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Exercise in the Treatment and Prevention of Diabetes

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COLBERG, S.R. and C.R. GRIECO. Exercise in the treatment and prevention of diabetes. *Curr. Sports Med. Rep.*, Vol. 8, No. 4, pp. 169–175, 2009. *The inclusion of regular physical activity is critical for optimal insulin action and glycemic control in individuals with diabetes. Current research suggests that Type II diabetes mellitus can be prevented and that all types of diabetes can be controlled with physical activity, largely through improvements in muscular sensitivity to insulin. This article discusses diabetes prevention and the acute and chronic benefits of exercise for individuals with diabetes, along with the importance and impact of aerobic, resistance, or combined training upon glycemic control. To undertake physical activity safely, individuals also must learn optimal management of glycemia.*

INTRODUCTION

Although physical activity is a cornerstone in the management of diabetes mellitus (DM), many individuals with this chronic disease fail to become or remain regularly active (40). However, high-quality studies proving the importance of exercise and fitness in diabetes were lacking until recent years, and this lifestyle choice was the least prescribed by physicians compared with dietary changes and medication use. It is now well established that participation in regular physical activity usually improves glycemic control and can prevent type 2 (T2) DM (12,21,30,32,35,48). In fact, lifestyle interventions have been shown to lower T2 DM risk by 58% in high-risk populations (30,48).

Moreover, most benefits of physical activity upon diabetes management and prevention are realized through acute and chronic improvements in insulin action (5,8,26,27,47,50). The acute effects of a recent bout of exercise are more prominent; however, regular exercise training generally results in a more lasting effect (3,22,41,50). Both aerobic and resistance training bestow benefits, particularly with regard to prevention of T2 DM and improvements in insulin action and glycemic control. In this review, the benefits of these types of physical training are addressed, along with guidelines for maintenance of glycemic balance with exercise.

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PREVENTION OF DIABETES WITH LIFESTYLE IMPROVEMENTS AND PHYSICAL ACTIVITY

Many physicians still fail to realize that T2 DM is largely preventable with lifestyle modifications and that diabetes management is facilitated by regular physical activity participation. Prospective studies have assessed the exercise habits of large numbers of high-risk individuals, concluding that regular physical activity is associated with a lower risk of T2 DM (25,38). While these studies have relied upon self-reported exercise habits, "active" individuals were generally leaner and had reduced levels of abdominal fat, lower fasting glucose levels, enhanced insulin action, and a lower risk of developing T2 DM.

A more recent clinical trial, the Diabetes Prevention Program (DPP), studied 3234 overweight adults with impaired glucose tolerance at high risk for DM, many of them high-risk minorities like African American and Hispanic individuals (30). The "lifestyle arm" participants followed a low-fat diet and averaged 150 min of weekly moderate-intensity activity (*e.g.*, brisk walking). After 3 yr, their average risk of developing DM was 58% lower than control subjects, regardless of ethnicity, age, or sex. Risk was reduced more among individuals aged 60 yr and older. DPP participants lost an average of only 7% of their body weight, but not all of it was maintained. However, follow-up studies have concluded that weight loss was the predominant predictor of reduced DM occurrence; for every kilogram of weight loss, there was a 16% reduction in risk, adjusted for changes in diet and activity (21). However, a lower percentage of calories from fat and increased physical activity predicted weight loss, and regular exercise was critical in sustaining a lower body weight. Among the 495 participants

who failed to meet the weight loss goal at the end of the first year (5%–7% overall weight loss), those who achieved the physical activity goal experienced a 44% lower DM incidence as well, suggesting that physical activity may bestow a greater benefit than weight loss alone.

A similar Finnish lifestyle study concurred with the DPP findings, demonstrating a 58% reduction in DM risk in its participants who lost body weight, lowered their fat intake, ate less saturated fat and more fiber, and added 30 minutes of daily walking and occasional resistance training (48). Moreover, those engaging in moderate to vigorous physical activities were approximately 64% less likely to develop DM (32), and low-intensity training, leisure-time physical activity, and walking conferred additional benefits afterwards (Fig. 1). Individuals engaging in at least 2.5 $h \cdot wk^{-1}$ of walking during follow-up (N = 157) were 63%–69% less likely to develop DM than those who walked less than 1 $h \cdot wk^{-1}$ (N = 192). Total increase in physical activity, rather than intensity, was most strongly associated with a lower DM risk (32).

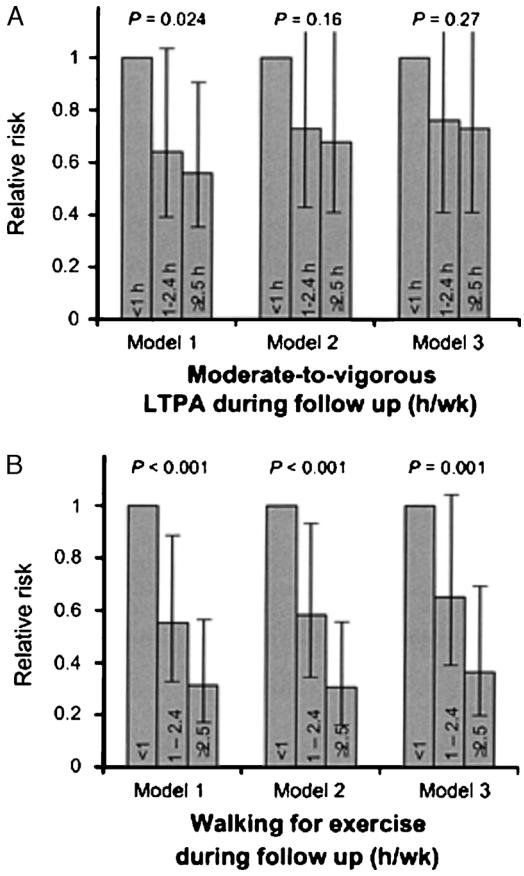

Figure 1. Relative risk of developing diabetes mellitus according to (A) average duration of moderate and vigorous physical activity during follow-up (<1, 1–2.4, and ≥2.5 $h \cdot wk^{-1}$) and (B) average duration of walking for exercise (<1, 1–2.4, and ≥2.5 $h \cdot wk^{-1}$) in the Finnish Diabetes Prevention Study. Model 1: Adjusted for age, sex, and *either* 1) change in moderate to vigorous LTPA from baseline and average low-intensity LTPA during follow-up and its change from baseline or 2) change in duration of walking for exercise from baseline and other forms of LTPA. Model 2: Adjusted for variables in Model 1 and average values of dietary intake of energy, total and saturated fat, and fiber during follow-up and their changes from baseline. Model 3: Adjusted for variables in Model 2 and average body mass index during follow-up and its change from baseline. LTPA = leisure time physical activity. (Reprinted from Laaksonen DE, Lindstrom J, Lakka TA, *et al.* Finnish diabetes prevention study: physical activity in the prevention of type 2 diabetes. *Diabetes*. 2005; 54:158–65. Copyright © 2005 American Diabetes Association. Used with permission.)

Others also have concluded that T2 DM may be preventable and that prediabetes is reversible. A lifestyle intervention in sedentary, insulin-resistant, middle-aged adults (12) found that 30 min of moderate walking done 3–7 $d \cdot wk^{-1}$ for 6 months reversed their prediabetic state. Insulin sensitivity was enhanced without dietary alterations, loss of body weight, or additional insulin release. However, their insulin action was retested only 24–48 h after the last session of exercise, possibly confounding the results with acute effects. Similarly, the China Da Quing Diabetes Prevention Study, a 6-yr lifestyle intervention involving dietary and exercise improvements (singly and in combination), investigated the development of T2 DM in a population of 577 adults with impaired glucose tolerance. At follow-up 14 yr later, the lifestyle intervention group exhibited a 43% lower incidence of T2 DM, combined with a significant delay (3.6 yr) in its onset (35).

ACUTE AND CHRONIC BENEFITS OF PHYSICAL ACTIVITY

Acute Effects

Although responses can vary in insulin-requiring individuals, most with T2 DM experience a decrease in their blood glucose levels during mild- and moderate-intensity exercise and for 2–48 h afterwards (1,22). The decrease in blood glucose is related to the duration and intensity of the exercise, preexercise glycemic control, and state of physical training (1,3,9,47). The ability of more intense or prolonged exercise to acutely enhance insulin sensitivity usually results in more effective postexercise glycemic control in people with insulin resistance or DM when they undertake such activities (47). For 2 h or more afterwards, prior activity causes an enhanced uptake of blood glucose for glycogen synthesis.

Why are blood glucose levels generally decreased by exercise? In muscle cells, at least two distinct mechanisms exist to stimulate glucose uptake and glycogen synthesis during and after a bout of physical activity. One of these is insulin-dependent and predominates during rest, while the other results from muscle contractions (22,47). A single bout of exercise increases skeletal muscle glucose uptake via the latter mechanism, bypassing typical defects in insulin action associated with T2 DM, although the resultant, postexercise increase in insulin action is generally short-lived and disappears after approximately 48 h (22,29). Immediately after exercise, glycogen depletion stimulates glucose uptake, which occurs more rapidly for several hours after an acute session of exercise with little insulin required (20).

Acute improvements in insulin sensitivity in women with T2 DM have been found for equivalent total energy expenditures whether they engaged in low-intensity or high-intensity walking (5). However, Braun *et al.* (4) also found that total carbohydrate oxidation and estimated

muscle glycogen use during an acute session of exercise were lower in overweight, insulin-resistant women, even though blood glucose uptake was unaltered by differences in insulin action. Lower glycogen use during an activity would likely result in a less sustained impact upon post-exercise glucose uptake and insulin action. In any case, it is best to advise patients to engage in a bout of exercise minimally every 48 h to keep insulin action enhanced.

Chronic Effects

Regular exercisers benefit not only from the acute effects of the last bout of exercise, but also from more sustained physical changes resulting from repeated exercise participation. For example, chronic physical training can enhance both the responsiveness of skeletal muscles to insulin and basal blood glucose uptake. The molecular mechanisms attributed to this are increased expression and/or activity of proteins involved in glucose uptake and metabolism and insulin signal transduction, such as adenosine monophosphate–activated protein kinase (22). In addition, increased lipid oxidative capacity of skeletal muscle likely is involved. Likewise, in healthy young men, 6 wk of moderate-intensity cycling performed for 1 h, 5 $d \cdot wk^{-1}$ increased not only insulin sensitivity, but also glucose effectiveness (the ability of hyperglycemia to promote glucose disposal at basal insulin levels) for at least 1 wk after training (41).

Even short-term aerobic training lasting 7 d can lead to improvements in whole-body insulin sensitivity in individuals with T2 DM, demonstrating that peripheral, but not hepatic, insulin action can be enhanced acutely by regular physical training without weight loss and without evoking a true training adaptation in muscle (50). While the effects of a single workout may last from 1 h (after short, mild exercise) to up to 1 or 2 d (for prolonged, intense activities), the effects of training also may begin to reverse within 2–3 d, regardless of enhancements in muscle mass. By way of example, in middle-aged, moderately trained subjects, 5 d of moderate training done 45 min daily resulted in a 43% loss in insulin sensitivity after 1 d of inactivity and a 66% loss in insulin sensitivity after 3 d (29), reinforcing the need for regular exercise participation regardless of an individual's training state.

Insulin sensitivity also may be affected by age and training status (3,14,19,45). In a recent study of a group of individuals 77–87 yr old, both aerobic power and insulin action improved in response to high-intensity aerobic exercise training, but their improvements in insulin action were attenuated compared with middle-aged subjects (14). Moderate- to heavy-intensity aerobic training undertaken 3 times a week for 6 months improved insulin sensitivity in both younger and older women (19); however, although the older women alone decreased their body weight (by 4%), enhancements in insulin sensitivity persisted only in the younger women for 72–120 h after the last exercise session. Thus, in older women, improvements in insulin sensitivity may result more from the acute effect of the last session than chronic training adaptations. Conversely, muscle mitochondrial function, muscle fat oxidative capacity, and insulin sensitivity in trained young and older individuals improved in both training groups after 8 wk of aerobic training compared with their sedentary, age-matched counterparts, suggesting that the elderly subjects were not impaired by age as much as physical inactivity (45).

Finally, regular physical activity (both aerobic and resistance) actually may play a role in preventing not only diabetes, but also DM-related complications (including both microvascular and macrovascular) by ameliorating a state of low-level inflammation associated with insulin resistance and chronic hyperglycemia (8,28). Likewise, aerobic exercise training also may slow the progression of or prevent the onset of diabetic peripheral neuropathy (2), improve endothelial function (8), and enhance β-cell insulin production in individuals with T2 DM as long as adequate amounts of functioning β-cells remain in the pancreas (11).

MANAGING DIABETES USING AEROBIC EXERCISE

To keep insulin action optimized, it is recommended that successive exercise sessions be separated by no more than 72 h and occur at least 3 $d \cdot wk^{-1}$ (nonconsecutive). They should comprise low- to moderate-intensity physical activities, lasting a minimum of 10–15 min per session, with 30 min at a time being a goal (47). The type of aerobic exercise undertaken may vary based upon the presence of coexisting complications like peripheral neuropathy or degenerative arthritis, which may require non-weight-bearing alternatives (*e.g.*, stationary cycling, armchair exercises, swimming, or other aquatic activities) (1,47).

Aerobic exercise has been the mode traditionally prescribed for diabetes management. Acute changes in blood glucose levels during aerobic exercise usually reflect carbohydrate use. When undertaking even 30 min of continuous, moderate exercise, blood glucose levels decrease (43), and 8 wk of moderate aerobic exercise training has been shown to increase glycogen synthase (GS) activity and GLUT4 protein expression, but not insulin signaling, in both overweight diabetic and control subjects (7). Greater duration activity depletes more muscle glycogen and actually may require intake of carbohydrates during the activity to delay fatigue and provide an alternate carbohydrate source as glycogen becomes depleted (1,33).

An individual's training status additionally will affect the absolute use of carbohydrates during an activity. Aerobic training increases fat use for a similar bout of low- or moderate-intensity activity done after training, and fat use spares muscle glycogen and blood glucose (6). In diabetic individuals, greater fat use may result in a lesser decrease in glycemia during acute exercise (22,47). Thus, changes in fuel use with training may require smaller compensatory adjustments to carbohydrate or insulin intake when greater amounts of fat are used during activities. For hard-intensity exercise, however, the use of carbohydrates (including blood glucose) actually is increased further by aerobic training (37). Fat oxidation is a key aspect of improved insulin sensitivity in obese individuals (18), and exercise training increases intramyocellular lipid content and fat oxidation capacity (3,44).

Improvements in insulin action underlie enhanced glycemic control and lipid management in diabetic individuals. However, exercise intensity and duration may determine the

impact of aerobic exercise upon insulin action. Higher-intensity aerobic training clearly improves insulin sensitivity (14), but some studies have investigated whether less vigorous workouts have a similar effect. Houmard *et al.* (24) studied 154 middle-aged, sedentary, overweight subjects engaging in aerobic training for 6 months. One group did low-volume/moderate-intensity training consisting of the equivalent of approximately 12 miles of walking weekly, performed at 40%–55% of peak oxygen consumption; another was assigned to low-volume/high-intensity workouts entailing approximately 12 miles of weekly jogging; and the last group jogged approximately 20 miles weekly (high-volume/high-intensity aerobic training). As shown in Figure 2, all training groups had enhanced insulin sensitivity, but those that exercised approximately 170 min·wk^{-1} (*i.e.*, low/moderate and high/high groups) experienced greater improvements in insulin action than the low/high group that only exercised for approximately 115 min·wk^{-1}, suggesting that exercise duration may be relatively more important than training intensity to improve insulin action in middle-aged, overweight individuals.

THE ROLE OF RESISTANCE TRAINING IN DIABETES MANAGEMENT

Resistance training also is recommended for all diabetic individuals because it has the potential to improve muscular strength and endurance, enhance flexibility, improve body composition, and decrease risk for cardiovascular disease, all while increasing amounts of insulin-sensitive muscle mass (8,13,15,26,27). It ideally should be undertaken a minimum of twice weekly as part of a well-rounded exercise program (including aerobic and flexibility training) and include 8–10 exercises involving the major muscle groups and a minimum of one set of 10–15 repetitions to near fatigue (1,47).

Although even twice weekly training has proven benefits for men with T2 DM, Dunstan *et al.* (13) showed that older, diabetic men successfully progressed over 6 months of supervised resistance training to completing thrice weekly training consisting of three sets of 8–10 repetitions performed at 75%–80% of maximal on 8–10 exercises. By doing so, they experienced much greater increases in muscle mass, decreases in body fat, and improvements in glycemic control than control subjects. Such training may, therefore, be a better goal for most individuals (47).

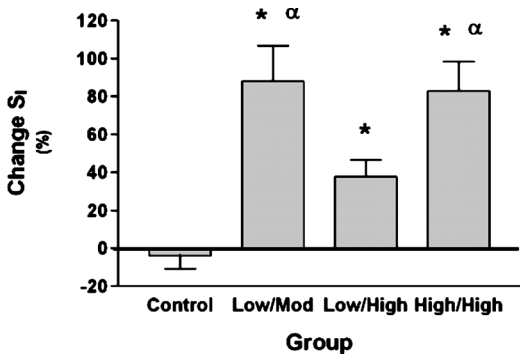

Figure 2. Relative changes (%) in the insulin SI derived from the intravenous glucose tolerance test in control and exercise groups (N = 154). Low/Mod = low-volume/moderate-intensity training; Low/High = low-volume/high-intensity training; High/High = high-volume/high-intensity training. Line at 0 represents no change. *Significant difference from control ($P < 0.05$). αSignificant difference from the Low/High group ($P < 0.05$). SI = sensitivity index. (Reprinted from Houmard JA, Tanner CJ, Slentz CA, *et al.* Effect of the volume and intensity of exercise training on insulin sensitivity. *J. Appl Physiol.* 2004; 96:101–6. Copyright © 2004 The American Physiological Society. Used with permission.)

Despite possibly resulting in transient elevations in blood glucose levels, resulting in an exaggerated release of glucose-raising hormones like epinephrine during intense workouts (31), weight training benefits glycemic control and insulin sensitivity, particularly in individuals with T2 DM (1,47). It chronically increases GLUT4 glucose transport proteins in trained muscle, along with acutely elevating insulin receptors, protein kinase B, GS, and GS total activity (23,49). In T2 DM subjects, insulin action was shown to be enhanced by 48% after 4–6 wk of moderate-intensity resistance training done 5 d·wk^{-1}, with minimal change in body fat or muscle and no change in maximal aerobic capacity (27). Moreover, twice weekly progressive resistance training done for 16 wk by 10 older men with newly diagnosed T2 DM resulted in a 46.3% increase in insulin action, along with a 7.1% reduction in fasting glycemia and a significant loss of visceral fat, with a 15.5% increase in overall calorie intake (26).

BENEFITS OF COMBINED AEROBIC AND RESISTANCE TRAINING

A training program that combines the use of aerobic and resistance training may provide the greatest benefit to glycemic management in individuals with any type of DM. For instance, in older, postmenopausal women with T2 DM, combining aerobic and resistance training results in even greater improvements in insulin action and glycemia (as measured by glycated hemoglobin levels, or HbA1c, as shown in the Table), as well as a larger decrease in their abdominal fat compared with aerobic training alone (10). Some of these additional benefits of combined training may simply be the result of a greater caloric expenditure, however.

Nonetheless, the two types of training appear to confer their benefits through divergent mechanisms. For example, in nonobese young women (18–35 yr of age), 6 months of either aerobic or resistance training improved muscle glucose disposal. Enhanced fat-free mass resulting from resistance training contributed to glucose uptake via a mass action effect without altering the intrinsic capacity of muscle to respond to insulin, whereas aerobic training enhanced glucose disposal independently of changes in muscle mass or maximal aerobic capacity, suggesting that these changes resulted from enhanced insulin sensitivity instead (42). In another study, when 4 months of resistance exercise were added to an existing aerobic training program for overweight men (15), insulin responses to an oral glucose load decreased by 25%, although they did not change at all in men engaging in aerobic activities only. Thus, resistance training may exert an even more substantial effect upon insulin action than aerobic.

TABLE. Effect of training interventions upon overall glycemic control in type 2 diabetes mellitus.

Study (Year)	Training Duration and Mode(s)	Subjects	Change in HbA1c (% Units)
Cohen (2008) (8)	14 months; resistance, supervised and non-supervised (2–3×·wk^{-1})	28 men/women with T2 DM, mean age 60.5 yr	−0.6 (center-based); −0.2 (home-based)
Cuff (2003) (10)	16 weeks; aerobic (moderate), combined (3×·wk^{-1})	28 women with T2 DM, mean age 60.9 yr	−0.1 aerobic; −0.1 combined
Dunstan (2002) (13)	6 months; resistance (2×·wk^{-1})	26 men/women with T2 DM, aged 60–80 yr	−1.2 for resistance, plus moderate weight loss
Ibañez (2005) (26)	16 wk; resistance (2×·wk^{-1})	9 men with T2 DM, mean age 66.6 yr	No HbA1c values; −11.6 mg·dL^{-1} fasting blood glucose
Ishii (1998) (27)	4–6 wk; resistance (5×·wk^{-1})	9 adults with T2 DM, mean age 46.8 yr	No change in HbA1c; insulin sensitivity increased 48%
Maiorana (2002) (36)	8 wk; combined circuit training (with aerobic and resistance modalities) (3×·wk^{-1})	14 men, 2 women with T2 DM, mean age 52 yr	−0.6
Marcus (2008) (39)	16 wk; aerobic, combined (3×·wk^{-1})	15 adults with T2 DM, mean age 54.6 yr	−0.31 aerobic; −0.59 combined
Sigal (2007) (46)	6 months; aerobic, resistance, combined (3×·wk^{-1})	251 adults with T2 DM, aged 39–70 yr	−0.51 aerobic; −0.38 resistance; −0.97 combined

HbA1c = glycated hemoglobin; T2 DM = type 2 diabetes mellitus.

A recent study assessed the effects of aerobic, resistance, and combined exercise training upon 251 adults aged 39–70 yr with T2 DM (46). Exercise training was performed three times weekly for 22 wk. Compared with aerobic or resistance training alone, combined training resulted in an additional improvement in overall glycemic control, suggesting that inclusion of both types is most beneficial. One limitation of this study, however, was that the total duration of exercise was greatest in the combined exercise training group, which also was the case in a similar study conducted by Marcus *et al.* (39) that investigated aerobic training compared with combined aerobic and high-force eccentric resistance exercise. During that 16-wk intervention in 15 adults with T2 DM, both groups experienced significant improvements in long-term glycemic control and physical performance, but the combined-exercise group exhibited greater improvements in body mass index and mid-thigh cross-sectional area. In any case, it is clear that both types of exercise confer physical benefits and should be considered critical to include in a regular training program for anyone with diabetes.

MANAGEMENT OF GLYCEMIA DURING EXERCISE

Many individuals with diabetes or prediabetes fail to engage in regular physical activity. Morrato *et al.* (40) concluded that just 39% of adults with diabetes are physically active (*i.e.*, engaging in moderate or vigorous activity at least 30 min, three times weekly), compared with 58% of those without the disease. However, appropriate exercise can be undertaken safely, and glycemia can be managed effectively (1,47).

Of greatest concern to many is the risk of upsetting glycemic balance with exercise, thereby resulting in hypoglycemia during or after exercise or exaggerated hyperglycemia. Undoubtedly, diabetes management can be further complicated by the inclusion of physical activity, particularly in insulin users. For instance, antecedent moderate-intensity exercise has been shown to blunt autonomic, neuroendocrine, and metabolic counter-regulatory responses to subsequent exercise or to hypoglycemia (16,17). Moreover, repeated episodes of prolonged exercise of low (*e.g.*, slow walking) and moderate (like brisk walking) intensities may blunt the release of glucose-raising hormones like epinephrine and glucagon and subsequent production of endogenous glucose in response to next-day hypoglycemia (17). Thus, prior activity may increase the risk of experiencing a more severe hypoglycemic event, particularly when exogenous insulin is taken.

As discussed, the glycemic effect of exercise can vary, and intense activities can cause transient elevations in blood glucose levels (31). Intermittent high-intensity exercise undertaken immediately after breakfast by eight individuals with T2 DM treated by diet only resulted instead in reduced blood glucose levels and insulin secretion, likely from elevated circulating insulin following a meal (33). Activities that are of longer duration and lower intensity generally result in a decline in blood glucose levels in all individuals with DM, regardless of the type (1,3,9,47).

In individuals with T2 DM controlled with diet and exercise alone, the risk of developing low blood glucose levels during exercise is minimal, making stringent measures unnecessary to maintain blood glucose control with exercise (47). However, blood glucose monitoring should be performed before and after an activity to assess its unique effect upon glycemia. Supplementing with carbohydrates generally is not needed with these individuals, although monitoring will reveal who may need additional carbohydrates to prevent hypoglycemia during and after exercise. Carbohydrate can be supplemented at 15 g·h^{-1} of exercise until optimal intake can be determined with glucose monitoring (47).

Prolonged exercise has a greater potential to cause hypoglycemia both during and after exercise, particularly in people treated with certain oral hypoglycemic agents or exogenous insulin. To prevent hypoglycemia, individuals

likely will need to consume rapidly absorbed carbohydrates to counterbalance declines in blood glucose levels and/or to reduce their oral medications or insulin dosing before (and possibly after) exercise (1,33,47). Use of oral hypoglycemic agents with a longer duration (*e.g.*, glyburide) increases the risk of exercise-induced hypoglycemia compared with treatment with diet and exercise alone (34). Even newer generation, shorter-acting sulfonylureas such as glimepiride and glibenclamide can increase the risk of hypoglycemia with exercise (34).

Before regular, planned exercise, short-acting insulin doses likely will need to be reduced to prevent hypoglycemia in insulin users. Newer, synthetic insulin analogues, such as insulin lispro, also induce more rapid decreases in blood glucose concentrations than regular human insulin. Individuals will need to monitor blood glucose levels before, occasionally during, and after exercise and compensate with appropriate dietary and/or medication regimen changes, particularly when exercising at times when these insulins are peaking (1). If only longer-acting insulins, such as insulin glargine, are being absorbed from subcutaneous depots during physical activities, immediate declines in glycemia during exercise are less likely.

Later-onset hypoglycemia becomes a greater concern when carbohydrate stores (*i.e.*, muscle and liver glycogen) are depleted during an acute bout of exercise. In particular, high-intensity exercise (*e.g.*, repeated interval or intense resistance training) can result in substantial depletion of muscle glycogen, thereby increasing the risk for later onset hypoglycemia, particularly if supplemental insulin or longer lasting sulfonylureas are taken (1,34). The consumption of moderate amounts of carbohydrates (5–30 g) during and within 30 min after exhaustive, glycogen-depleting exercise will lower hypoglycemia risk and allow for more efficient restoration of muscle glycogen (20). To maintain effectively glycemic control and optimize glycogen resynthesis, a smaller-than-usual insulin dose still may be required for carbohydrate ingested immediately after exercise.

For pre-exercise blood glucose levels of less than 100 $mg \cdot dL^{-1}$ (5.5 mM), the American Diabetes Association recommends that carbohydrate be ingested before any activity is undertaken. However, this recommendation would apply only to insulin users (with T1 or T2 DM taking insulin injections) or T2 DM individuals using certain sulfonylureas (34). If controlled with diet or oral diabetic medications alone, most individuals will not need to eat carbohydrates for exercise lasting less than 1 h. Others should ingest at least 15 g of carbohydrate before exercise for an initial blood glucose level of 100 $mg \cdot dL^{-1}$ or lower, with the actual amount dependent upon injected insulin peaks and exercise duration (47). Intense, short exercise requires lesser or no carbohydrate intake (31).

Although hyperglycemia is often defined as any blood glucose value in excess of 125 $mg \cdot dL^{-1}$ (6.9 mM), an abnormal glycemic response to exercise usually is not experienced until levels exceed twice that, or 250 $mg \cdot dL^{-1}$ (13.9 mM), and then likely only in individuals who are insulin-deficient and ketotic (1). Although uncommon in T2 DM, ketone body production may increase and result in diabetic ketoacidosis. A more usual recommendation for T2 DM exercisers is to "use caution" when undertaking physical activities with elevated glucose levels (over 300 $mg \cdot dL^{-1}$, or 16.7 mM, but without ketosis) by monitoring blood glucose levels frequently and hydrating adequately (1,47). If hyperglycemic after a meal for which insulin was injected, individuals still will likely experience a reduction in blood glucose during aerobic exercise if circulating insulin levels are elevated (43).

Safe exercise participation can be complicated by the presence of DM-related health complications like cardiovascular disease, hypertension, neuropathy, or microvascular changes. For more information, readers are referred to the American College of Sports Medicine Position Stand on exercise and T2 DM (1) and a review by Sigal and others (47). Before embarking on a new exercise program, diabetic individuals are advised to undergo a medical evaluation to screen for the presence of complications that potentially could be worsened by exercise (*e.g.*, cardiovascular disease, peripheral arterial disease, proliferative retinopathy, nephropathy, and peripheral and autonomic neuropathies). Examinations should screen for glycemic control, physical limitations related to joint problems, prescribed medications, and the type and severity of complications. A graded exercise test may be recommended to screen for cardiovascular disease depending upon the age of the person and DM duration (1).

CONCLUSION

Physical activity plays a major role in the prevention and control of insulin resistance, prediabetes, and T2 DM, in combination with other lifestyle improvements. Both aerobic and anaerobic forms of training improve sensitivity to insulin in diabetic individuals and, as a result, can assist with management of glycemia, both acutely and chronically. Individuals with diabetes can exercise safely without fear of unbalancing their glycemic control, as long as certain precautions are taken, and should participate in regular physical activity, including both aerobic and resistance. The inclusion of an exercise program in the management of DM is critical for optimal health.

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