

# Type 1 Diabetes and Vigorous Exercise: Applications of Exercise Physiology to Patient Management

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*This manuscript is based in part on a presentation given at the 8th Annual Canadian Diabetes Association Professional Conference and Annual Meetings, October 27–30, 2004, Quebec City, Quebec, Canada*

## A B S T R A C T

Special considerations are needed for the physically active individual with type 1 diabetes mellitus. Although regular activity is beneficial for all patients, vigorous exercise can cause major disturbances in blood glucose. The glycemic response depends largely on the type, intensity and duration of the activity, as well as the circulating insulin and glucose counterregulatory hormone concentrations. This review highlights a number of strategies to optimize blood glucose levels in patients with type 1 diabetes who exercise vigorously.

## R É S U M É

Il faut prendre des mesures particulières chez une personne atteinte de diabète de type 1 qui est physiquement active. L'activité régulière profite à tous les patients, mais les exercices violents peuvent provoquer de graves déséquilibres de la glycémie. La réponse glycémique dépend largement de la nature, de l'intensité et de la durée de l'activité, de même que des concentrations circulantes d'insuline et d'hormones de la contre-régulation glycémique. Ce compte rendu présente plusieurs stratégies visant à optimiser la glycémie chez les patients atteints de diabète de type 1 qui font des exercices violents.

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**Keywords:** aerobic exercise, anaerobic exercise, hyperglycemia, hypoglycemia

## INTRODUCTION

Despite decades of improved insulin therapy and significant advancements in blood glucose (BG) monitoring, large excursions in BG concentration remain a major challenge for the active person with type 1 diabetes mellitus. The purpose of this review is to highlight the benefits and risks associated with vigorous exercise, discuss the possible metabolic responses to various forms of exercise and suggest management strategies for patients who participate in vigorous exercise.

## BENEFITS AND RISKS OF EXERCISE IN DIABETES MANAGEMENT

Even before the 19th century, it was known that BG concentrations typically decrease with endurance-type exercise in most individuals with diabetes (1). In the 1950s, the American physician E.P. Joslin emphasized the importance of regular physical activity to effectively manage his patients' symptoms. His idea of 'victory' was the triad of nutrition, insulin and regular exercise to properly manage BG levels and thus provide a life free from complications from diabetes.

For patients with either type 1 or type 2 diabetes, there are both benefits and risks of regular exercise (Table 1). An individual's unique characteristics (e.g. age, sex, psychosocial milieu), comorbid medical conditions and medications need to be considered by healthcare providers when prescribing a training program. In fact, target work rates, used to determine training intensity, require modification and defined limits in the presence of coronary heart disease, hypertension or microvascular complications. In particular, the type and intensity of exercise may need to be limited in some patients with retinopathy and neuropathy.

Given the demonstrated benefits of low- to moderate-intensity exercise, with its minimal associated risks, the benefits of regular physical activity almost certainly outweigh the potential side effects in the majority of individuals with diabetes, even those with some complications from the disease. Unfortunately, training and competition are frequently associated with either hypo- or hyperglycemia in active people with diabetes and very little is known about the effects of diabetes on athletic performance.

## AEROBIC VS. ANAEROBIC EXERCISE

Exercise can be classified into 2 forms—anaerobic and aerobic—based on the dominant metabolic energy sources used during the activity. *Anaerobic* activities are characterized by higher intensities of muscular contraction. Contractions are sustained by the phosphagen and anaerobic glycolytic systems to produce lactic acid and energy in the form of adenosine triphosphate. *Anaerobic* activities include sprinting, power lifting, hockey and some motions during basketball and racquet sports. *Anaerobic fitness* refers to the ability to work at a very high level during these activities for relatively short periods (5 to 30 s). *Aerobic* activities are characterized by lower rates of muscular contraction. These contractions

usually have more prolonged durations and use carbohydrates, fats and some protein for oxidation by mitochondria within the muscle. *Aerobic* metabolism is the primary method of energy production during endurance activities such as running, cycling, rowing, swimming, soccer and ultra-endurance events. *Aerobic fitness* indicates the endurance capacity ( $VO_{2max}$ ) of the individual's heart, lungs and muscles that allows him/her the ability to offset fatigue over the course of an activity (game, practise, competition, etc.). It is crucial to note that these and similar activities often include short bursts of anaerobic metabolism. The distinction between the 2 types of exercise is important because of their distinct effects on BG concentration. For example, many individuals find that aerobic-type exercise causes BG to decrease both during and post-activity. On the other hand, anaerobic activities, which may only last for seconds, tend to cause dramatic increases in BG levels.

## MECHANISMS OF GLUCOSE REGULATION DURING AEROBIC EXERCISE

To understand the possible metabolic responses to exercise in diabetes, it is useful to first describe the mechanisms of glucose regulation in people without diabetes.

### People without diabetes

The increased metabolic demand of exercise requires a dramatic increase in fuel mobilization from sites of storage and an increase in fuel oxidation within the working muscle. Normally, the increase in fuel mobilization for oxidation is under neuroendocrine control. During the transition from rest to exercise, the working muscles shift from using predominantly free fatty acids released from adipose tissue to a complex mixture of circulating fats, muscle triglycerides (TG), muscle glycogen and BG derived from liver glycogen.

**Table 1. Benefits and risks of regular exercise in diabetes**

Benefits	Risks
<ul style="list-style-type: none"> <li>• Potentially lower A1C in children and adolescents with type 1 diabetes</li> <li>• Reduced risk of CVD, hypertension, colon cancer, obesity and osteoporosis</li> <li>• Increased overall life expectancy</li> <li>• Increased CV endurance, muscle fitness and flexibility</li> <li>• Increased whole-body insulin sensitivity</li> <li>• Enhanced self-esteem and sense of well-being</li> </ul>	<ul style="list-style-type: none"> <li>• Hyperglycemia</li> <li>• Hypoglycemia</li> <li>• Musculoskeletal injury</li> <li>• CV accident (angina, myocardial infarction, dysrhythmia, sudden death)</li> <li>• Deterioration of underlying retinopathy and nephropathy</li> </ul>

A1C = glycosylated hemoglobin  
CVD = cardiovascular disease

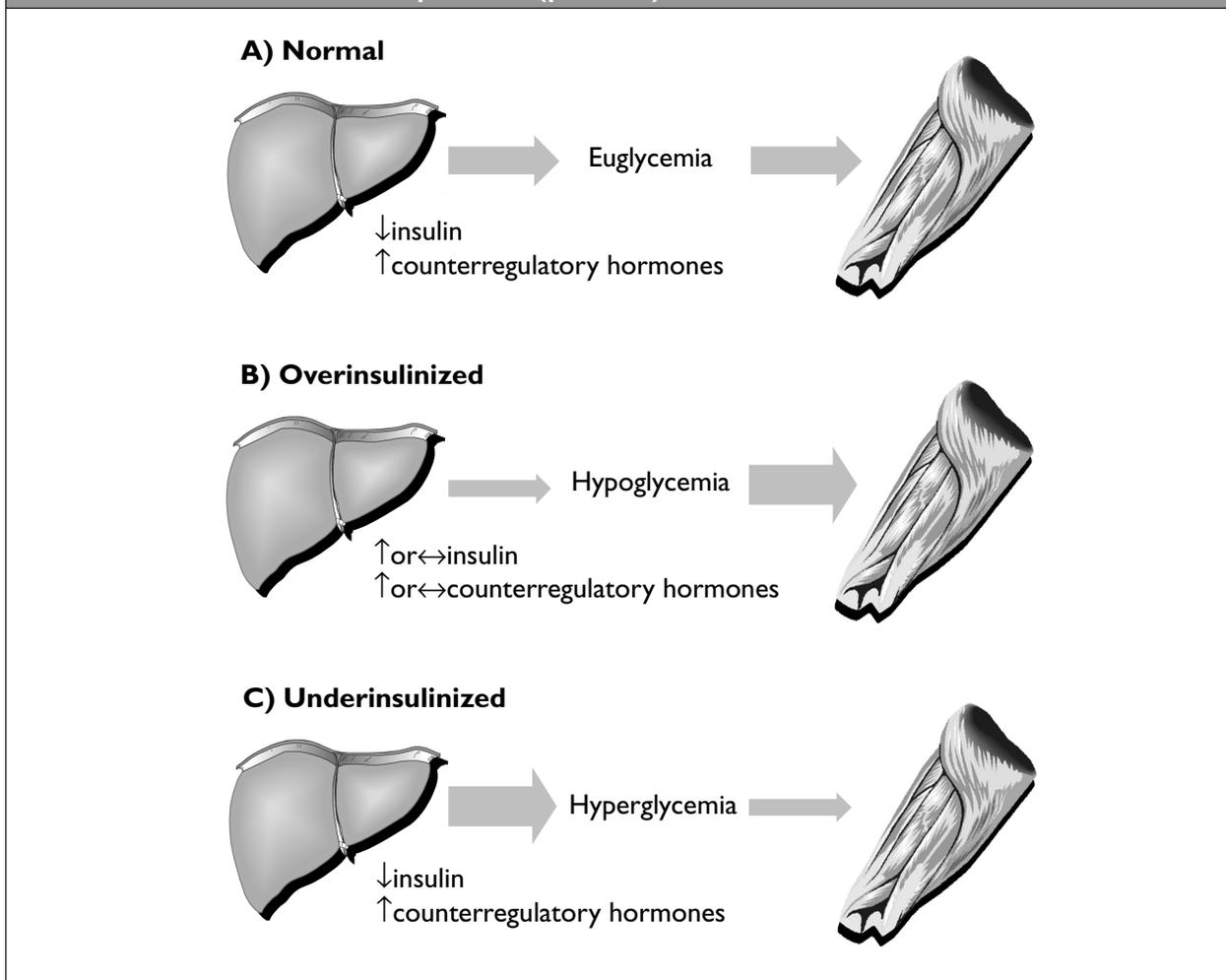
During the initial stages of exercise, muscle glycogen is the main source of energy, but the reliance on this limited fuel source decreases as the duration of exercise increases. As a result, contributions from circulating free fatty acids and glucose in the blood stream increase to replace diminishing muscle glycogen stores. This greater reliance on liver glycogen can have dramatic effects on BG levels.

The mixture of fuel utilization differs markedly depending on the intensity of exercise. During low to moderate intensities, plasma-derived free fatty acids make up the majority of oxidized substrate. As the intensity increases, there is a greater reliance on carbohydrates. During heavy

exercise, BG utilization may be as great as 1 to 1.5 g/min and this fuel source must be continuously replaced at an equal rate or hypoglycemia will ensue (2). The mix of fuel utilization during exercise in people with type 1 diabetes appears to be similar to that of people without diabetes, except that individuals with diabetes may have a slightly greater reliance on fat as an energy source and a slightly lower rate of carbohydrate oxidation (3,4).

To facilitate the changes in glucose delivery during exercise, pancreatic insulin secretion decreases and circulating levels of glucagon, growth hormone, cortisol and catecholamines increase. The primary role of these hormonal changes is to

**Figure 1. Schematic illustration of the BG response to exercise in non-diabetic or ideally controlled persons with diabetes (panel A), overinsulinized patients (panel B) or underinsulinized patients (panel C)**



BG balance is primarily a function of circulating insulin levels, counterregulatory hormone levels, parameters related to the exercise itself (mode, duration, intensity) and characteristics of the individual. In this schema, the thickness of the block arrows represents BG flux. In panel A, glucose production matches glucose utilization and BG concentration is maintained in a euglycemic state. In panel B, a relatively high insulin concentration lowers hepatic glucose production and may further enhance glucose uptake resulting in a decrease in BG concentration. In panel C, a relatively low insulin concentration or an elevation in glucose counterregulatory hormone levels increase hepatic glucose production and lower glucose uptake resulting in an increase in BG concentration.

BG = blood glucose

ensure an adequate supply of glucose for the exercising muscles (Figure 1A). Usually, the magnitude of change in these hormones is greater with increasing exercise duration and intensity. That is, during prolonged heavy aerobic exercise (i.e. exercising for over 30 min at 60 to 80% of  $\text{VO}_{2\text{max}}$ ), the reduction in insulin secretion is more pronounced, while the release of the other glucose counterregulatory hormones is increased to a greater extent.

### People with type 1 diabetes

In the individual with type 1 diabetes, the pancreas does not regulate insulin levels in response to exercise, making normal fuel regulation nearly impossible. Moreover, there may be deficiencies in the release of glucose counterregulatory hormones that would normally help facilitate glucose production and release by the liver. As patients quickly discover, they may have either increases or decreases in BG levels during exercise. The inability to regulate the delivery of exogenous insulin into the bloodstream based on “real-time” glucose measurements and the failure to reduce insulin levels during exercise severely hampers the ability to exercise in a euglycemic state. The following sections outline the typical problems of over- and underinsulinization during exercise that cause hypo- and hyperglycemia, respectively.

## OVERINSULINIZATION AND HYPOGLYCEMIA

The results of the Diabetes Control and Complications Trial clearly show that near-normal and normal glycosylated hemoglobin (A1C) levels limit the progression of long-term complications from diabetes (5). Indeed, most competitive athletes with diabetes may find that intensive insulin therapy, particularly insulin pump therapy, helps with BG management during exercise, since it allows for frequent changes in insulin dosages.

Although tight metabolic control is desirable, the move toward more aggressive insulin therapy increases the risk of exercise-associated hypoglycemia for some active people with diabetes. Simply stated, hypoglycemia is the most severe acute complication of intensive insulin treatment, and exercise is a common behaviour that causes hypoglycemia (2).

Intensive insulin therapy, whether it occurs via subcutaneous injection or via insulin pump, frequently causes overinsulinization and hypoglycemia in active individuals with type 1 diabetes. Several factors contribute to overinsulinization and hypoglycemia during exercise:

1) The absorption of subcutaneously injected insulin may be increased with exercise. The increase in subcutaneous and skeletal muscle blood flow resulting from exercise can be associated with a concurrent increase in insulin absorption and accelerated hypoglycemia (6). In addition, a rise in body temperature may increase insulin absorption rate and the incidence of hypoglycemia. It is important to note that exercise does not appear to alter insulin glargine absorption rate (7).

2) Plasma insulin levels do not decrease during exercise. The inability to decrease insulin levels during exercise after injection causes a relative hyperinsulinemia that impairs hepatic glucose production and initiates hypoglycemia, usually within 20 to 60 min after the onset of exercise (8,9).

3) There is an exercise-induced increase in skeletal muscle insulin sensitivity. During exercise there is a dramatic increase in non-insulin-mediated muscle glucose uptake that considerably reduces the need for circulating insulin levels (2). Even when insulin dose is decreased prior to exercise, there is often a relative overinsulinization in patients, as the pharmacokinetics of injected insulin do not precisely meet the body's requirements for a prolonged decrement of insulin secretion. Since the increase in insulin action persists for several hours after the end of exercise (likely to help replenish muscle and liver glycogen stores), patients are at increased risk of hypoglycemia for several hours after the cessation of exercise (Figure 1B) (10).

### Counterregulatory failure

Hypoglycemia during exercise may result from impaired release of glucose-counterregulatory hormones caused by previous exposure to either exercise or hypoglycemia (Figure 1B). Normally, both hypoglycemia and exercise cause similar increases in glucagon secretion, reductions in insulin secretion, sympathetic nervous system activity and activation of the hypothalamic-pituitary-adrenal axis. Neuropathic complications of diabetes and poor glycemic control reduce the counterregulatory responses to either exercise or hypoglycemia (2). Not surprisingly, this finding of a blunted counterregulatory response to exercise is similar to the scenario that occurs in intensively treated patients with diabetes who develop defects in counterregulatory responses to hypoglycemia (11). For those pursuing vigorous aerobic activity, a vicious cycle may be created whereby an episode of prior hypoglycemia may reduce counterregulatory responses to subsequent exercise, thereby predisposing the individual to hypoglycemia. This, in turn, could blunt counterregulatory responses to additional episodes of exercise and/or hypoglycemia. As a result, individuals with type 1 diabetes who experience hypoglycemia on days preceding competition may have an elevated risk of hypoglycemia and autonomic counterregulatory failure during exercise. It should also be noted that the energy expenditure itself will predispose the individual to hypoglycemia for ~24 h after the end of exercise, as insulin sensitivity remains elevated (see point 3 above) (10).

## UNDERINSULINIZATION AND HYPERGLYCEMIA

In individuals with poor metabolic control, exercise can cause an additional increase in BG concentration and ketoacidosis. The rise in BG is caused by exaggerated hepatic glucose production and impairment in exercise-induced glucose utilization (Figure 1C). Hyperglycemia and excessive ketosis

during exercise are particularly undesirable since they cause dehydration and may decrease blood pH, both of which impair exercise performance. Intense exercise (i.e. >60 to 70%  $\text{VO}_{2\text{max}}$  or >75 to 85% of maximal heart rate) may particularly aggravate this condition, since increases in catecholamines and glucocorticoids will further exaggerate the elevations in BG concentrations and ketone production (12).

### HIGH-INTENSITY EXERCISE AND HYPERGLYCEMIA

High-intensity exercise may be defined as activities above the “lactate threshold,” which is approximately >60 to 70%  $\text{VO}_{2\text{max}}$  or 85 to 90% maximal heart rate. This threshold coincides with dramatic elevations in catecholamines, free fatty acids and ketone bodies, all of which impair muscle glucose utilization. Even those individuals on multiple daily insulin injections or those on pump therapy may have increases in BG levels during and after high-intensity exercise (13), likely due to a failure in insulin release to offset the increases in counterregulatory hormones (Figure 1C). This rise in BG concentration is usually transient and tends to last only as long as there are elevations in counterregulatory hormones (i.e. 30 to 60 min). Although some individuals can

easily correct the elevations with an insulin bolus, particularly if they take rapid-acting insulin analogues, others may be resistant to taking additional insulin following exercise, since there will be greater risk of late-onset post-exercise hypoglycemia in the next several hours (particularly if the prior exercise bout was >30 min).

### COMPETITION STRESS, HEAT STRESS AND HYPERGLYCEMIA

The psychological stress of competition is frequently associated with increases in BG levels even if the pre-exercise BG concentrations are normal. Those pursuing vigorous aerobic exercise may find that on regular training or practice days they become hypoglycemic, but on the day of competition they develop hyperglycemia. Although empirical data do not exist for patients with type 1 diabetes, excessive increases in counterregulatory hormones likely occur just prior to exercise, when anticipatory stress is high. It is also probable that the stress during competition can further increase BG levels. The elevated levels of these stress hormones are known to increase hepatic glucose production dramatically and decrease peripheral glucose uptake. In people with diabetes, the body's failure to compensate for the “stress” associated

**Table 2. Practical guidelines to limit BG excursions before, during and after exercise**

<b>Before exercise</b>	<ul style="list-style-type: none"> <li>• Determine the timing, mode, duration and intensity of exercise</li> <li>• Eat a carbohydrate meal 1–3 h prior to exercise</li> <li>• Assess metabolic control:               <ul style="list-style-type: none"> <li>– If BG is &lt;5.0 mmol/L and levels are decreasing, extra calories may be needed</li> <li>– If BG is 5–13.9 mmol/L, extra calories may not be needed, depending on the duration of exercise and individual response to exercise</li> <li>– If BG is <math>\geq 14.0</math> mmol/L and urine or blood ketones are present, delay exercise until levels are normalized with insulin administration</li> </ul> </li> <li>• If the activity is aerobic, estimate energy expenditure and determine if insulin or additional carbohydrate will be needed based on peak insulin activity               <ul style="list-style-type: none"> <li>– If insulin dose is to be adjusted for long-duration and/or moderate- to high-intensity activities, try a 50% pre-meal insulin dose reduction 1 h prior to exercise. Dosages can be altered on subsequent exercise days, based on the measured individual response. Insulin should be injected into a site distal to the exercising muscles and into subcutaneous tissue.</li> <li>– If carbohydrate intake is to be increased, try 1 g/kg body weight/hour of moderate- to high-intensity exercise performed during peak insulin activity and less carbohydrate as the time since insulin injection increases. The amount of carbohydrate can be altered on subsequent exercise days, based on the measured individual responses. The total dose of carbohydrate should be divided equally and consumed at 20-min intervals.</li> </ul> </li> <li>• If the exercise is anaerobic or occurring during heat or accompanied by competition stress, an increase in insulin may be needed.</li> <li>• Consider fluid intake to maintain hydration (~250 mL 20 min prior to exercise)</li> </ul>
<b>During exercise</b>	<ul style="list-style-type: none"> <li>• Monitor BG every 30 min</li> <li>• Continue fluid intake (250 mL every 20–30 min)</li> <li>• If required, consume carbohydrate at 20–30-min intervals (see above)</li> </ul>
<b>After exercise</b>	<ul style="list-style-type: none"> <li>• Monitor BG, including overnight, if amount of exercise is not habitual</li> <li>• Consider adjusting insulin therapy to decrease immediate and delayed insulin action</li> <li>• Consider consuming additional slow-acting carbohydrate to protect against post-exercise late-onset hypoglycemia</li> </ul>

BG = blood glucose

with exercise by increasing insulin secretion may make them particularly susceptible to hyperglycemia during some forms of competition (Figure 1C). Some patients may find this hyperglycemic response to stressful competition frustrating, particularly when they are participating in team sports that necessitate breaks in play (e.g. baseball, basketball, hockey). In these instances, periods of physical inactivity coupled with elevations in stress hormones may cause particularly large increases in BG concentration. Again, frequent BG monitoring and small boluses of rapid-acting insulin may be required to recover from these fluctuations.

Individuals may find that training or competing in warm and humid environments also elevates BG levels, likely because of excessive increases in circulating plasma catecholamines, glucagon, cortisol and growth hormone (14).

### **PRACTICAL CONSIDERATIONS FOR PREVENTING HYPOGLYCEMIA OR HYPERGLYCEMIA DURING EXERCISE**

There are a number of strategies available to help stabilize BG concentrations during exercise (Table 2). Unfortunately, it is impossible to give precise guidelines for diet and insulin therapy that will be suitable for all individuals who wish to be physically active. The factors most affecting BG fluctuations during exercise appear to be circulating plasma insulin levels, intensity and duration of exercise and the type of exercise (aerobic vs. anaerobic). Other variables that influence the metabolic responses to exercise, albeit to a lesser extent, include age, gender, level of metabolic control, level of aerobic fitness and prevailing concentrations of the glucose counterregulatory hormones. Although highly specific guidelines are not possible, some general strategies can be applied to help prevent dramatic BG excursions during exercise.

#### **Monitoring**

Frequent self-monitoring of BG and information about the exercise, insulin administration and carbohydrate intake should be recorded so that immediate risks of hypoglycemia and hyperglycemia may be identified. First, it is helpful to have an exercise-training log that documents the type, timing and duration of exercise performed. Second, it is best to have 2 or 3 pre-exercise glucose measurements at 30-min intervals so that directions of change in BG can be determined prior to the activity. Making insulin and/or carbohydrate adjustments based solely on 1 glucose measurement is risky, as the individual is aware of neither the direction nor the rate of this change. Third, individuals should be monitoring BG every 30 min during exercise and every 2 h after the end of exercise for up to 2 readings. Monitoring this frequently prior to, during and after exercise is recommended, particularly if the individual is undertaking a new training regimen or exercise activity. Additional post-exercise measurements may be needed for up to 24 hours after the end of exercise to guard against post-exercise late-onset hypoglycemia. Both patients

and healthcare providers should be aware that although some individuals may claim to know or “feel” their BG levels during exercise, there is no evidence to support this. Exercise may mask many of the symptoms of changes in BG, and individuals tend to overestimate their levels when they are hypoglycemic and underestimate levels when they are hyperglycemic (15). Finally, there should also be documentation of the estimated carbohydrate intake prior to, during and after exercise as well as the location, dose and timing of insulin injection. Self-monitoring of BG and accurate record keeping of all these variables provide feedback for both patients and healthcare professionals that will form the basis for implementing insulin and/or nutritional strategies for subsequent exercise bouts.

Although exercise conditions vary, evidence suggests that BG responses to exercise have some degree of reproducibility (16), making individualized therapeutic recommendations possible. Ideally, for maximal metabolic control and performance, athletes may consider newly developed continuous glucose monitoring devices and insulin pump therapy, since changes in BG concentration can be particularly rapid during exercise and the risk of post-exercise late-onset hypoglycemia is high.

#### **Pre-exercise BG**

If pre-exercise BG readings are  $<5$  mmol/L, not rising and the activity is primarily aerobic (e.g. prolonged running, cycling, soccer), the risk of exercise-associated hypoglycemia is substantial (2). In these cases, it is suggested that exercise not be initiated without the ingestion of at least 15 g of carbohydrate. On the other hand, if fasting BG is  $\geq 14.0$  mmol/L and ketone bodies are present in the urine, patients are generally advised to administer more insulin and delay exercising (2). The knowledge of ketone production may be particularly important since it is common for individuals to exercise post-meal with starting BG levels between 10 and 15 mmol/L, although not ketotic. These individuals may experience dramatic decreases in BG, likely due to rising insulin levels during the activity. Again, it is important to know the direction and rate of change in BG, both prior to and during exercise, so that diet and/or insulin regimens can be modified to prevent hypo- or hyperglycemia. For example, a BG level of 5.5 mmol/L may be considered safe for exercise if the previous measurement was 5 mmol/L, whereas the same reading of 5.5 mmol/L indicates a potentially dangerous imbalance of glucose production and utilization if the preceding measurement was 7 mmol/L. Clearly, in the latter situation, carbohydrate intake would be necessary to prevent hypoglycemia. Some individuals may wish to allow BG levels to be slightly higher just prior to and during exercise as a further safeguard against hypoglycemia.

#### **Insulin adjustments for exercise**

It is clear that reducing insulin dosage in anticipation of exercise decreases the risk of hypoglycemia and is the best way to

mimic the normal physiological response to exercise. This is particularly important if the exercise is performed postprandially when insulin levels are generally the highest. Individuals treated with intensive insulin therapy can become hypoglycemic within 45 min of strenuous exercise performed 2 h after a standard meal and their usual insulin dose (8,9). A 30 to 50% reduction in bolus insulin delivery reduces the likelihood of developing hypoglycemia in these same patients (9). It is important to note that it may not be necessary to reduce the insulin dose if the start of exercise occurs several hours after a meal, when insulin levels are low.

For prolonged exercise (i.e.  $\geq 90$  min), a greater reduction in insulin dosage may be needed. For example, it has been shown that cross-country skiers with type 1 diabetes are able to exercise for several hours without becoming hypoglycemic if the insulin dose is reduced by 80% compared with only 90 min if the dose is reduced by only 50% (17). In general, higher aerobic exercise intensities that last for prolonged periods elicit a greater drop in BG and a greater need for reduced insulin dosage (17-20).

Injecting insulin in a subcutaneous depot well away from an exercising muscle may minimize the risk of hypoglycemia to some degree (21), although the effects of exercise on the kinetics of rapid-acting insulin analogues are not well understood. Individuals should be cautious about injecting insulin into the muscle tissue itself, since it may dramatically increase the insulin absorption rate (22). Further considerations for quantifying insulin pump adjustments during exercise are provided in the companion paper (23).

### *Carbohydrate adjustments for exercise*

Because exercise is often spontaneous, it is not always possible to anticipate the need to decrease the insulin dosage. In addition, some patients find that lowering their insulin dose at the meal prior to exercise causes an initial hyperglycemic response that impairs their exercise performance (see "BG levels and exercise performance" below). In these instances, carbohydrate ingestion is a viable option, and possibly the only alternative to maintain BG levels during exercise (24). Recommendations for the amounts and forms of carbohydrate intake during competition are discussed below.

## **MACRONUTRIENT ADJUSTMENTS FOR TRAINING AND COMPETITION**

For the endurance athlete, nutritional strategies to optimize carbohydrate stores in muscle and liver are essential to optimize training and performance. Nutritional strategies can be divided into 4 categories: 1) daily caloric intake during training; 2) caloric intake hours prior to exercise; 3) nutrient intake during exercise; and 4) intake following exercise.

### **Daily macronutrient intake**

The recommended distribution of macronutrients for the athlete with diabetes is 55 to 60% of total energy intake as

carbohydrate: 25 to 30% as fat, and 12 to 15% as protein (25). It is important to note that intense daily training will acutely reduce the body's stores of carbohydrate. If carbohydrate stores are not replenished after each exercise session, endurance capacity will be impaired and the individual may be at increased risk of hypoglycemia. It is generally recommended that the majority of carbohydrates be complex (e.g. whole grains, beans) to limit post-meal hyperglycemia and elevated needs for insulin. Endurance athletes should consume approximately 8 to 10 g carbohydrate/kg body weight/day (25). Although protein is not a major fuel source oxidized during exercise, adequate intake is essential to allow for muscle regeneration and hypertrophy during training. It is generally recommended that 0.8 to 1 g protein/kg body weight is sufficient for recreational athletes, while those involved in competition and heavy training may need up to 1.7 g protein/kg body weight/day (26).

### **Precompetition nutrition**

During the hours prior to competition, it is critical to maintain BG levels in the near-normal range (4 to 7 mmol/L) to limit the risks of dehydration, extreme lethargy, hypoglycemia and associated autonomic counterregulatory failure. This may be accomplished by frequent BG monitoring and refinements in either insulin and/or carbohydrate intake. Individuals may need small increases in insulin prior to competition to counter the stress-associated increases in glucose counterregulatory hormones. Ideally, a meal 3 to 4 h prior to competition is desirable since it will maximize energy stores and should not cause gastric upset. If possible, a carbohydrate beverage containing 1 to 2 g carbohydrate/kg body weight should be consumed 1 h prior to competition to maximize pre-exercise glycogen stores, providing energy for oxidation and fluid for maintenance of adequate hydration. Generally, 6% carbohydrate-electrolyte beverages composed of simple sugars (i.e. sucrose, fructose) are best, since they have optimal fluid and carbohydrate absorption rates compared with other more concentrated beverages such as juice or carbonated drinks that may delay gastric absorption and cause stomach upset. Water may also be suitable if pre-exercise BG concentrations are elevated (i.e.  $>10$  mmol/L). Although many endurance athletes train or compete after an overnight fast, perhaps to limit any gastric upset that may occur with eating during periods of stress, this practice in a patient with type 1 diabetes will reduce hepatic glycogen storage and may predispose the individual to exercise-associated hypoglycemia and premature fatigue.

### **During competition**

During prolonged competitions, particularly if the pre-exercise insulin dose is not reduced significantly, carbohydrate ingestion is essential for maintaining BG concentrations. Carbohydrate intake delays fatigue and provides fuel for oxidation by the working muscles. The amount of carbohydrate

consumed depends on a number of variables including the exercise intensity, gender, age, and the timing and dose of the last insulin injection. As a general rule, 1 to 1.5 g carbohydrate/kg body weight/h should be consumed during exercise performed during peak insulin action (2). This amount may be considerably lower if exercise is performed when circulating insulin levels are low. For example, 60 min of moderate-intensity aerobic exercise performed 1 h after insulin administration was shown to require  $\sim 1$  g/kg body weight of carbohydrate to prevent hypoglycemia, while these same individuals needed only  $\sim 0.5$  g/kg and only 0.25 g/kg when the same exercise was performed 2.5 and 4 h after insulin administration, respectively (27). It is important to note that these guidelines for carbohydrate intake are in addition to any carbohydrates ingested in the meal prior to exercise. For active children with type 1 diabetes, carbohydrate intake matched to total carbohydrate utilization ( $\sim 1.5$  g carbohydrate/kg body weight/h of exercise) limits hypoglycemia during moderate-intensity exercise (8). Consuming extra carbohydrate, rather than adjusting insulin, is advantageous since a child's activities are often spontaneous and of unpredictable duration and intensity. In these instances, tables of "exercise exchanges" can be used to prescribe carbohydrate intake for a variety of activities and sports performed during peak insulin times (15, 24). As discussed in the companion paper by Perkins (23), insulin pump therapy provides the option of spontaneous insulin dose changes for exercise. In addition, estimates of insulin adjustments based on energy expenditures can be calculated and tested. Nonetheless, the individual with type 1 diabetes should be encouraged to consume extra carbohydrate for training and competition, since high ingestion rates have been associated with improved performance by both maintaining high rates of glucose oxidation and by sparing hepatic glycogen stores (2).

During exercise, it is imperative to prevent dehydration, which is associated with impaired performance, muscle cramps, hyperglycemia and heat stroke, the latter of which may even result in death. Ideally, fluid intake should closely match sweating rate during exercise. Sweating rates range from 0.75 to 1 L/h depending on the exercise intensity, ambient temperature and relative humidity. On average, fluid intake should be approximately 250 mL every 20 min of exercise and the first drink should precede exercise by 20 min (26). Again, carbohydrate-electrolyte beverages may be best for activities lasting  $>60$  min, since they contain both requirements of carbohydrate and fluid.

#### *Nutrient intake following exercise*

Post-exercise, carbohydrate intake is necessary to help replenish liver and muscle glycogen stores. During this period, which may last up to 12 to 24 h, insulin sensitivity remains elevated and there is a high risk of hypoglycemia in patients with type 1 diabetes (10). To quickly replenish muscle glycogen stores, perhaps for subsequent competition, carbohydrate intake

should be started within the first few hours after the end of exercise. For patients who tend to experience post-exercise late-onset hypoglycemia during the night, a complex carbohydrate (e.g. uncooked corn starch) or a mixed snack containing fat and protein may be particularly beneficial at bedtime (28).

#### **BG LEVELS AND ATHLETIC PERFORMANCE**

Surprisingly, there are few published studies on BG concentrations and athletic performance in individuals with type 1 diabetes. Clearly, in athletes without diabetes, hypoglycemia dramatically lowers exercise performance, increases the rating of perceived exertion (RPE) and causes premature fatigue during prolonged exercise. Likely, the same is true for athletes with type 1 diabetes who become hypoglycemic more frequently. Hypoglycemia and the deterioration of exercise performance can be dramatically reversed with carbohydrate ingestion during exercise. For example, in active adolescents with type 1 diabetes, hypoglycemia reduces exercise endurance and the consumption of carbohydrate that attenuates the drop in BG improves their capacity (8).

Hyperglycemia likely impairs performance in individuals with type 1 diabetes for 2 reasons. First, if hyperglycemia exists prior to exercise, the individual may already be dehydrated. Evidence suggests that even a 1% decrease in body mass because of dehydration noticeably impairs performance (26). Second, hyperglycemia has been associated with the reduced ability to secrete beta-endorphins during exercise (29) and is associated with increases in RPE for leg effort (29) and whole-body effort (30).

Interestingly, poor metabolic control, as measured by A1C levels, is associated with poor maximal aerobic capacity in both patients with type 1 (31) and type 2 (32) diabetes. As such, it may be particularly important to maintain good metabolic control during training and competition to maximize aerobic performance.

#### **CONCLUSION**

Special considerations are needed for the physically active individual with diabetes. Although regular exercise is beneficial for all patients, exercise training and competition can cause major disturbances in BG control. Both insulin and nutritional adjustments are often required because of the stress associated with activity and the profound changes in insulin sensitivity that accompany exercise. Reductions in insulin and increases in carbohydrate and fluid intake allow individuals to compete and excel during vigorous exercise.

#### **ACKNOWLEDGEMENTS**

Dr. Riddell is grateful to K. Iscoe for her assistance in preparing this manuscript.

#### **AUTHOR DISCLOSURE**

No duality of interest declared.

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