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Review Article
Role of exercise in diabetes

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Abstract

The role of exercise in the treatment of diabetes is becoming more defined today. In individuals treated by diet alone, regulation of blood glucose during exercise is usually not a significant problem and exercise can be used as an adjunct to diet to achieve weight loss and improved insulin sensitivity. When obese type II diabetic patients are treated with very low calorie diets, adequate amounts of carbohydrate must be provided to ensure maintenance of normal muscle glycogen content, particularly if individuals wish to participate in high intensity exercise that places a heavy workload on specific muscle groups. On the other hand, moderate intensity exercise such as vigorous walking can be tolerated by individuals on very low calorie, carbohydrate-restricted diets after an appropriate period of adaptation. A number of strategies can be employed to avoid hypoglycemia in type I diabetic patients, and both type I and II diabetic patients should be examined carefully for long-term complications of their disease, which may be made worse by exercise. These considerations have led many diabetologists to consider exercise beneficial in the management of diabetes for some individuals but not recommended for everyone as a necessary part of diabetes treatment as in the past. The goals should be to teach patients to incorporate exercise into their daily lives if they wish and to develop strategies to avoid the complications of exercise. The rationale for the use of exercise as part of the treatment program in type II diabetes is much more clear than for type I diabetes; regular exercise may be prescribed as an adjunct to caloric restriction for weight reduction and as a means to improve insulin sensitivity in the obese insulin-resistant individual.

Introduction

Physical activity is any body movement produced by skeletal muscles that result in energy expenditure beyond resting expenditure. Exercise is defined as a physical activity that is planned, structured, repetitive, and purposeful, usually aimed at improving or maintaining physical fitness.

Physical activity has the potential to yield several health benefits for people with diabetes. These benefits can include improvements in glucose control, insulin sensitivity, lipid profile, blood pressure coagulation properties, body composition and psychological well being. Nearly everyone with diabetes can derive some benefit from an exercise program, although not all benefits will be realized by each person with diabetes. Both health care professionals and patients with diabetes need to remember this when determining the components of an exercise program. When chronic complications of diabetes develop, the benefits and risks of exercise must be carefully considered to maximize the benefits and assure safety.

Diabetes Mellitus

Diabetes is a group of chronic metabolic disorders characterized by hyperglycemia resulting from a relative deficiency in insulin through either reduced insulin secretion or reduced insulin action or both [1].

The major forms of diabetes can be categorized as type 1 (T1DM) or type 2 (T2DM). Less common forms include gestational diabetes (GDM), which is associated with a 40%-60% chance of developing T2DM in the next 5-10 yr. [1].

Blood Glucose Control

At Rest

At rest, muscle derives only —10% of its energy requirements from glucose oxidation, 85-90% from the oxidation of fatty acids, and 1-2% from amino acids [1].

During Exercise

With the onset of exercise, glucose utilization increases rapidly. The initial response is a rapid breakdown of muscle glycogen stores stimulated by activation of the sympathetic nervous system. Lactate accumulates rapidly and is released into the circulation. With continued exercise, blood flow to the muscles is increased, glucose uptake from the circulation

occurs, and lactate release declines as aerobic metabolism is established. The increase in glucose uptake by exercising muscles is closely matched by increased hepatic glucose production, and blood glucose concentrations stay relatively constant. Hepatic glucose production is due predominantly to glycogenolysis with only —25% coming from gluconeogenesis [1].

The onset of exercise is also associated with the activation of lipolysis in adipose tissue and the release of free fatty acids (FFAs) and glycerol into the circulation. FFA concentrations rise and are taken up and utilized by exercising muscle in proportion to their concentration in plasma. Lactate, pyruvate, alanine, and other gluconeogenic amino acids released from muscle and glycerol released from adipose tissue are extracted by the liver and utilized for gluconeogenesis. These reactions are regulated by a complex but highly integrated system of neural and hormonal responses. Insulin secretion is inhibited by increased sympathetic nervous system activity via a-adrenergic receptors, and plasma insulin concentrations decline to low levels. This results in increased lipolysis in adipose tissue and increased hepatic glucose production. Because exercise stimulates glucose uptake in muscle in the presence of very low concentrations of insulin, the fall in plasma insulin does not impair glucose utilization by working muscles or by other vital type II tissues such as the central nervous system. During exercise, plasma concentrations of glucose counterregulatory hormones increase gradually and play an important role in maintaining glucose homeostasis. These hormones include glucagon, growth hormone, cortisol, norepinephrine, and epinephrine. Glucagon plays a major role in stimulating hepatic glucose production, increasing both glycogenolysis and gluconeogenesis. Norepinephrine is important in stimulating glycogenolysis in both liver and muscle and in stimulating lipolysis in adipose tissue. Epinephrine increases in response to high-intensity exercise or declining blood glucose and has its major effect on hepatic glycogenolysis. Growth hormone and cortisol appear to be less important in the response to short-term exercise but do act to increase lipolysis, decrease insulin-stimulated glucose uptake in peripheral tissues, and increase hepatic gluconeogenesis over longer periods [1].

Several factors influence the relative amounts of glucose and FFAs utilized during exercise. These include intensity and duration of exercise, level of physical training, antecedent diet, and effects of meals taken shortly before or during exercise. As the intensity of exercise is increased, glucose progressively becomes a more important substrate for energy production. When working at 50% of the maximum aerobic capacity (Vo2max), muscle derives —50% of its energy from glucose oxidation. At intensities of 70-75% Vo2max, glucose becomes the predominant metabolic fuel, and when exercise is at or near 100% of Vo2max, most of the energy is derived from glucose oxidation. Amino acids contribute only 1-2% of the energy required for muscular contraction at all intensities of exercise, and oxidation of lipids makes up the difference.

Thus during very high intensity exercise, glucose oxidation rates are markedly increased, muscle glycogen stores are depleted rapidly, and glucose uptake from the circulation is high. If hepatic glycogen stores are adequate, hepatic glucose production is able to match or exceed peripheral utilization and blood glucose concentrations remain constant or may actually increase. With increasing duration of exercise, muscle and hepatic glycogen stores fall and plasma FFA concentrations increase. Fatty acid oxidation by exercising muscle increases gradually and glucose oxidation decreases. Hepatic glucose production decreases and becomes progressively more dependent on gluconeogenesis but is usually sufficient to maintain normal blood glucose concentrations. Hypoglycemia rarely develops but may occur during prolonged, exhaustive exercise such as long distance running or cycling [1].

Type 1 Diabetes

In type 1 diabetes there is autoimmune destruction of the pancreas leading to a failure to secrete insulin. [2].

Blood Glucose Control

Ambient insulin concentrations are vital in normal glucose homoeostasis both during exercise and in recovery. In normal subjects, plasma insulin concentrations decrease to low levels during exercise. This decrease, in conjunction with increasing plasma concentrations of glucagon and other counter regulatory hormones, promotes increased hepatic glucose production to match the increased rate of peripheral glucose utilization. The low insulin concentration during exercise also promotes lipolysis, making FFAs available for oxidation by exercising muscle and glycerol available to the liver for gluconeogenesis. In the insulin-treated diabetic patients, plasma insulin concentrations do not decrease during exercise

and may even increase substantially if exercise is undertaken within an hour or so of an insulin injection. This effect is due to increased absorption of insulin from the subcutaneous tissue, particularly if the injection site is in an exercising part of the body. Enhanced insulin absorption by exercise is most likely to occur when the insulin injection is immediately before or within a few minutes of the onset of exercise. The longer the interval between injection and onset of exercise, the less significant this effect will be and the less important it is to choose the site of injection to avoid an exercising area. Some diabetologists point out that there is considerable variation of insulin absorption rates from different injection sites such as the thigh, abdomen, or arm and that this may have more of an effect on the rate of insulin absorption than the exercise itself. A good rule of thumb is to avoid vigorous exercise within 60-90 min of an insulin injection to minimize enhanced absorption.

However, even with this precaution, plasma insulin concentrations do not fall normally during exercise in type I diabetic patients and glucose homeostasis may be impaired.

The sustained insulin levels during exercise may enhance peripheral glucose uptake and stimulate glucose oxidation by exercising muscle. However, the major effect is an inhibition of hepatic glucose production. Both glycogenolysis and gluconeogenesis are inhibited by the high insulin levels and, even though counter regulatory hormone responses may be excessive, the hepatic glucose production rate cannot match the rate of peripheral glucose utilization, and blood glucose concentration falls. During mild to moderate exercise of short duration, this decrease in blood glucose concentration may be considered a beneficial effect of exercise, but during more vigorous or prolonged exercise, hypoglycemia may result. The risk of hypoglycemia is particularly high in some diabetic patients with glucagon deficiency, because both insulin and glucagon play major roles in the regulation of hepatic glucose production during exercise. Strategies to avoid hypoglycemia during prolonged, vigorous exercise include decreasing insulin dosage before exercise and taking supplemental carbohydrate feedings before and during exercise [2-3].

Effect of exercise and meal

The metabolic responses of people with type I diabetes to moderate-intensity exercise 30 min after breakfast have been studied and compared to those of normal subjects. In normal subjects the expected postprandial rise in blood glucose and insulin concentrations is rapidly reversed by exercise, returning to fasting levels within 45 min. When exercise is stopped, there are moderate rebound increases in glucose and insulin concentrations that do not exceed those occurring after breakfast alone. Thus, 45 min of cycle exercise started 30 min after a meal has a significant but transient effect to lower blood glucose concentrations [2-3].

In patients treated with subcutaneous insulin, responses to exercise started 30 min after breakfast have been found to be variable, with the majority having improved blood glucose concentrations that persist even through lunch. Some subjects, however, show improved glucose levels during lunch only and a few show no significant improvement at all. Thus, the effect of exercise after meals on blood glucose concentrations and the appropriate adjustments in insulin dosage may vary considerably, and individual responses should be determined to achieve improved glucose control and avoid symptomatic hypoglycemia [2-3].

Risks of Exercise in Type 1 Diabetes

Another major problem for the type I diabetic patient is the occurrence of post exercise hypoglycemia. Many diabetic patients experience increased insulin sensitivity and hypoglycemic reactions for several hours after exercise, in some cases even the next day. This residual effect is due to increased glucose uptake and glycogen synthesis in the previously exercised muscle groups, which is associated with increased insulin sensitivity and activation of glycogen synthase. Hepatic glycogen stores are also rebuilt after exercise, but at a slower rate than occurs in muscle, so that increased requirements for dietary carbohydrate may persist for up to 24 h after prolonged glycogen-depleting exercise [3-4].

A third problem encountered by type I diabetic patients occurs when exercise is undertaken during insulin deficiency. In this situation, plasma insulin concentrations are very low or absent, and hyperglycemia and ketosis are present. With the onset of exercise, peripheral glucose utilization is impaired, lipolysis is enhanced, and hepatic glucose production and ketogenesis are stimulated. These changes result in a rapid rise in the already elevated blood glucose concentration and the rapid development of ketosis. In other words, the already poor metabolic control rapidly becomes worse, and instead of having a blood glucose-lowering effect, the exercise causes a rapid deterioration of the metabolic state. To avoid this deterioration, the diabetic patient should check his/her blood glucose concentration and urine ketones before undertaking vigorous physical activity. If blood glucose is >250 mg/dl and ketones are present in urine or blood, the exercise should be postponed and the individual should take supplemental insulin to reestablish good metabolic control. Likewise, if blood glucose is <100 mg/dl and the individual has taken insulin within the past 60-90 min, supplemental feedings should be taken before and during exercise to avoid hypoglycemia [3-4].

There are a large number of significant physical and psychological benefits of exercise which demand regular physical activity where possible. Unfortunately the evidence for improvements in glycaemic control in type 1 diabetes is not well established. Only a handful of studies, often relatively small, have provided mixed evidence for improvements in glycaemic control. Several have shown reduced insulin secretion and hypoglycemia but little reduction in HbA1c. In addition, some evidence suggests that the timing of exercise is

an important factor, with regular postprandial exercise improving long term blood glucose control [3-4].

A checklist of factors to consider before the onset of exercise is provided in Table 1 [1].

Type 2 Diabetes

In contrast to type I diabetes, in which deficiency of endogenous insulin is the primary defect, type II diabetes is characterized by insulin resistance and impaired insulin secretion but not total insulin deficiency. Obesity, hyperlipidemia, and hypertension are commonly associated with type II diabetes and treatment is frequently aimed at reversing all of these abnormalities by weight reduction by a combination of caloric restriction and increased energy expenditure by regular physical exercise. A major aim of therapy in type II diabetes is to improve insulin sensitivity through appropriate use of diet and exercise [1].

Table 1: Considerations before exercise

Type of exercise
Estimated intensity and duration of exercise
Estimated caloric expenditure
Is the exercise habitual or unusual?
How does the exercise relate to the level of physical conditioning?
Blood glucose
If < 100 mg/dl, take preexercise snack
If 100-250 mg/dl, all right to exercise
If >250 mg/dl, delay exercise and check urine ketones
Urine ketones
If negative, all right to exercise
If positive, take insulin; don't exercise until ketones are negative
Insulin
Type and dose
Time of injection
Site of injection
Food
Time of last meal
Preexercise snack
Carbohydrate feedings during exercise
Extra food after exercise

A number of strategies that may be useful to avoid either hypo- or hyperglycemia are outlined in Table 2 [1].

Table 2: Strategies to avoid hypo- or hyperglycemia with exercise

Food
Eat a meal 1-3 h before exercise
Take supplemental carbohydrate feedings during exercise, at least every 30 min if exercise is vigorous and of long duration
Increase food intake ^24 h after exercise, depending on intensity and duration of exercise
Insulin
Take insulin >1 h before exercise
Decrease insulin dose before exercise
Alter daily insulin schedule
Blood glucose monitoring
Monitor blood glucose before, during, and after exercise
Delay exercise if blood glucose is >250 mg/dl and ketones are present
Learn individual glucose responses to different types of exercise

Exercise helps to:

- Improve glucose control:
  - Improving insulin sensitivity.
  - Increasing GLUT4 (glucose transporter 4)
- Muscle uses more glucose, even at rest.
- Helps in weight lose.
- Improves physical fitness & reduces fat %.
- Improves CV (cardiovascular) function & CHD (coronary heart disease) risk profile.

Acute Effects of exercise

A.Fuel mobilization, glucose production, and muscle glycogenolysis.

The maintenance of normal BG at rest and during exercise depends largely on the coordination and integration of the sympathetic nervous and endocrine systems. Contracting muscles increase uptake of BG, although BG levels are usually maintained by glucose production via liver glycogenolysis and gluconeogenesis and mobilization of alternate fuels, such as free fatty acids (FFAs) [5-7].

Several factors influence exercise fuel use, but the most important are the intensity and duration of PA. Any activity causes a shift from predominant reliance on FFA at rest to a blend of fat, glucose, and muscle glycogen, with a small contribution from amino acids. With increasing exercise intensity, there is a greater reliance on carbohydrate as long as sufficient amounts are available in muscle or blood. Early in exercise, glycogen provides the bulk of the fuel for working muscles. As glycogen stores become depleted, muscles increase their uptake and use of circulating BG, along with FFA released from adipose tissue. Intramuscular lipid stores are more readily used during longer-duration activities and recovery. Glucose production also shifts from hepatic glycogenolysis to enhanced gluconeogenesis as duration increases [5-7].

B.Insulin-independent and insulin-dependent muscle glucose uptake during exercise.

There are two well-defined pathways that stimulate glucose uptake by muscle. At rest and postprandially, its uptake by muscle is insulin dependent and serves primarily to replenish muscle glycogen stores. During exercise, contractions increase BG uptake to supplement intramuscular glycogenolysis. As the two pathways are distinct, BG uptake into working muscle is normal even when insulin-mediated uptake is impaired in type 2 diabetes. Muscular BG uptake remains elevated postexercise, with the contraction-mediated pathway persisting for several hours and insulin-mediated uptake for longer [5-7].

Glucose transport into skeletal muscle is accomplished via GLUT proteins, with GLUT4 being the main isoform in muscle modulated by both insulin and contractions. Insulin activates GLUT4 translocation through a complex signaling cascade. Contractions, however, trigger GLUT4 translocation at least in part through activation of 5′-AMP–activated protein kinase. Insulin-stimulated GLUT4 translocation is generally impaired in type 2 diabetes. Both aerobic and resistance exercises increase GLUT4 abundance and BG uptake, even in the presence of type 2 diabetes [5-7].

C.Postexercise glycemic control/BG levels
Aerobic exercise effects.

During moderate-intensity exercise in nondiabetic persons, the rise in peripheral glucose uptake is matched by an equal rise in hepatic glucose production, the result being that BG does not change except during prolonged, glycogen-depleting exercise. In individuals with type 2 diabetes performing moderate exercise, BG utilization by muscles usually rises more than hepatic glucose production, and BG levels tend to decline. Plasma insulin levels normally fall, however, making the risk of exercise-induced hypoglycemia in anyone not taking insulin or insulin secretagogues very minimal, even with prolonged PA (physical activity). The effects of a single bout of aerobic exercise on insulin action vary with duration, intensity, and subsequent diet; a single session increases insulin action and glucose tolerance for more than 24 h but less than 72 h. The effects of moderate aerobic exercise are similar whether the PA is performed in a single session or multiple bouts with the same total duration.

During brief, intense aerobic exercise, plasma catecholamine levels rise markedly, driving a major increase in glucose production. Hyperglycemia can result from such activity and persist for up to 1–2 h, likely because plasma catecholamine levels and glucose production do not return to normal immediately with cessation of the activity [5-7].

Resistance exercise effects.

The acute effects of resistance exercise in type 2 diabetes have not been reported, but result in lower fasting BG 9blood glucose) levels for at least 24 h after exercise in individuals with IFG (impaired fasting glucose) [5-7].

Combined aerobic and resistance and other types of training.

A combination of aerobic and resistance exercise training may be more effective in improving BG control than either alone; however, more studies are needed to determine if total caloric expenditure, exercise duration, or exercise mode is responsible [5-7].

Chronic effects of exercise

A.Metabolic control: BG levels and insulin resistance.

Aerobic exercise has been the mode traditionally prescribed for diabetes prevention and management. Even 1 week of aerobic training can improve whole-body insulin sensitivity in individuals with type 2 diabetes. Moderate and vigorous aerobic training improve insulin sensitivity, albeit for only a period of hours to days, but a lesser intensity may also improve insulin action to some degree. Training can enhance the responsiveness of skeletal muscles to insulin with increased expression and/or activity of proteins involved in glucose metabolism and insulin signaling. Moderate training may increase glycogen synthase activity and GLUT4 protein expression but not insulin signaling. Fat oxidation is also a key aspect of improved insulin action, and training increases lipid storage in muscle and fat oxidation capacity [5-10].

An individual's training status will affect the use of carbohydrate during an aerobic activity. Aerobic training increases fat utilization during a similar duration bout of low- or moderate-intensity activity done after training, which spares muscle glycogen and BG and results in a lesser acute decrease in BG. Type 2 diabetes may be associated with a decrease in lipid oxidation and shift toward greater carbohydrate oxidation at all exercise intensities.

Resistance exercise training also benefits BG control and insulin action in type 2 diabetes. In a randomized controlled trial (RCT), twice-weekly progressive resistance training for 16 weeks by older men with newly diagnosed type 2 diabetes resulted in a 46.3% increase in insulin action, a 7.1% reduction in fasting BG levels, and significant loss of visceral fat. An increase in muscle mass from resistance training may contribute to BG uptake from a mass effect, and heavy weight training in particular may reverse or prevent further loss of skeletal muscle due to disuse and aging. In another RCT, all 20 men with type 2 diabetes who participated in either resistance or aerobic exercise thrice weekly for 10 weeks improved their overall BG control, but those doing resistance training had significantly lower A1C values. Diabetic women undergoing 12 weeks of low-intensity training with resistance bands had gains in strength and muscle mass and loss of fat mass but had no change in insulin sensitivity [5-10].

A.Lipids and lipoproteins.

Blood lipid responses to training are mixed but may result in a small reduction in LDL cholesterol with no change in HDL cholesterol or triglycerides. Combined weight loss and PA may be more effective than aerobic exercise training alone on lipids [5-10].

B.Hypertension.

Aerobic training may slightly reduce systolic BP, but reductions in diastolic BP are less common, in individuals with type 2 diabetes [5-10].

C.Mortality and CV risk.

Observational studies suggest that greater PA and fitness are associated with a lower risk of all-cause and CV mortality [5-10].

D. Body weight: maintenance and loss.

Recommended levels of PA may help produce weight loss. However, up to 60 min/day may be required when relying on exercise alone for weight loss [5-10].

E. Supervision of training.

Individuals with type 2 diabetes engaged in supervised training exhibit greater compliance and BG control than those undertaking exercise training without supervision [5-10].

F. Psychological effects.

Exercise likely has psychological benefits for persons with type 2 diabetes, although evidence for acute and chronic psychological benefits is limited. However, it seems that individuals who undertake exercise to prevent a chronic disease fare better than those who undertake it to manage an existing one.

Potential mechanisms of exercise include psychological factors, such as increased self-efficacy, a sense of mastery, distraction, and changes in self-concept, as well as physiological factors such as increased central norepinephrine transmission, changes in the hypothalamic adrenocortical system, serotonin synthesis and metabolism, and endorphins. Regular PA may improve psychological well-being, health-related QOL (quality of life), and depression in individuals with type 2 diabetes, among whom depression is more common than in the general population [5-10].

Guidance for type 2 diabetes mellitus:

- May need to modify oral hypoglycemic regimen
- Target of 20-60 minutes moderate intensity exercise at least 4 days per week
- Couple exercise regimen with diet planning to optimize treatment

Effect of Physical Activity on Blood Glucose
Depends on:

- **Physiologic Factors**: Status of Metabolic Control; Fitness Level; Blood Glucose at onset of Exercise; Insulin Resistance
- **Pharmacologic Factors**: Type of Insulin / Oral Hypoglycemic Agent; Site of Insulin Injection; Time of Insulin Injection
- **Exercise Factors**: Timing of Exercise; Intensity of Exercise; Duration of Exercise; Type of Exercise; Frequency of Exercise
- **Caloric Intake**: Timing of Pre-Exercise Meal; Caloric Content of Pre-Exercise Meal (Quantity and Type) [10-12].

Conclusion

In the context of diabetes, it is becoming increasingly clear that the epidemic of type 2 diabetes sweeping the globe is associated with decreasing levels of activity and an increasing prevalence of obesity. Thus, the importance of promoting physical activity as a vital component of the prevention as well as management of type 2 diabetes must be viewed as a high priority. It must also be recognized that the benefit of physical activity in improving the metabolic abnormalities of type 2 diabetes is probably greatest when it is used early in its progression from insulin resistance to impaired glucose tolerance to overt hyperglycemia requiring treatment with oral glucose-lowering agents and finally to insulin.

For people with type 1 diabetes, the emphasis must be on adjusting the therapeutic regimen to allow safe participation in all forms of physical activity consistent with an individual's desires and goals. Ultimately, all patients with diabetes should have the opportunity to benefit from the many valuable effects of physical activity.

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