Competitive athletes with diabetes present a significant challenge to themselves and the medical staff who care for them on a daily basis. The physiological demands induced by intense exercise and training, nutritional needs and varied meal timing to support and enhance training regimens and competition, and the stress of competition are just a few factors that athletes with diabetes endure during their daily management.

They are also at risk for both acute hypoglycemia or ketoacidosis and chronic complications—microvascular and macrovascular disease. Therefore, a thorough understanding of the unique metabolic demands is critical to their systematic, thorough management. This article provides a general overview of exercise and nutritional considerations and a detailed review of the management of diabetic athletes.
physicians, trainers, and coaches to develop effective pre-exercise management plans to avoid the harmful consequences associated with poor glycemic control.

The American Diabetes Association (ADA) has classified DM into 2 general forms; type 1 diabetes (T1D) and type 2 diabetes (T2D). There are an estimated 16 million people living in the United States with DM, of whom 10% have T1D and 80% to 90% have T2D. The prevalence of T2D, which is highest among African Americans, is increasing at an alarming rate both in the United States of America and worldwide. Unfortunately, the incidence of this disease is also increasing among children and adolescents.

T1D results from a highly specific immune-mediated destruction of pancreatic β cells, leading to chronic hyperglycemia. Individuals with T1D are usually diagnosed in adolescence and must rely on the injection of exogenous insulin for survival. Interestingly, in 1950, a diagnosis of T1D was associated with a 1 in 5 risk of mortality within 10 years. However, due to several advances in insulin therapy and methods for self-blood glucose monitoring, individuals with T1D now live near-normal life spans and are capable of competing at the highest levels of sport (eg, Olympic games) and in the most extreme endurance events (eg, Ironman triathlon). Yet effective management is a constant challenge, because insulin therapy is an “imperfect science.”

Conversely, T2D correlates strongly with obesity and unhealthy lifestyle behaviors (poor diet, smoking, physical inactivity, etc), and diagnosis is often delayed. The hallmark characteristic of T2D is insulin resistance, which is characterized by a defect in the ability of skeletal muscle to respond to insulin-mediated glucose uptake. Additionally, with advancing disease, pancreatic β-cell dysfunction results in increasingly less insulin secretion.

Although regular exercise is generally thought to be most beneficial in the long-term management of T2D, it is rare to encounter young, competitive athletes with T2D. This is because most individuals are usually diagnosed with the T2D later in adulthood (~age 40 years), yet, alarmingly maintain sedentary lifestyles. However, with an increasing emphasis being placed on the importance of physical activity, it is probable that a greater percentage of individuals with T2D will participate in recreational sports as “masters” athletes.

T2D is also rapidly increasing at an alarming rate in adolescents. Early therapeutic intervention is imperative, and their teachers and parents should encourage participation in physical activity, which may positively influence compliance with exercise programs in adulthood. Children with T2D may participate in sports as part of their usual school curriculum or may be motivated by external factors, such as the desire to “fit-in” socially.

This section first reviews substrate regulation during moderate- and high-intensity exercise under normal circumstances. Subsequently, exercise considerations in athletes with T1D and T2D are discussed. A majority of the discussion in this section focuses on exercise considerations in individuals with T1D, since it is more prevalent in competitive athletes; however, implications of exercise in T2D are also reviewed.

Overview of Normal Glucoregulation During Exercise

Metabolic responses to exercise are determined primarily by the intensity (ie, moderate or intense), duration (ie, short, prolonged, or intermittent), and environmental conditions (ie, hot, humid, time of day). During moderate-intensity exercise (40%–59% of maximal oxygen consumption [Vo2 max] or 55%–69% of maximum heart rate), the fuel for muscular contraction is obtained almost exclusively from aerobic metabolism—by using a mixture of carbohydrate (CHO) from muscle glycogen stores and circulating free fatty acids (FFA) as fuel. Most endurance sports
are performed within the moderate-intensity domain (eg, long-distance running and cycling).

The transition from rest to moderate-intensity exercise is characterized by an increase in sympathetic nervous activity, which aids in increasing endogenous glucose production by the liver (gluconeogenesis) and stimulates the release of FFA from adipose tissue (lipolysis). In addition, $\alpha$-adrenergic stimulation of pancreatic islet cells inhibits insulin secretion, which in turn signals the release of glucagon from pancreatic $\alpha$ cells. Since high insulin levels normally inhibit glycogenolysis and hepatic gluconeogenesis, a decrease in insulin effectively primes the liver to the effects of glucagon. This mechanism precisely matches glucose utilization by exercising muscle tissue for hepatic glucose production and is the key factor maintaining blood glucose within a narrow range during moderate-intensity exercise (80–100 mg/dL). The decrease in insulin is a critical mechanism to balance glucose utilization at the muscle, which may reach ~3 mg/kg/min, and prevent hypoglycemia and maintain the ability to exercise effectively.

It is important to note that hepatic sources of glucose, derived from liver glycogen stores or produced via gluconeogenesis, are able to enter muscle cells despite low insulin concentrations for mainly 2 reasons: (1) exercise independently increases glucose transport via a pathway independent of insulin-stimulated glucose uptake, and (2) muscle contraction results in the enhanced recruitment of capillaries, which augments the surface area available for nutrient exchange. It is well known that muscle contraction increases glucose transport even in the complete absence of insulin and that the synergistic effects of insulin and muscle contraction are additive to enhanced glucose transport. Sustained muscular contraction during exercise also provides an improvement in insulin sensitivity, which may extend for several hours, lasting into the postexercise period.

High-intensity exercise, 85% to 100% Vo$_2$ max or greater than 90% maximal heart rate, sustained for 10 to 30 minutes or intermittent bouts of 3 to 5 minutes is common in team-oriented sports, such as lacrosse, football, hockey, soccer, track and field, and swimming. Exercise to Vo$_2$ max is sustained primarily by aerobic metabolism, including oxidative phosphorylation and to a limited extent, beta oxidation. In contrast, high-intensity, supramaximal-effort (>Vo$_2$ max) activities sustained for only 3 to 30 seconds use the anaerobic energy system (glycolysis and the Adenosine triphosphate-phosphocreatine [ATP-PCr system]). In either situation, high-intensity exercise is highly dependent on glucose as a fuel, derived from either hepatic or muscle glycogenolysis. In addition, exercise at high intensity is characterized by lactate accumulation and a marked increase in catecholamine (norepinephrine and epinephrine) concentrations, by up to 14- to 18-folds above basal levels. This is in sharp contrast to moderate-intensity exercise in which catecholamine levels increase by only 2- to 4-folds above baseline.

During high-intensity exercise, hepatic glucose production exceeds muscle glucose utilization, resulting in slight hyperglycemia. This is largely due to the fact that norepinephrine and, to a lesser extent, epinephrine act as powerful stimulators of muscle and liver glycogenolysis. Norepinephrine reaches the liver as a “spillover” from other tissues or by direct sympathetic nerve stimulation, which results in increased hepatic glucose production. Moreover, akin to moderate-intensity exercise, insulin levels remain very close to basal levels despite the fact that glucose production exceeds utilization.

In the immediate postexercise period, insulin levels rapidly increase both in response to high blood glucose levels and following removal of circulating catecholamines. The next 20 to 60 minutes are characterized by a state of hyperglycemia
and hyperinsulinemia, which creates an environment favorable for glycogen replenishment in anticipation of future exercise.\(^9\)

**Exercise Considerations in Athletes with Type 1 Diabetes**

Compared to healthy peers, athletes with T1D experience nearly all the same health-related benefits from exercise.\(^2\) These include improvements in health-related quality of life, reduction in blood pressure, improvement in lipid abnormalities (i.e., high low-density lipoprotein cholesterol, triglycerides),\(^16\) increased insulin sensitivity, decreased insulin requirements, lower hemoglobin A\(_{1c}\) (HbA\(_{1c}\)) levels,\(^17\) improved endothelial function,\(^18\) and improvement in cardiorespiratory fitness (Vo\(_2\) max), which has repeatedly been shown to be a powerful predictor of future cardiovascular disease (CVD).\(^19\)

Despite these benefits, without proper, proactive education and precautions, exercise may predispose the diabetic athlete to hypoglycemia—the most common adverse event associated with insulin therapy.\(^10\) For good reasons, individuals with T1D should be encouraged to maintain “tight” glucose control to lower HbA\(_{1c}\) levels to a measure that strongly correlates with a lower risk of long-term complications, including peripheral neuropathy, retinopathy, nephropathy, and CVD. It is important to note, however, that the Diabetes Complications and Controls Trial (DCCT) reported a significant 3-fold increase in the incidence of hypoglycemia in individuals with strict glycemic control on insulin therapy.\(^20\) In addition, it is hard to simulate “precompetition” anxiety and excitement, which tends to increase blood glucose above and beyond levels noted and treated during “practice”. Finally, exercise in hot and humid environments can increase counter-regulatory responses to high-intensity exercise as well as alter the rate of insulin absorption.\(^21\) Thus, athletes with T1D need to find a suitable balance with their exercise regimens, nutritional adequacy, and insulin dosing to avoid either hypo- or hyperglycemia.

**Exercise Considerations in Athletes with Type 2 Diabetes**

Exercise training in T2D results in increased translocation of muscle glucose transporter-4 receptors, an increased capacity for insulin-stimulated glucose uptake, and decreased insulin resistance. The defects in insulin signaling and/or secretion cannot be fully reversed, yet increased physical activity as a routine “lifestyle” change is a major intervention in the management of T2D.\(^1\)

To further highlight the importance of “lifestyle” change in the treatment of T2D, the Diabetes Prevention Program (DPP) trial concluded in 2002 that after ~3 years, “lifestyle” interventions reduced the incidence of diabetes by 58% compared with only 31% in a group treated with the antidiabetic drug metformin.\(^22\) Currently, the ADA recommends that all individuals with T2D engage in at least 150 min/wk of moderate to vigorous-intensity exercise.\(^3\)

Other important exercise considerations in T2D, especially in those being treated for concomitant hypertension, include the potential for negative electrolyte homeostasis (diuretics), reduction of exercise capacity and athletic performance during high-intensity exercise (β-blockers), and increased susceptibility to hypoglycemia (aspirin or angiotensin-converting enzyme [ACE] inhibitors). The mechanisms by which aspirin or ACE inhibitors may induce hypoglycemia is poorly understood.\(^3\)

Unfortunately, the majority of individuals with T2D are not likely to engage in physical activity,\(^1\) yet the benefits of exercise far outweigh risks of either hypoglycemia or hyperglycemia and should be a major component of any therapeutic intervention.
THE DIABETIC ATHLETE—NUTRITIONAL CONSIDERATIONS

Healthy Eating for Peak Performance—Balance of Nutrients

In order to exercise and compete at peak levels of performance, diabetic athletes have unique nutritional needs. Optimal blood glucose management to prevent hypo- or hyperglycemia is a daunting task and requires a thorough understanding of the current recommendations for caloric and fluid intake before, during, and after exercise. In addition to balanced and timely CHO intake, diabetic diets should contain optimal amounts of proteins and fats that provide calories and have essential functions for glycemic control and health.

The recommended balance of these energy-yielding nutrients for athletes does not differ significantly from recommendations for the general population. However, additional calories and fluids may be required for diabetic athletes and depends upon exercise intensity, total energy expenditure, type of exercise and training program, duration of exercise, gender, and environmental circumstances. Calorie requirements can range from 2,000 to more than 6,000 cal/d. Therefore, a thorough nutritional assessment of “usual” food intake followed by monitoring of weight, appetite, and blood glucose levels is the best way to evaluate adequacy of caloric intake.23

A joint position statement by the ADA, Dietitians for Canada, and the American College of Sports Medicine has recommended the following general energy requirements for competitive athletes.24

1. CHO consumption range of 6 to 10 g/kg body weight is required to maintain blood glucose levels during and after exercise and for replacement of glycogen stores.
2. Protein consumption range of 1.2 to 1.7 g/kg body weight. Generally, this level of protein can be obtained from the diet. Protein is needed for tissue repair and muscle growth.
   a. For endurance athletes, 1.2 to 1.4 g/kg body weight.
   b. For strength-trained athletes, 1.6 to 1.7 g/kg body weight.
3. Fat consumption range of 20% to 25% of total daily calories. Fat provides needed calories as well as fat-soluble vitamins, and essential fatty acids. No performance benefits have been found in reducing fat intake to less than 15% of daily calories.

Healthy Eating for Peak Performance—Timing and Types of Nutrients

Diabetic athletes who participate in regular endurance exercise should consume ~60% CHO daily and coordinate food intake with “timing” of exercise and insulin dosing. This approach is critical for optimal glycemic control, to maintain muscle and liver glycogen stores, prevent fatigue, optimize exercise performance, and prevent complications.

Carbohydrate intake before exercise

General principles and recommendations for timing and choices for food include the following:

1. Eating an easily digested meal in the amount of 200 to 350 g (or 4 g/kg of body weight) of CHO 3 to 6 hours before an exercise event has been shown to enhance performance.25,26
2. Although 60 to 90 g of CHO are recommended per meal in most adults with diabetes, athletes with diabetes who “CHO load” (200–350 g per meal) to increase glycogen stores before long athletic events should monitor blood glucose levels regularly and adjust insulin doses accordingly.
3. The recommended pre-event CHO intake is approximately 1 g of CHO/kg of body weight 1 hour before the exercise. Low-fat CHO foods, such as crackers, muffins, toast, fruit, and yogurt, instead of sugary sweets are good choices.

4. If the exercise is of short duration (<45 minutes), a pre-exercise snack of ~15 g of CHO eaten 15 to 30 minutes before the event has been reported to be an adequate amount.28

5. Foods such as pancakes, potatoes, bread, and fruit are appropriate choices.

**Carbohydrate intake during exercise**

CHO are needed during exercise of long duration to maintain CHO oxidation and to replenish muscle glycogen stores on a regular basis.29 As stated previously, the recommended percentage CHO consumption for an athlete with diabetes, either during training or an event, is ~60%, which equates to 6 to 10 g of CHO/kg body weight, depending on the duration of the exercise period. Because average CHO intake is generally 4 to 5 g/kg body weight per day (~45% of calories), increasing CHO may be an additional challenge for the exercising athlete.23 Recommendations include30 the following:

1. For exercise periods of 1 h/d, 5 to 6 g CHO/kg body weight.
2. For exercise periods more than 2 h/d, 8 g CHO/kg body weight may be needed.
3. For endurance activities, this may be increased to 10 g CHO/kg body weight.
4. During prolonged (>45–60 minutes) or intense exercise (>80% maximal heart rate), an intake of 15 g CHO every 30 to 60 minutes of activity is a safe starting guideline.23

5. Solid or liquid forms of CHO may be consumed. Each form has its distinct advantages. Liquids provide fluid for hydration, whereas solids may prevent hunger. For exercise lasting more than 60 to 90 minutes, a liquid CHO form is most recommended, because it is more practical and contributes to adequate hydration.31

**Carbohydrate intake after exercise**

1. Consuming CHO immediately after exercise as opposed to waiting for a period of time has been shown to replace CHO stores more efficiently.
2. Intake of 1.5 g of CHO/kg body weight within 30 minutes after an extended exercise session (lasting >90 minutes) and intake of an additional 1.5 g of CHO/kg body weight 1 to 2 hours later will replete glycogen to pre-exercise levels and will reduce risk of postexercise hypoglycemia.32

3. Blood glucose levels should be monitored at 1- or 2-hour intervals to assess the response to exercise and to make the necessary adjustments in insulin and food intake.

**Adjustment of carbohydrate and insulin**

A reduction in the dose of insulin before exercise may be required. A reduction of short-acting insulin of 30% to 50% has been reported to decrease the risk of hypoglycemia.33 Another method is to decrease the insulin used during exercise by 10% of the total daily insulin dose.23 This approach is discussed in more detail later in the article.

**Hypoglycemia—nutritional prevention**

In order to prevent the most common complication of exercise in diabetic athletes, the amount of CHO, fat, and protein consumed before, during, and after exercise should be determined by blood glucose levels and the proposed duration of exercise.

1. Planned pre-exercise snacks should be high in CHO, low in fat, and moderate in protein content.
2. The postexercise snack should consist of CHO and protein.
3. Examples of pre- or postexercise snack choices include the following:
   a. CHO: whole-grain bread, breadsticks, crackers, cereal, fig bars, oatmeal-raisin cookies, granola bar, fruit, juice, yogurt, and milk.
   b. Protein: 1 to 2 oz lean meat, nut butter, and cheese.

4. Diabetic athletes should also have fast-acting CHO sources readily available. Fifteen grams of CHO will raise blood glucose 30 to 50 points within a 15- to 30-minute timeframe. Convenient fast-acting CHO sources include glucose gels, 3 to 4 glucose tablets, 2 tablespoons of raisins, 1/2 cup fruit juice, or 1 cup low-fat or nonfat milk.

**Nutritional Myths**

Athletes often believe that nutritional supplements improve performance. However, adequate consumption from a variety of food sources precludes the need for vitamin and mineral supplements. The 2005 Dietary Guidelines for Healthy Americans emphasize that supplements may be useful when they fill a specific identified “nutrient-gap” that either cannot or is not being met by an individual’s intake of food. Nutrient supplements are not a substitute for a healthy diet. In addition, any supplements or ergogenic aids being considered for use should be evaluated by a trained health care professional. Stringent regulations on the nutritional supplement industry are sparse; therefore, caution is advised on the use of supplements.

Consuming large amounts of protein and protein supplements is also a common practice of athletes. Studies have found that large amounts of protein (>2.4 g/kg/d) may place undue stress on the kidneys. In the athlete with diabetes, this may compound the risk of long-term renal complications.

**Fluids and Electrolytes**

It is a well-established fact that the thirst mechanism is blunted with exercise. Therefore, in order to prevent complications secondary to dehydration, diabetic athletes should monitor and consume adequate fluid before, during, and after exercise. Adequate hydration helps prevent a rise in “core” body temperature and reduces heat-induced stress of the cardiovascular system. Cool, plain water is recommended as the beverage of choice.

Recommendations for fluid intake for persons with diabetes engaging in physical activity include 3 cups of water ~2 hours before an event. Another 1 to 2 cups of water should be consumed 10 to 15 minutes before the beginning of the event. The most effective method to monitor fluid needs during exercise is to note weight changes from fluid losses during exercise. Approximately 2 cups of fluid should be consumed for every pound lost. Persons who are sedentary lose approximately a quart of water daily from sweating, whereas athletes undergoing strenuous exercise may lose more than or equal to 2 qt. Specifically, during exercise, the athlete should continue drinking small amounts (1/2–1 cup) of fluid at 10- to 20-minute intervals. This is necessary to ensure replacement of body fluid lost by sweating and to maintain optimal blood volume. Furthermore, consumption of small amounts of fluids at frequent intervals can reduce abdominal discomfort and “bloating.”

For exercise sessions greater than or equal to 60 minutes, beverages that contain at least 8% CHO (eg, sports drinks or diluted fruit juices of 50% dilution) are the best source of “replacement” beverages, because they replace fluid and calories. Drinks with a concentration of sugars greater than 10%, via increased osmosis, can produce undesirable gastrointestinal symptoms, such as cramps, nausea, diarrhea, or bloating. Fruit juices and regular soft drinks contain about 12% CHO and should be diluted with an equal amount of water prior to consumption. Ounce for ounce, common sports drinks are a better choice.
drinks such as Gatorade and PowerADE contain less CHO than soft drinks or fruit juices and have a lower osmolality, which may make them more desirable for consumption during exercise.

Because individuals differ in their metabolic responses to exercise, dictated by varying levels of emotional and physical stress of competition, we recommend that in the postexercise period, rehydration be guided by estimation of body weight and blood glucose levels. Diabetics should work with their athletic trainers, nutritionists, and physicians to determine individualized needs for either plain water or an additional CHO-containing beverage to promote euhydration and normoglycemia.23

THE DIABETIC ATHLETE—MANAGEMENT

Education

Appropriate glucose management in diabetic athletes is dependent on both the athlete and the care provider having a firm understanding of the pathophysiology of diabetes and its nuances with respect to athletic participation. The cornerstone of management for T1D athletes is the prevention of both hypo- and hyperglycemia while maintaining adequate energy balance for exercise performance. Exercise for T1D is a nutritional challenge38,39 because of the delicate balance between insulin use and caloric intake. In contrast, T2D should be viewed as an energy-excess syndrome in which skeletal muscles and the liver become progressively insulin resistant. As skeletal muscle accounts for the major site of insulin-stimulated glucose utilization in humans, endurance or combined endurance and resistance exercise training may improve overall glucose tolerance and/or insulin sensitivity40,41 but may also create challenges in optimal glucose control.

All diabetic athletes should be educated about the importance of establishing a daily pattern of consistency for all aspects of their diabetes management. It is ideal to have the athlete begin a routine of insulin and/or oral medication administration, consistent caloric intake, regimented exercise program every day, and frequent monitoring of blood glucose levels. Each of these steps will not only aid in maximizing blood glucose control but also help the diabetic athlete understand how best to manage the diabetes in the face of high-level exercise. Each athlete is unique and will require individualized adjustments until an optimal routine is established.

In the setting of scholastic athletics, children may be prone to greater variability in blood glucose levels, and adolescents demonstrate hormonal changes, which can contribute to greater difficulty in controlling blood glucose levels as well. In this special group of diabetic athletes, it is vital to ensure that parents, coaches, teachers, and other adults understand the importance of scheduled meals, snacks, and adequate fluids, as well as appreciating the features and management of hypoglycemia.42,43

Blood Glucose Control

Type 1 diabetes

In general, T1D athletes exhibiting poor metabolic control (HgA1c >9%) should refrain from moderate- or higher-level exercise until adequate blood sugar control has been obtained. This is prudent to avoid the risk of exacerbating hyperglycemia and to minimize the risk of progression to frank diabetic ketoacidosis (DKA).44 The duration and intensity of exercise will determine the specific modifications that need to be made in the treatment regimen.

Adjustments in both dietary intake and insulin dosing are essential for optimal performance and prevention of deleterious glucose fluctuations. Waiting 60 to 90 minutes after a meal before exercising and monitoring blood glucose both during
and after exercise/sport are important baseline management measures. With respect
to diet, CHO-rich, low-glycemic-index (see later section) meals should be consumed 1
to 3 hours before exercise.38,45 Immediately before and during an exercise bout,
consumption of additional CHO (17 g at initiation and 17 g every 15 minutes for 60
minutes for exercise at 65% VO2 max) is beneficial in maintaining glucose levels during
exercise and particularly after exercise in patients with both T1D and T2D.46

The consumption of a low-glycemic-index diet improves metabolic regulation,47
because these foods require less insulin for optimal glucose utilization. Such foods
give a low and slow glucose rise when consumed and include raw cornstarch, non-
starchy vegetables, fruits, nuts, milk, and fructose and lactose sugars. Characteristic
high-glycemic-index foods that give rise to rapid and high-glucose responses include
white bread and glucose sugars.48 Provided that the diet contains enough CHO (at
least 35% of total calories) to maintain normal glycogen levels, low-calorie diets can
be used in this population without affecting exercise tolerance.49

Comfort with insulin adjustments with exercise is essential for athletic success and
prevention of acute complications. If pre-exercise blood glucose is 100 to 250 mg/dL,
it is generally safe to begin exercising.50 American Diabetes Association guidelines42
for regulating the glycemic response to exercise include the following:

1. Metabolic control before exercise—avoid exercise if fasting glucose is greater than
250 mg/dL and ketosis is present; use caution if glucose is greater than 300 mg/dL
with no ketosis; ingest added CHO if glucose levels are less than 100 mg/dL.
2. Blood glucose monitoring before and after exercise—identify when changes in
insulin or food intake are necessary, and learn the glycemic response to different
exercise conditions.
3. Food intake—consume added CHO to avoid hypoglycemia with exercise; CHO-
rich foods should be readily available during and after exercise.

T1D athletes should avoid exercise during peak insulin times, and the dose of short-
or rapid-acting insulin should be decreased by 30% to 50% if given before exercise.42
Near euglycemia has been shown to be obtainable in T1D athletes during exercise
even with reductions of 70% to 90% in insulin dose.51 In fact, doses of insulin may
also need to be further reduced 10% to 30% as an athlete becomes more fit.52 For
a morning workout, the dose of the athlete’s short-acting insulin (regular insulin, onset
1–2 hours, peak 2–4 hours) should be reduced. For an afternoon workout or compe-
tition, the dose of the intermediate-acting insulin should be reduced (NPH insulin,
onset 1–3 hours, peak 4–10 hours). Long-acting, peakless insulin such as insulin glar-
gine should have the total dose reduced as above. Insulin absorption is more rapid
and less predictable when injected into the leg before exercise.53 The abdomen is
the preferred site for athletes because of its ease of access during meals and more
predictable absorption time.54

Special note should be made of high-intensity exercise as would be common with
competitive sports. Such activity may elevate blood sugar levels in diabetics, but this
response is generally temporary and results from a number of hormonal factors. Blood
glucose levels usually fall for several hours after exercise, so such a transient increase
after high-intensity workouts should not be treated with insulin.55

**Insulin pumps**

Athletes using insulin pumps should precede intense exercise by reducing the action
of the pump by 50% about 1 hour before activity.56 For lower-intensity activity, the
standard basal rate may be maintained with a small reduction in the premeal bolus.
If the insulin pump must be removed before contact/collision sport, it should be
removed 30 minutes prior to exercise to compensate for the persistent insulin effect after pump removal. For prolonged activity more than 1 hour, small boluses may be needed to prevent a hypoinsulinemic state. Boluses should be given every hour, and the amount of insulin given should represent about 50% of the usual hourly basal rate.  

**Type 2 diabetes**

For T2D athletes, the primary goals for management include not only preserved performance and prevention of hypo- and hyperglycemia but also improved insulin sensitivity and uptake of glucose in skeletal muscle, with a concomitant improvement in postprandial blood glucose. The major metabolic problem for active T2D patients is a reduced capacity to store excess glucose as muscle glycogen. Consequently, the most important effect of exercise for improving glucose regulation is the lowered glycogen storage in skeletal muscles. Ideally, if energy stores remain low, the calories consumed in the next meal can be stored as glycogen rather than contributing to hyperglycemia.

T2D athletes on diet therapy alone should be able to exercise with no further caution than individuals with normal glucose tolerance provided no major vascular complications are present. No pre-exercise CHO intake is necessary. In general, far lower CHO intake during exercise is required of T2D athletes, as these patients have a lower rate of glucose metabolism and a much lower risk of hypoglycemia with exercise training relative to T1D patients. CHO intake for these athletes should only be undertaken during exercise to prevent hypoglycemia, particularly as weight reduction is desirable, and limited CHO intake will be beneficial to this goal. The major adjustment for T2D athletes involves adjustment of oral hypoglycemic therapy as dictated by their glucose values. These medications are reviewed later in this article.

**Postexercise**

After exercise, glycogen resynthesis and storage occur in skeletal muscles and require insulin to be most efficient. This process is essential to allow the athlete to physically prepare for the next bout of high-level exertion. Repletion is faster after ingestion of high-glycemic-index foods such as glucose and sucrose rather than fructose. After training, T1D athletes should immediately consume a high-CHO, high-glycemic-index meal with insulin to refill glycogen stores. Postexercise consumption should consist of 30 to 40 g of CHO for every 30 minutes of intense exercise as part of the 500 to 600 g of CHO required daily in typical endurance athletes. If adequate caloric intake does not occur, hypoglycemia may arise after exercise because of the increased insulin sensitivity of exercising skeletal muscles. Restoration of glycogen stores normalizes insulin sensitivity. For extensive and/or late afternoon/evening exercise, blood glucose levels should be monitored once or twice during the night.

**Children**

Current ADA recommendations for the management of T1D in active children include the following:

1. Eat 15 to 30 minutes before vigorous activity or activity of longer than 30 minutes duration (15 g of CHO are generally adequate for 30 minutes of moderate-intensity activity).
2. Always carry a CHO source.
3. Decrease insulin typically by 10% to 20% before sport activity, with adjustments over time based on glucose values and intensity of training.
Medications

Medication management of the diabetic athlete requires a delicate balance between maintenance of adequate glucose levels to allow for sport activity while minimizing the likelihood of hypoglycemia. Insulin and medications that stimulate insulin production increase the risk for hypoglycemia and must be used with caution in this population. The following subsection is a brief review of available medications for use in active diabetics.

**Insulin**

Insulin may be used by both T1D and T2D athletes. Insulin enhances the peripheral uptake of glucose primarily by muscle and liver while inhibiting glucose production and glycogenolysis. Insulin is available in short-acting forms (regular insulin, insulin lispro) for use at the time of caloric consumption as well as medium (NPH) and long-acting (insulin glargine) forms, which provide baseline glucose-controlling effect throughout the day. Because of its glucose-lowering effect, insulin dosing requires adjustment prior to exercise as addressed here.

In T2D patients, relative insulin deficiency is due to cellular resistance to insulin action at the levels of the muscle cell, the adipocyte, the hepatocyte, and the β cell in addition to abnormally elevated glucagon. Medications for T2D thus target 3 major mechanisms: (1) impaired peripheral glucose uptake (liver, fat, muscle), (2) excessive hepatic glucose release (with elevated glucagon), and (3) insufficient insulin secretion.

**Insulin sensitizers**

Biguanides (metformin) and thiazolidinediones (TZD) (rosiglitazone, pioglitazone) specifically target insulin resistance. Metformin enhances the sensitivity of both peripheral (primarily muscle) and hepatic tissues to insulin. It also inhibits hepatic gluconeogenesis and glycogenolysis. TZDs improve insulin sensitization at the level of muscle and adipocyte by activating peroxisome proliferators-activated gamma receptors (PPAR-gamma). Because of their mechanism of action, these agents do not place patients at risk for hypoglycemia and can be safely used without dosage adjustment before or after exercise.

**Insulin secretagogues**

These agents primarily address the progressive decline in β-cell function seen in T2D diabetes. Sulfonylurea agents (glipizide, glyburide, glimepiride, chlorpropamide) are best used as an adjunct to insulin sensitizer therapy to achieve goal levels of control. Sulfonylureas enhance insulin secretion after binding to specific receptors on β cells. Receptor activation leads to closure of a potassium-dependent ATP channel, leading to decreased potassium influx and depolarization of the β-cell membrane. Sulfonylureas suppress hepatic glucose production as well.

Glinides are newer insulin secretagogues, which include repaglinide, a meglitinide, and the amino acid derivative nateglinide. Each is taken before meals and acts rapidly to increase insulin production in an effort to restore premeal glucose levels and control postprandial glucose. They do not need to be taken if meals are missed, or dosing can be skipped if exercise follows the meal.

In general, insulin secretagogues should be half-dosed on days of exercise, particularly if the athlete is near “goal HgA1c”, as their hypoglycemic risk will be correspondingly higher. Nonetheless, the severity of hypoglycemia with these agents is, in general, low.
**Carbohydrate-absorption blockers (alpha-glucosidase [α-glucosidase] inhibitors)**
Acarbose and miglitol are taken before meals to reduce glucose absorption, decrease meal-associated blood glucose elevations, and thus reduce the required insulin response. The usefulness of this class of medications can be limited by gastrointestinal discomfort and variability in gastric emptying. They can be useful in addition to combination oral therapy or in patients with mild fasting hyperglycemia.

**Incretin potentiators**
Glucagon-like peptide-1 gut-derived incretin hormone stimulates insulin, suppresses hepatic glucose release, inhibits gastric emptying, and reduces appetite and food intake. Exenatide is injected subcutaneously twice daily, is approved for use in combination with sulfonylureas, metformin, or both, and is not associated with weight gain. Exenatide does not appear to increase the risk of hypoglycemia unless used in combination with a sulfonylurea. It also does not blunt the glucagon response to hypoglycemia, and no dose adjustment is warranted before exercise.

Dipeptidyl peptidase IV (DDP-4) inhibitors target both excess glucagon and inadequate postmeal insulin secretion. Sitagliptin was the first approved DDP-4 inhibitor, having shown the ability to enhance normal insulin action while suppressing glucagon during a meal. The overall incidence of hypoglycemia with DDP-4 inhibitors is equal to that with placebo, and there is no dose adjustment necessary before, during, or after exercise.

**On-Field Management of Complications**

**Hypoglycemia**
Hypoglycemia may arise for many reasons in the active population. Common causes include too high a daily dose of insulin or oral hypoglycemics, errors in dosage, increased activity duration or intensity, insufficient or delayed food intake, and alcohol intake during or immediately after exercise. As glycogen is used during exercise, the reduced glycogen concentration increases insulin action. Although the rate of CHO utilization depends on the intensity and duration of exercise, training status, and prior diet, as glycogen stores in active muscles and liver are depleted, the risk for hypoglycemia correspondingly increases.

Diabetic athletes with prior episodes of hypoglycemia generally demonstrate blunted neuroendocrine (glucagon, insulin, catecholamines) and metabolic (endogenous glucose production, lipolysis, ketogenesis) counter-regulatory responses during subsequent exercise. This “counter-regulatory failure mechanism” is postulated to be the result of cortisol stimulation that occurs during a stress such as hypoglycemia and the effect that it exerts on the central nervous system. These individuals maintain a higher susceptibility to hypoglycemia in the future.

**Pre-exercise prevention**
In T1D athletes, adjustments in insulin dosing are needed prior to exercise, but adequate CHO replacement during and after exercise appears to have the most profound effect on preventing hypoglycemia. If the insulin schedule is not altered before exercise, a CHO snack must be ingested to minimize the likelihood of hypoglycemia.

Individualization of insulin dosing, timing, and caloric intake before, during, and after exercise is critical. Decreasing 1 U of regular insulin from the usual dose or adding 15 g of CHO increases blood glucose by approximately 50 mg/dL. As noted earlier, hypoglycemia during exercise can be minimized by reducing the typical insulin dose by 30% to 50%. If exercise is beyond 60 minutes, insulin dose must be reduced by 80% of the initial dose in T1D athletes. Decreased insulin dose is not always possible
to anticipate, because exercise is often unplanned, particularly in children. Diet modification takes on additional significance in this group.

As stated above, no major dietary adjustments are necessary for T2D athletes. Those using oral hypoglycemics may need to adjust their dosing based on the relative risk of hypoglycemia inherent in the therapy that they are taking.

**Management of acute hypoglycemia**

Patients and providers should readily recognize the symptoms of hypoglycemia (glucose <70 mg/dL): dizziness, weakness, sweating, headache, hunger, pallor, blurred vision, slurred speech, confusion, irritability, and poor coordination. If hypoglycemia occurs, exercise should be stopped, and blood sugar should be monitored every 15 minutes until it rises above 80 mg/dL. Acute hypoglycemia should be treated immediately with 15 g of CHO: 1/2 cup of fruit juice, 4 glucose tablets, 6 oz of sweetened carbonated beverage, or 8 oz of low-fat milk. Special note should be made that patients using α-glucosidase inhibitors (acarbose or miglitol) concurrently with insulin or insulin secretagogues will require treatment with glucose, because these agents prevent rapid absorption of nonglucose CHO.

More pronounced hypoglycemia may require intravenous glucose, and severe hypoglycemia should be treated with glucagon 1 mg subcutaneously or intramuscularly to produce a rapid release of liver glycogen. It should be noted that this therapy is ineffective if all liver glycogen stores have been depleted by prolonged, intense exercise.

**Late-onset postexercise hypoglycemia**

The risk of hypoglycemia in insulin-treated patients persists long after strenuous exercise via several mechanisms. Late-onset postexercise hypoglycemia (LOPEH) has been seen in T1D 6 to 24 hours after activity. After exertion, muscle and hepatic glycogen stores are filled by using circulating plasma glucose. That, coupled with increased insulin sensitivity and glucose uptake by peripheral tissues and a blunting of the glucoregulatory response to insulin-induced hypoglycemia, may lead to late-onset hypoglycemia, often nocturnal. This syndrome most commonly occurs with increases in training level or during 2-a-day practices during preseason, although it may occur at any time.

Research in this area demonstrated that regardless of postexercise supplementation, glucose concentrations fell after 22.00 hours, and prebedtime snacks were important for helping to correct or avoid nocturnal hypoglycemia. Consumption of any commercially available sports drink has been shown to be effective in helping to avoid LOPEH; however, sports drinks with a mix of CHO, fat, and protein were associated with sustained hyperglycemia (and lack of hypoglycemia) during most of the postexercise period. This late-onset hypoglycemia is also effectively prevented by whole milk and slowly absorbed snacks, such as chips, chocolate, and most fruits.

**Hyperglycemia**

Frank hyperglycemia in athletes (>250 mg/dL) occurs more commonly in T1D generally as a result of low circulating insulin levels. Hyperglycemia may also result from inadequate insulin administration, excessive food intake, inactivity, failure to take oral hypoglycemics, illness, stress, or injury.

If pre-exercise blood glucose is more than 250 mg/dL, T1D athletes should check for urinary ketones. If ketonuria is moderate to high, exercise should be avoided until glucose values improve and ketones resolve. Aggressive lowering of blood glucose in these patients may prevent development of ketoacidosis. It has been suggested that T1D athletes with moderate hyperglycemia (250–300 mg/dL) but no ketones may
exercise as long as they monitor their glucose every 15 minutes and demonstrate that glucose values are falling.\textsuperscript{50}

Patients with T2D should avoid exercise if blood glucose values are more than 400 mg/dL. The key differentiation between T1D and T2D diabetics with exercise is the risk of ketosis and acidosis in T1D patients with relatively inadequate insulin and poorly controlled blood glucose. Individuals with T2D demonstrate hyperglycemia primarily from overeating or poor glucose utilization from insulin resistance and/or insufficient activity.

**SUMMARY**

Although regular exercise is a pivotal component of management in diabetics, the demands of sports and competition can predispose athletes with diabetes to potentially harmful complications such as hypo- and hyperglycemia. A basic understanding of substrate metabolism, special nutritional needs, blood glucose control, medications, and management of on-field complications in athletes with diabetes is important for medical professionals charged with the daily care of diabetic athletes. Individual metabolic responses to exercise, differences in diets and timing of meals, the interplay between various stressors of daily life, anxiety provoked by the anticipation of competition, and varying medications and doses preclude the development of a "generalized" algorithm for the daily management of diabetic athletes. "Individualized" management strategies should be developed only after consultation with a team of medical professionals, including the athletic trainer, sports nutritionist, and physician.

**REFERENCES**

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