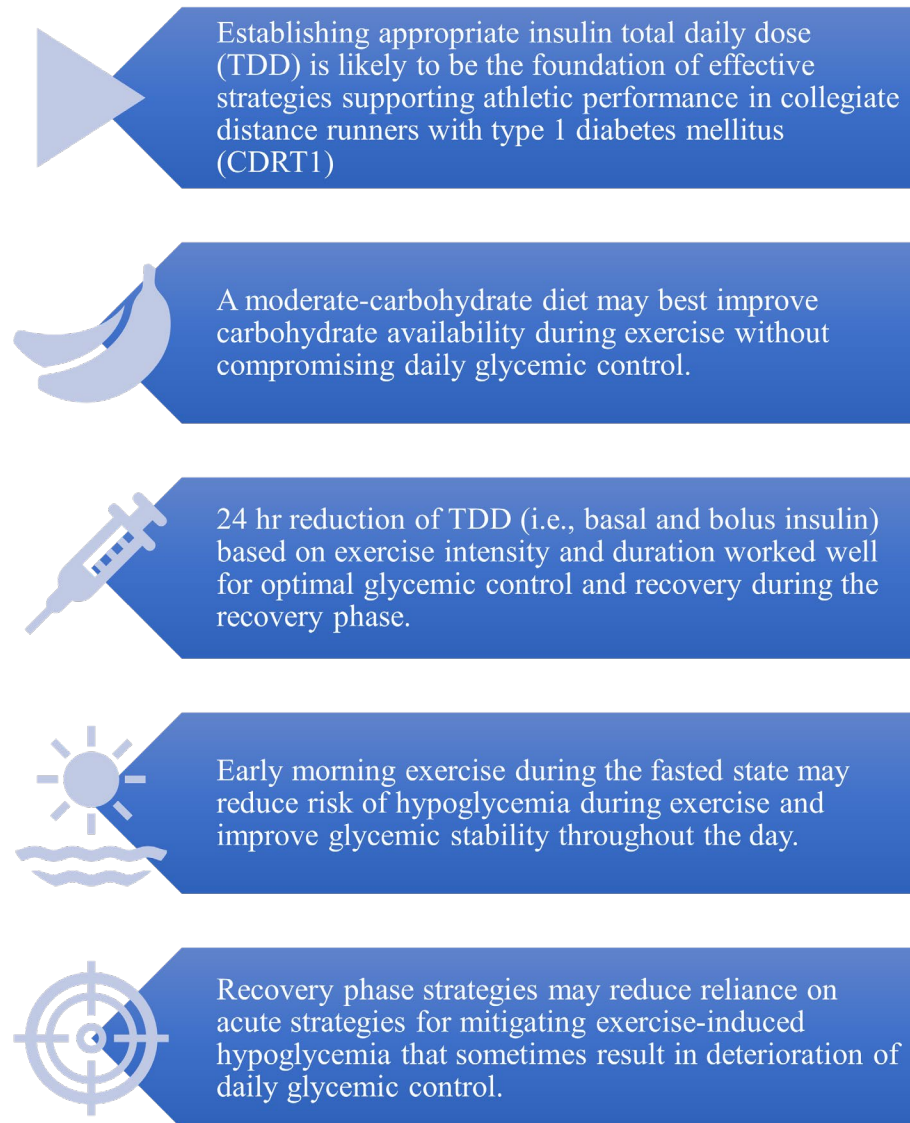


Figure 4. Clinical Implications.

Clinical implications from an 8-day case study in which a collegiate distance runner with type 1 diabetes mellitus utilized recovery phase (i.e., period between workout bouts) strategies for glycemic control, including a moderate-carbohydrate nutrition program, daily 24 hr insulin adjustments based on exercise intensity and duration, and early morning exercise regimen.

Case Study Limitations

As discussed above, generalizability is a clear limitation of any case study. As such, the strategies employed during this case study will require further research in order to determine generalizability of any of the outcomes. The use of VO₂max estimates based on the athlete's

pace per mile to calculate insulin reductions using the Insulin Equation is an obvious limitation of the present case study. We acknowledge that laboratory measurements of VO₂max would provide a clearer assessment of insulin adjustments (i.e., TBR, ICR, and CF) calculated by the Insulin Equation. However, estimates based on field tests such as the one used in the current case study may be valuable for athletes without access to laboratory assessments. However, population studies using laboratory measurement of VO₂max to develop advanced algorithms for automated insulin pump delivery or manual calculations for daily insulin adjustments may be a potential opportunity for future research. The comparison of data (i.e., CGM graphs and TIR) collected during the current case study with previously collected data from periods where a different CGM and insulin pump were used, presents another potential limitation of the present case study. While differences in technology may have partially explained differences in glucose outcomes, other case study outcomes including improved athletic performance and positive well-being suggest negligible influence of CGM/insulin pump differences on current results. Finally, it should be noted that due to the athlete choosing to be disconnected from her insulin pump while exercising, the athlete's precise BG during exercise throughout the case study is unknown. Moreover, BG reflected in Figure 2 at the time of exercise may not be accurate as the athlete's BG sensor may have been beyond the necessary proximity of her insulin pump for accurate data capture. Nonetheless, based on BG measurements before and after exercise and athlete-perceived glycemia during exercise, it appears that the athlete's BG remained in a safe range during all exercise performed throughout the case study.

Conclusion

The present case study demonstrates the potential for a distance runner (i.e., 5,000 and 10,000 m) with T1DM to successfully train and compete at the collegiate level without compromising glycemic control and general well-being, and provides specific strategies for athletic performance in CDRT1. The strategies outlined in the present case study may offer a beneficial alternative to existent acute strategies for athletes with T1DM by potentially supporting increased TIR and reduced reliance on acute strategies for hypoglycemia prevention. Holistic strategies that emphasize the recovery phase, including consuming a low-GL ModCHO diet, 24 hr insulin dose adjustment based on exercise duration and intensity, and fasted-state morning exercise, may improve glycemia during exercise and the recovery phase. Together, these strategies may ultimately improve athletic performance and promote general well-being (Figure 5).

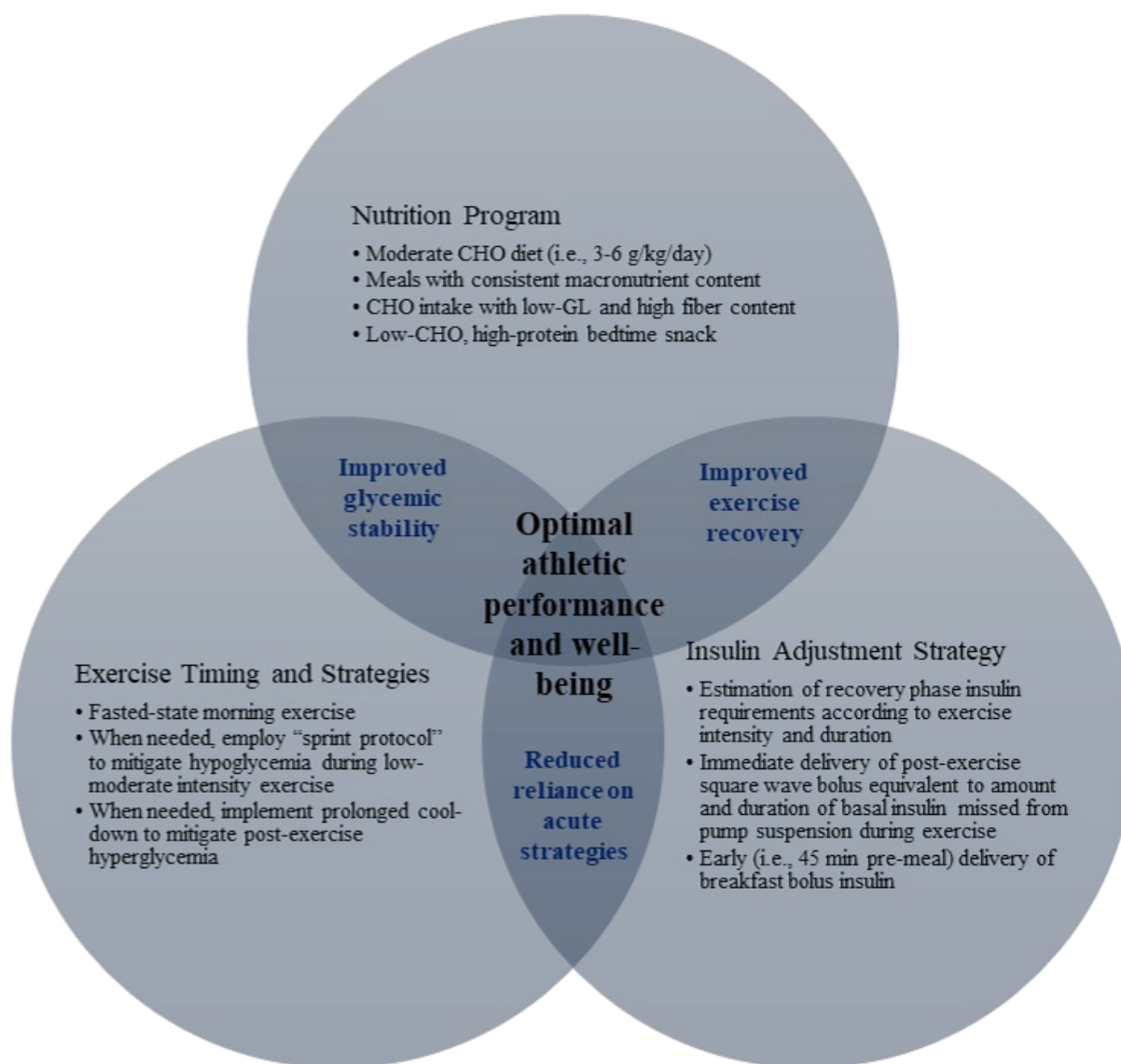


Figure 5. Recovery Phase Strategies for Athletic Performance and Well-Being.

Venn diagram depicting how recovery phase (i.e., period between workout bouts) nutrition, insulin, and exercise strategies may improve glycemic stability and exercise recovery, while minimizing acute strategies for hypoglycemia prevention, thereby supporting optimal athletic performance and wellbeing in a collegiate distance runner with type 1 diabetes mellitus.

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