AMERICAN COLLEGE of SPORTS MEDICINE®
POSITION STAND

Exercise and Type 2 Diabetes

This pronouncement was written for the American College of Sports Medicine by: Ann Albright, Ph.D., R.D. (Chairperson); Marion Franz, M.S., R.D., C.D.E.; Guyton Hornsby, Ph.D., C.D.E.; Andrea Kriska, Ph.D., FACSM; David Marrero, Ph.D.; Irma Ullrich, M.D.; and Larry S. Verity, Ph.D., FACSM.

SUMMARY

Physical activity, including appropriate endurance and resistance training, is a major therapeutic modality for type 2 diabetes. Unfortunately, too often physical activity is an underutilized therapy. Favorable changes in glucose tolerance and insulin sensitivity usually deteriorate within 72 h of the last exercise session; consequently, regular physical activity is imperative to sustain glucose-lowering effects and improved insulin sensitivity. Individuals with type 2 diabetes should strive to achieve a minimum cumulative total of 1000 kcal·wk^{-1} from physical activities. Those with type 2 diabetes generally have a lower level of fitness ($\dot{V}O_{2max}$) than nondiabetic individuals, and therefore exercise intensity should be at a comfortable level (RPE 10–12) in the initial periods of training and should progress cautiously as tolerance for activity improves. Resistance training has the potential to improve muscle strength and endurance, enhance flexibility and body composition, decrease risk factors for cardiovascular disease, and result in improved glucose tolerance and insulin sensitivity. Modifications to exercise type and/or intensity may be necessary for those who have complications of diabetes. Individuals with type 2 diabetes may develop autonomic neuropathy, which affects the heart rate response to exercise, and as a result, ratings of perceived exertion rather than heart rate may need to be used for moderating intensity of physical activity. Although walking may be the most convenient low-impact mode, some persons, because of peripheral neuropathy and/or foot problems, may need to do non-weight-bearing activities. Outcome expectations may contribute significantly to motivation to begin and maintain an exercise program. Interventions designed to encourage adoption of an exercise regimen must be responsive to the individual's current stage of readiness and focus efforts on moving the individual through the various "stages of change."

INTRODUCTION

Diabetes is one of the leading causes of death and disability in the United States with type 2 diabetes accounting for 90–95% of all diabetic cases (77). Based on national data, there are about 10.3 million diagnosed cases of diabetes in the United States with an estimated 5.4 million additional undiagnosed cases in the general population (40). Unfortunately, the diagnosis of type 2 diabetes is often delayed for years after the onset of the disease. A large portion of the burden of the disease falls upon the minority populations of the U.S., demonstrated by the fact that the prevalence rates of diabetes are higher among Native Americans, African Americans, Hispanic Americans, and Asian and Pacific Island Americans when compared with non-Hispanic whites (99). The long-term complications associated with type 2 diabetes are both microvascular and macrovascular in nature and include the following: retinopathy, peripheral and autonomic neuropathy, nephropathy, peripheral vascular disease, atherosclerotic cardiovascular and cerebrovascular disease, hypertension, and susceptibility to infections and periodontal disease (for an extensive description of the complications associated with diabetes, the reader is referred to *Diabetes in America*, 1995; 80,81).

The diagnosis and classification of diabetes have been revised by the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus (59). The new classification system emphasizes etiology and pathogenesis rather than modalities of treatment. Diabetes is divided into four major categories depending on etiology: type 1, type 2, gestational, and other specific types. In type 1 diabetes, the final common pathway is beta cell destruction by autoimmune processes, which leads to insulin deficiency. Type 2 diabetes is characterized by varying degrees of insulin resistance and relative insulin deficiency. Gestational diabetes is defined as any degree of glucose intolerance with onset or first recognition during pregnancy. The final category includes diabetes due to specific genetic defects, medications, and other diseases (59).

Guidelines for diagnosing diabetes have also been revised and are much simpler than the previous scheme. The new diagnostic criteria reflect more closely the prevalence of microvascular complications specific for diabetes. One of three criteria must be met for the diagnosis of diabetes: 1) a fasting plasma glucose $\geq$ 126 mg·dL^{-1}; 2) symptoms of diabetes such as polyuria, polydipsia and unexplained weight loss plus a casual plasma glucose of 200 mg·dL^{-1} or more; and 3) 2-h plasma glucose $\geq$ 200 mg·dL^{-1} during an oral glucose tolerance test using 75 g of glucose. If there is no acute metabolic decompensation, these criteria should be confirmed on a different day (59).

Both genetic and environmental factors have been implicated in the etiology of type 2 diabetes. There is a strong genetic predisposition for this type of diabetes although the exact genetic defects are not currently well defined (59). Among the risk markers for the disease are older age, obesity, minority ethnicity, family history, and lower socioeconomic status (81). Along with overall obesity, fat distribution (specifically, intra-abdominal fat distribution) predicts type 2 diabetes (22,23,51,72,78,101,132,138,169,177). Lifestyle factors that are implicated in the development of type 2 diabetes are physical inactivity and more inconsistently, diet, and parity (169). Type 2 diabetes is a dynamic disease in which individuals often become more insulin deficient with time.

The pathophysiology of type 2 diabetes appears to involve defects in both insulin action (insulin resistance) and secretion (insulin deficiency) (149). Insulin resistance is manifested by decreased insulin-mediated storage of glucose as glycogen in the liver and muscle. At the cellular level muscle glucose transporters (GLUT 4) may not be normally translocated from cytoplasm to plasma membrane although GLUT 4 protein and mRNA are normal (47,105). Insulin receptor substrate (IRS) phosphorylation is an important intermediary step in this process and may play a central role (91). IRS-1 is a cytoplasmic protein with multiple phosphorylation sites. After stimulation by insulin, it serves as a docking protein that facilitates phosphorylation of other intracellular proteins such as phosphatidylinositol kinase (PI 3-kinase). PI 3-kinase may be an important effector in the pathway by which GLUT 4 transporters are inserted into the plasma membrane. Abnormalities in IRS-1 (91) or other insulin receptor substrates have been postulated to be involved in insulin resistance. A separate defect in glycogen synthesis may also exist (170) and be found in nondiabetic relatives of persons with type 2 diabetes (188). These defects in insulin action may be either genetic or acquired through such factors as abdominal obesity. Chronic hyperglycemia and increased free fatty acid (FFA) levels may also contribute to acquired insulin resistance (147). Each of these may cause decreased muscle glucose transport and phosphorylation and are reversible (11). Insulin secretion is abnormal in type 2 diabetes with the first phase of insulin release generally being absent (145). Hyperglycemia alone may further inhibit insulin secretion. This concept has been termed glucose toxicity (213). Elevated products of fat metabolism may also impair beta cell function.

The goal of treatment in type 2 diabetes is to achieve and maintain near-normal blood glucose levels and optimal lipid levels, in order to prevent or delay the microvascular, macrovascular, and neural complications (52). Because exercise improves insulin sensitivity (diminishes resistance), it is a logical treatment modality. Exercise also modifies lipid abnormalities and hypertension. It, along with medical nutrition therapy, is an important component of obesity management. Use of oral antidiabetic agents and/or insulin may also be required to achieve normal glucose levels. These oral medications and insulin are described in reference 2. The aim of this position stand is to provide appropriate background and recommendations for safe and effective participation in physical activity by those with type 2 diabetes. Additionally, physical activity is an underutilized mode of therapy for type 2 diabetes, often due to lack of understanding. This position stand provides a breadth and depth of information that should facilitate understanding and use of exercise in the management of type 2 diabetes. For information about a wider range of specific sports and diabetes, the reader is referred to the Health Professionals Guide to Diabetes and Exercise (6).

Acute Effects of Exercise/Physical Activity

Physical activity is one of the principal therapies to acutely lower blood glucose in type 2 diabetes due to its synergistic action with insulin in insulin-sensitive tissues. Abnormal insulin secretion and peripheral insulin resistance (38) are primary factors that influence the acute effects of physical activity on metabolic responses in those with type 2 diabetes. In addition, oxygen delivery to peripheral tissues in type 2 diabetic individuals may be impaired during acute bouts of graded exercise (82,102), as the rate of oxygen consumption during submaximal and maximal work loads is significantly lower than age- and activity-matched persons without diabetes (94,151). Hence, functional capacity of those with type 2 diabetes is frequently lower than age-matched nondiabetic counterparts (102,161,164). Acute bouts of physical activity can favorably change abnormal blood glucose and insulin resistance (113).

Glucose levels. Most obese, type 2 diabetic individuals exhibit decreases in blood glucose after mild-to-moderate exercise (85,107,131,186). The magnitude of decrease in blood glucose is related to the duration and intensity of physical activity (138) and is further modified by preexercise glucose level and novelty of the activity.

Blood glucose reduction during physical activity is attributed to an attenuation of hepatic glucose production, whereas muscle glucose utilization increases normally (29,102,131). Reduced hepatic glucose production may include a negative feedback mechanism associated with sustained insulin levels during exercise and elevated glucose levels before activity.

Mild-to-moderate intensity exercise lowers blood glucose, and this effect is sustained into the postexercise period (85,131). Thus, mild-to-moderate intensity exercise is recommended to facilitate glucose reductions in those with type 2 diabetes. Blood glucose response to moderate exercise in lean, type 2 diabetic individuals is highly variable (87) and is not as predictable as in their obese counterparts. Such variability during exercise is related to impaired feedback control of hepatic glucose regulation and may be due to a defective nonpancreatic glucoregulatory mechanism. During short-term, high-intensity exercise, blood glucose frequently increases in obese, type 2 diabetic individuals who have hyperinsulinemia and remains elevated for about 1 h postexercise due to counter-regulatory hormone increase (102).

Insulin resistance. Insulin resistance is a frequent abnormality in type 2 diabetes (8,10). Insulin resistance reduces insulin-mediated glucose uptake in those with early stage type 2 diabetes by 35–40% of the level of glucose uptake in individuals who do not have diabetes (38,46). Insulin-mediated glucose uptake occurs primarily in skeletal muscle and is directly related to the amount of muscle mass, and inversely associated with fat mass (212). Some studies (35,38,48), but not all (154), show that exercise increases peripheral and splanchnic insulin sensitivity in those with type 2 diabetes. This increased sensitivity persists from 12 up to 24 h postexercise. Moreover, the insulin dose-response curve is not fully normalized by an acute bout of activity (35). There is no consensus regarding the effects of high intensity exercise on insulin sensitivity in persons with type 2 diabetes, as some (33) have found improved insulin sensitivity regardless of exercise intensity, whereas others (154) have shown insulin resistance for up to 60 min after high-intensity work. Such disparate findings of exercise intensity on insulin sensitivity can be partly explained by: 1) the different methods of assessing insulin sensitivity, including oral glucose challenge or insulin clamp technique; 2) the intensity of exercise administered; and/or 3) the heterogeneity of those with type 2 diabetes and their respective responses to acute exercise. The effect of an acute bout of exercise on insulin action is lost within a few days (79,164), and the benefit of a single bout of physical activity is short-lived for persons with type 2 diabetes. Thus, regular activity performed at a low-to-moderate intensity is recommended to lessen insulin resistance in type 2 diabetic individuals.

Most studies examining the effects of acute exercise on insulin sensitivity and glucose disposal in type 2 diabetes have included relatively small sample sizes and have not adequately distinguished the impact of physical activity among therapies, including medical nutrition therapy alone, oral antidiabetic medications, and/or insulin. Further research is needed regarding exercise-related changes in insulin sensitivity in the heterogeneous make-up of type 2 diabetes to more clearly understand the acute impact of physical activity on insulin resistance.

Chronic Effects of Exercise/Physical Activity

Genetic factors associated with insulin resistance and impaired glucose tolerance may result in low initial fitness and a reduced capacity to adapt to physical training (21,43,53). There is evidence of a reduced functional capacity in healthy individuals at high risk for development of type 2 diabetes even before the appearance of glucose intolerance (136). It is well established that patients with a diagnosis of type 2 diabetes have low $\dot{V}O_{2max}$ values when compared with healthy age-matched controls (58,94,151). Specific pathogenic mechanisms such as hyperglycemia, low capillary density, alterations in oxygen delivery, increased blood viscosity, or presence of vascular and neuropathic complications may also contribute to the decreased $\dot{V}O_{2max}$.

Regular physical activity promotes beneficial physiological changes in those with type 2 diabetes (84,97,98, 107,113,135,163,187,195,196,198,200,215), including lower resting and submaximal heart rate; increased stroke volume and cardiac output; enhanced oxygen extraction; and lower resting and exercise blood pressure (27,41,61,65,106,113,135). Those with type 2 diabetes are at increased risk for several cardiovascular risk factors, including hypertension and dyslipidemia (7,10,58,93,158,167). Thus, therapy to control glucose levels and reduce long-term complications should focus on behavioral interventions that include a physically active lifestyle.

Hypertension. Essential hypertension is a common cardiovascular risk factor occurring in over 60% of persons with type 2 diabetes (10). The efficacy of physical activity to favorably alter blood pressure is well-documented in those without diabetes (5,187) and is commonly stated as an outcome of physical activity participation in those with type 2 diabetes. Some studies (110,165), but not all (171), have observed that regular physical activity lowers blood pressure in persons with type 2 diabetes. To date, there is a paucity of studies specifically investigating the effect of physical activity on lowering blood pressure in persons with type 2 diabetes. Further research to more clearly identify blood pressure response to exercise in those with type 2 diabetes is needed.

Metabolic control: glucose control and insulin resistance. Aerobic power is inversely related to modest, favorable changes in glycosylated hemoglobin (e.g., HbA1 or HbA1c) and/or glucose tolerance (29,82,110,112,152,157,161,162,186, 190). In these studies, duration of physical training ranged from 6 wk to 12 months, and improved glucose tolerance was shown in early stage type 2 diabetes with as little as seven consecutive days of training (154). Some studies (131,171,192) have shown that mild-to-moderate physical training ranging from 12 wk up to 2 yr did not improve glucose control in type 2 diabetic subjects. Also, older diabetic individuals (e.g., over 55 yr) may not evince the same exercise-induced blood glucose changes as usually occur in younger counterparts (215). Favorable changes in glucose tolerance usually deteriorate within 72 h of the last exercise bout in those with type 2 diabetes (164) and are a reflection of the last individual exercise bout, rather than training *per se* (107,108,154). Hence, *regular* physical activity is recommended for persons with type 2 diabetes to sustain glucose-lowering effects.

A strong inverse relationship has been shown to exist between physical fitness and mortality due to all causes (26,27). Furthermore, major reductions in all-cause death rates are apparent with only modest increases in $\dot{V}O_{2max}$, especially for those at the lowest levels of fitness. This finding is especially important in type 2 diabetes as $\dot{V}O_{2max}$ values of 6 METs (metabolic equivalent) and less are common in these patients. Kohl et al. (106) demonstrated a similar inverse relationship between fitness and mortality across levels of glycemic control. Although risk of death increases with less-favorable glycemic status, the adverse impact of hyperglycemia on mortality appears to be reduced with increased fitness.

In some people with type 2 diabetes, insulin-mediated glucose disposal is improved after a period of physical training (29,84,108,112,152,186). After physical training, insulin sensitivity of both skeletal muscle and adipose tissue can improve with or without a change in body composition (84,108,124,189). This effect is transient and, as observed in glucose tolerance, deteriorates within 72 h (164). Consequently, regular physical activity is imperative for those with type 2 diabetes to sustain improved insulin sensitivity.

Lipids and lipoproteins. Increased aerobic power of people with type 2 diabetes is related to a less atherogenic lipid profile, which may lessen the accelerated rate of atherosclerosis and related mortality rate (106). Some studies found that after physical training, those with type 2 diabetes showed desirable changes in triglycerides (18,19,154, 155,156,157,205), total cholesterol (18,19,154,157,192), and high-density lipoprotein (HDL)-cholesterol:total cholesterol ratio (19,192), whereas others studies have found no change (114,171). A single study found that physical training significantly increased HDL-cholesterol and lowered low-density lipoprotein (LDL)-cholesterol in exercising versus control type 2 diabetic subjects (156). Also, some research (18,19,206) suggests that favorable triglyceride and cholesterol reduction in persons with type 2 diabetes is best achieved through weight loss, even though training-induced changes in blood lipids are independent of body weight (215).

Intensity, duration, and frequency of exercise training may influence lipid and lipoprotein changes. The inclusion of nutrition advice, counseling, or behavioral intervention to aid in lowering dietary saturated fat and body weight can also influence the magnitude of lipid changes in those with type 2 diabetes participating in physical training. Clearly, more research that examines nutrition therapy and exercise-induced lipid alterations in type 2 diabetes is needed.

Weight loss/maintenance. Exercise and medical nutrition therapy are essential for the initial treatment of type 2 diabetes and, when drug therapy is needed, for maintaining efficacy of drug therapy. Moderate weight loss (~10–15% or 4.5–9.1 kg) can assist in achieving metabolic goals (74,202,209). Nutrition therapy and regular exercise combined are more effective than either alone in achieving moderate weight reduction and thereby improving metabolic control (153,176,205,206). Weight loss leads to a decrease in insulin resistance and may be most beneficial early in the progression of type 2 diabetes when insulin secretion is still adequate.

Exercise also results in preferential mobilization of upper body fat (134). Visceral adipose tissue correlates significantly with hyperinsulinemia and is negatively associated with insulin sensitivity (25). Visceral fat represents a significant source of FFAs which may be oxidized in preference to glucose, resulting in hyperglycemia (140). Loss of visceral fat may be an important benefit of exercise as reduction of abdominal obesity leads to significant improvement in metabolic indices (213). Furthermore, abdominal obesity is a major risk factor for cardiovascular disease (24) and the development of type 2 diabetes (113).

Persons with type 2 diabetes, however, are often not able to exercise at a level that is required for significant weight loss to occur and body weight and body fat losses with exercise alone are often reported to be small. To improve body weight and body composition, regular exercise at an intensity of about 50% $\dot{V}O_{2max}$, five times or more per week, for about 1 h per session sustained for years would appear to be necessary (34). Therefore, it is important for health professionals to guard against unrealistic expectations of quick or easy weight loss in individuals beginning an exercise program.

Although the mechanism is still unclear, exercise seems to be effective in promoting long-term weight loss and has consistently been one of the strongest predictors of long-term weight control (116). Exercise is, therefore, a valuable adjunct measure along with food changes in the long-term management of weight. Furthermore, individuals who exercise may adhere better to nutritional advice. Physical activity may improve mood and self-esteem and as a result contribute to better control of food intake (164).

Psychological issues. The impact of diabetes on lifestyle and health, and the psychosocial adjustments to diabetes required by those with type 2 diabetes in later life may have important consequences on perceived stress, glucose control, and psychological health (73,117,182). Diabetic complications are more prevalent in those with long-standing type 2 diabetes (8) and require increased psychosocial adjustments (210). Diabetic complications contribute to perceived stress of disease management (185) and affective disorders, especially depression (66). Thus, therapy for those with type 2 diabetes should include social or family support systems that assist in facilitating adherence to a recommended treatment plan.

There are presumed physiological and psychological benefits of regular exercise in those without diabetes; reduced stress response to psychosocial stimuli (96), lessened sympathetic nervous system activation to cognitive stress (174), favorable reductions in depression (191), heightened self-esteem (173), and reduced emotional perturbations associated with life's stressful events (116,127). Such benefits in type 2 diabetes have received little attention, yet appear to have importance relative to augmenting perceived health and sense of self, and lessening the negative impact of stress and depression on disease management.

Given that diabetes management is emotionally stressful, particularly later in life for those with type 2 diabetes, and that this stress can influence glycemic control (116), regular physical activity can play a role in reducing stress, enhancing psychological well-being, and augmenting the quality of life for people with type 2 diabetes (191). Few studies have examined the effect of regular physical activity on various psychosocial, psychological, and stress-related outcomes in type 2 diabetes. The favorable changes associated with regular exercise in those without diabetes are presumed to occur in those with type 2 diabetes; however, future research is necessary to elucidate the efficacy of physical activity to evince such favorable psychological alterations.

The Role of Physical Activity in the Prevention of Type 2 Diabetes

Studies reviewed above examine the acute and chronic effects of physical activity on carbohydrate metabolism and glucose tolerance and provide the physiological reasons why a relationship between physical activity and glucose tolerance is biologically plausible. Through the years, from early observations to randomized clinical trials, the existence of a potential relationship between physical activity and type 2 diabetes has also been supported by the epidemiology literature. Relationships between physical activity and type 2 diabetes were suggested early by the fact that societies which had abandoned traditional lifestyles (which typically had included large amounts of habitual physical activity) had experienced major increases in type 2 diabetes (204). Indirect evidence of this phenomenon was also provided by the observation that groups of subjects who migrated to a more modern environment had more diabetes than their ethnic counterparts who remained in their native land (75,95,148) and that rural dwellers had a lower prevalence of diabetes than their urban counterparts (45,100,216,217). In these studies, differences in physical activity were suggested as partial explanations for the differences in diabetes prevalence.

Cross-sectional and retrospective epidemiological studies have provided more direct evidence that physical inactivity is significantly associated with glucose intolerance within populations. Groups of subjects with type 2 diabetes were found to be less active currently (50,100,109,183,184) and reported less physical activity over their lifetime (109) than individuals without diabetes. In addition, cross-sectional studies that have examined the relationship between physical activity and glucose intolerance in individuals without type 2 diabetes generally showed that blood glucose values after an oral glucose tolerance test (39,51,109,115,141, 168,199), as well as insulin values (51,60,115,128,150,199), were significantly higher in the less active compared to the more active individuals. More recently, the fact that a sedentary lifestyle may play a role in the development of type 2 diabetes has been demonstrated in prospective studies of male college alumni (80), female college alumni (64), registered nurses (120), male physicians (119), and middle-aged British men from the general population (142) and perhaps in metabolically obese, normal-weight individuals (those with hyperinsulinemia, insulin-resistance, hypertriglyceridemia, and premature coronary heart disease who are not obese) (159).

Similar to measures of physical activity, poor physical fitness, as determined by maximal oxygen uptake or as estimated by vital capacity, also appears to play a role in the development of type 2 diabetes (54,55). In addition, support that physical fitness may provide some protection against mortality in men at all levels of glucose tolerance (from those with normal blood glucose to those with type 2 diabetes) was demonstrated in middle-aged men (106).

Physical activity was a major part of the intervention strategy of a feasibility trial of diabetes prevention in 47–49 yr old men from Malmo, Sweden. Of those with impaired glucose intolerance at baseline, at least twice as many of those who did not take part in the treatment program had developed diabetes at the 5-yr follow-up compared with those that participated (55). A major limitation to this study was that participants were not randomly assigned to the intervention treatment groups.

The most promising of the studies, however, was a 6-yr clinical trial of diabetes prevention in Da Qing, China (139). At the beginning of the study, 577 individuals with impaired glucose tolerance were identified from a city-wide health screening and randomized by clinic into one of four groups: exercise only, diet only, diet plus exercise, and a control group. Individuals assigned to the exercise group were encouraged to increase their daily leisure physical activity by one unit, which in most cases was comparable to a 20-min brisk walk daily. The cumulative incidence of diabetes at 6 yr was significantly lower in the exercise intervention groups compared with the control group (exercise = 41%, exercise plus diet = 46%, diet = 44%, control = 68%) and remained significant even after adjusting for baseline differences in body mass index and fasting glucose (139).

A randomized, multicenter clinical trial of type 2 diabetes prevention that incorporates physical activity as one of the possible treatments is currently underway in the United States (49). In this clinical trial, physical activity is combined with dietary modification to comprise the lifestyle intervention.

Recommended Physical Activity Program for People with Type 2 Diabetes

Physical activity programs for those with type 2 diabetes without significant complications or limitations should include appropriate endurance and resistance exercise for developing and maintaining cardiorespiratory fitness, body composition, and muscular strength and endurance. In order to facilitate weight management and achieve health-related benefits, it is strongly recommended that individuals with type 2 diabetes expend a minimum cumulative total of 1000 $kcal \cdot wk^{-1}$ (27,61) in aerobic activity. The addition of a well-rounded resistance training program should be effective in improving muscular strength and endurance as well as in improving body composition by increasing or maintaining fat-free weight. Appropriate frequency, intensity, duration, and mode(s) of physical activity should be identified for persons with type 2 diabetes.

Frequency. Those with type 2 diabetes should engage in at least three nonconsecutive days and up to five physical activity sessions each week to improve cardiorespiratory endurance and achieve desirable caloric expenditure (4). Recently, the U.S. Surgeon General (187) recommended that physical activity should be performed most, if not all days of the week, to effect favorable health-related benefits, such as weight loss, blood pressure reduction, and favorable lipid and lipoprotein changes. Given that the acute effect of a single exercise bout on blood glucose levels is less than 72 h (90,196), those with type 2 diabetes must participate in regular physical activity to lower blood glucose. Those with type 2 diabetes taking insulin may prefer to participate in daily physical activity, in order to lessen the difficulty of balancing caloric needs with insulin dosage. Moreover, obese diabetic individuals may need to participate in daily physical activity to maximize caloric expenditure for effective weight management (4).

Intensity. For the majority of persons with type 2 diabetes, low-to-moderate intensity physical activity (of 40–70% $\dot{V}O_{2max}$) is recommended to achieve cardiorespiratory and metabolic improvements. Favorable metabolic changes (e.g., blood glucose reduction, and increased insulin sensitivity and metabolic clearance rate) usually occur after regular physical activity performed at a low-to-moderate intensity (31,140,206,211), whereas others (29,110,164) have shown favorable metabolic changes with higher-intensity exercise (e.g., 70–90% of $\dot{V}O_{2max}$) as well. Although a low intensity level is adequate to facilitate metabolic changes (202), it may not meet the recommended minimum threshold of exercise intensity (e.g., $\geq$ 50% of $\dot{V}O_{2max}$) for improving cardiorespiratory endurance (4). Most importantly, implementing low-to-moderate intensity activities for persons with type 2 diabetes minimizes the risks and maximizes the health benefits associated with physical activity for this population. Moreover, the lower intensity activity affords a more comfortable level of exertion and enhances the likelihood of adherence, while lessening the likelihood of musculoskeletal injury and foot trauma, particularly when weight-bearing activity is recommended (68).

Monitoring the intensity of physical activity in persons with type 2 diabetes may require the use of heart rate or ratings of perceived exertion (RPE) (4). Although a percentage of heart rate reserve (50–85%) or maximal heart rate (60–90%) is commonly used to identify exercise intensity for nondiabetic individuals, those with type 2 diabetes may develop autonomic neuropathy (192), which affects the heart rate response to exercise. Consequently, using heart rate as the only means to monitor intensity may be unsuitable for some with type 2 diabetes. A more appropriate adjunct to gauge the intensity of physical activity may be to use the RPE scale, especially in those who do not require specific heart rate limits (4). It is imperative that those using this scale become familiar with its use (e.g., matching description of level of perceived effort with a corresponding number) for proper implementation.

Duration. The duration of physical activity for persons with type 2 diabetes is directly related to the caloric expenditure requirements and inversely related to the intensity. Initially, those with type 2 diabetes should engage in physical activity for 10–15 min each session (68). Ideally, it is recommended that the time of the physical activity session be increased to at least 30 min to achieve the recommended energy expenditure (4). Also, physical activity can be divided into three 10-min sessions, whereby 30 min of physical activity is accumulated in a single day to account for the necessary energy expenditure (27). As stated earlier, when weight loss is a primary goal, the intensity needs to be low-to-moderate (50% $\dot{V}O_{2max}$) and the duration needs to be incrementally increased to approximately 60 min (30).

Mode. The recommended types of physical activities for persons with type 2 diabetes are those that afford greater control of intensity, have little interindividual variability in energy expenditure, are easily maintained, and require little skill (4). Combined with personal interests and goals, the mode of physical activity is important to aid in motivating the person with type 2 diabetes to begin physical activity, as well as to sustain a life-long physical activity habit. The mode of physical activity dictates that the level of energy expended and/or improvement in cardiorespiratory endurance be directly influenced by the amount of muscle mass used over the time of activity, as well as the rhythmic and aerobic nature of the activity. For those with type 2 diabetes, it is important to identify a mode of physical activity that can safely and effectively maximize caloric expenditure. Walking is the most commonly performed mode of activity for those with diabetes (63) and is the most convenient low-impact mode of physical activity. However, because of complications or coexisting conditions such as peripheral neuropathy or degenerative arthritis, those with type 2 diabetes may require alternative modes that are non-weight-bearing activities (e.g., stationary cycling, swimming, aquatic activities) or alternate between weight bearing and non-weight-bearing activities (4).

Resistance training has the potential to improve muscular strength and endurance, enhance flexibility, enhance body composition, and decrease risk factors for cardiovascular disease (143,175,179,182). In nondiabetic subjects, resistance training has resulted in improvements in glucose tolerance and insulin sensitivity (86,130,160). Resistance training appears to prevent loss of, and may even increase, muscle mass during and after energy restriction (15,34,67). Treuth et al. (185) were able to demonstrate that intra-abdominal obesity was reduced after 16 wk of moderate-intensity resistance training. There are limited data on the use of resistance training in individuals with type 2 diabetes (56,62), but results appear to be consistent with the findings in nondiabetic subjects mentioned above.

It is recommended that resistance training at least 2 $d \cdot wk^{-1}$ should be included as part of a well-rounded exercise program for persons with type 2 diabetes whenever possible. A minimum of 8–10 exercises involving the major muscle groups should be performed with a minimum of one set of 10–15 repetitions to near fatigue. Increased intensity of exercise, additional sets, or combinations of volume and intensity may produce greater benefits and may be appropriate for certain individuals. More detailed information for developing the resistance exercise training plan for people with diabetes is available (83). All persons with type 2 diabetes should be carefully screened before beginning this type of training and should receive proper supervision and monitoring. Caution should be used in cases of advanced retinal and cardiovascular complications. Modifications such as lowering the intensity of lifting, preventing exercise to the point of exhaustion, and eliminating the amount of sustained gripping or isometric contractions should be considered in these patients.

Rate of progression. The rate of increasing physical activity for those with type 2 diabetes is dependent upon several factors, including age, functional capacity, medical and clinical status, and personal preferences and goals (4,68,201). Moreover, initial changes in progression should focus on the frequency and duration of physical activity, rather than intensity, in order to provide a safe activity level that can be performed without undue effort and to increase the likelihood of sustaining the activity habit (4,208). Initially, it is recommended that those with type 2 diabetes engage in physical activity at a comfortable level (RPE 10–12) for about 10–15 min at a very low intensity at least 3 times per week and preferably 5 times per week (187,207). Duration of physical activity should be gradually increased to accommodate the functional capacity and clinical status of the person with type 2 diabetes. Given that older age and obesity are common elements of type 2 diabetes (8,10), a longer period of time may be necessary for the older and/or obese person to adapt to a recommended physical activity program (4,200). After the desired duration of activity is achieved, any increase in intensity should be small and approached with caution to minimize the risk of undue fatigue, musculoskeletal injuries, and/or relapse.

Limitations. The feasibility and efficacy of using physical activity as treatment for type 2 diabetes has been questioned for many years (27). Motivation is difficult and drop-out rates are often very high. Those with type 2 diabetes often find endurance exercise to be uncomfortable. Insulin-resistant subjects, as well as those with type 2 diabetes, have an increased number of type IIb muscle fibers, a low percentage of type I fibers, and a low capillary density (28,29,31,122,137). These muscle fiber composition abnormalities may affect tolerance for aerobic activity. The intensity of exercise at the anaerobic threshold is also lower in subjects with type 2 diabetes (19,32). Care should be taken to keep exercise intensity at a comfortable level in the initial periods of training and should progress very cautiously as tolerance for activity improves.

Risks and Complications of Exercise

Acute glycemic responses. Moderate-intensity exercise increases glucose uptake by 2–3 $mg \cdot kg^{-1} \cdot min^{-1}$ above usual requirements (70-kg person: 8.4–12.6 $g \cdot h^{-1}$ of exercise). During high-intensity exercise, glucose uptake increases by 5–6 $mg \cdot kg^{-1} \cdot min^{-1}$; however, exercise of this intensity cannot usually be sustained for long intervals (201).

Adequate and appropriate nutrition is important for any person engaging in physical activity. Fatigue can result from deficiencies of oxygen, fluids, or fuel, which can occur separately or in combination. Carbohydrate is needed during events lasting longer than 60–90 min (44,197) as well as after exercise to replenish muscle glycogen stores (89). Fluid intake is essential. For exercise lasting up to 1 h, plain water is usually the best beverage, but for exercise lasting longer, water and extra carbohydrate are needed. Six to 8% carbohydrate solutions are absorbed better and cause less stomach distress than regular soft drinks and fruit juices, which are 13–14% carbohydrate solutions (111).

Hypoglycemic reactions in connection with exercise in persons with type 2 diabetes are rare, occurring mainly in persons being treated with sulfonylurea oral medications and/or insulin and participating in unusually strenuous or prolonged exercise. Instruction on appropriate treatment of hypoglycemia in those with type 2 diabetes who use these medications is necessary. Blood glucose regulation during exercise in the person with type 2 diabetes controlled by nutrition therapy alone is not significantly different from that in persons without diabetes. During mild-to-moderate exercise, elevated blood glucose concentrations fall toward normal but do not reach hypoglycemic levels. There is no need for supplementary food intake before, during, or after exercise, except when exercise is exceptionally vigorous and of long duration as explained above. In this case, extra food may be beneficial just as it is in the person who does not have diabetes.

To minimize the occurrence of low blood glucose, it is imperative to understand the relationship of the exercise bout to the: (a) time when medications were taken (e.g., time of oral antidiabetic medications, or time and site of insulin injection); (b) antecedent and postexercise nutrition; and (c) last blood glucose assessment. The timing of insulin injection should be at least 1 h before the onset of exercise and preferably in a nonexercising or nonactive area. (Use of a nonexercising area does not guarantee prevention of low blood glucose.) Depending on the duration and intensity of exercise, the insulin dose may need to be modified. Insulin dose adjustments must be made on an individual basis and should be done in consultation with appropriate members of the health care team.

Self blood glucose monitoring is recommended for those with type 2 diabetes who engage in physical activity, especially during the initial activity sessions (4,68). Moreover, glucose monitoring is appropriate before and after an exercise bout. Given the knowledge and understanding of glucose levels, persons with type 2 diabetes, in consultation with their health care professional, can take appropriate action by reducing medications before exercise or increasing carbohydrate consumption (~15 $g \cdot h$) before or after exercise to reduce the likelihood of hypoglycemia. Adjustment to medications is preferable over increasing caloric intake to prevent hypoglycemia in those trying to reduce body weight.

Long-term complications. Although macrovascular and microvascular complications are prevalent in type 2 diabetes (10,13), their existence is not an absolute contraindication for physical activity. However, the risk of exacerbating specific complications and provoking musculoskeletal injuries in persons with type 2 diabetes is increased with physical exertion (57,198). Thus, there are physical activity precautions for all persons with type 2 diabetes, and limitations for those who have diabetic complications (215).

Before commencing exercise, those with type 2 diabetes should have a thorough physical exam to assess the presence of macro- and/or microvascular complications, and obtain physician approval to ensure that a safe and effective individualized activity program is developed (4,68). Initially, medical approval should evaluate glucose control (e.g., HbA1c), physical limitations with respect to joint immobility common to diabetes, prescribed medications, and special considerations with reference to the type and severity of complications (see Table 1). Given the age of the person and duration of diabetes, the physician may recommend that a stress test be performed before safely participating in an exercise program. For those with type 2 diabetes that are

TABLE 1. Evaluation before starting an exercise program.

Evaluate glycemic control
Subject may need modification of medication or carbohydrate ingestion if hypoglycemia is a problem
Severe hyperglycemia may be worsened with intense exercise
Are complications present?
Is the subject known to have cardiovascular disease or is he/she at high risk?
Is the subject at risk for injury due to peripheral neuropathy?
Is diabetic renal disease present? High intensity aerobic or resistance exercise may worsen progression.
Does the subject have retinopathy which will be worsened by activities which increase ocular pressure, e.g., resistance training?

≥ 35 yr of age, it is recommended that a stress test be conducted before participating in most physical activity (4). The rationale for recommending a stress test electrocardiogram on persons who meet this age criterion is to assess cardiovascular and respiratory systems, as the risk for macrovascular disease is increased in type 2 diabetes (9). Moreover, the stress test electrocardiography will identify target heart rate limits within which the person with or without autonomic neuropathy can safely exercise. Additionally, physical exertion may induce a recognizable hypertensive response in some with diabetes (28). Exercise-induced hypertension can be identified during a stress test and avert abnormal blood pressure excursions during normal physical activity by identifying appropriate physical activities (e.g., intensity, or selection of activity). For additional information on the chronic complications of diabetes and exercise, the reader is referred to the *Health Professional's Guide to Diabetes and Exercise* (6).

Vascular disease. Diabetes is a major risk factor for the development of cardiovascular disease. The risk of myocardial infarction is 50% greater in diabetic men and 150% greater in diabetic women (203). The propensity for arrhythmias during exercise and the ischemic response to exercise should be evaluated. Moderate intensity activity (60–80% of maximum heart rate, 50–74% $\dot{V}O_{2max}$) is usually recommended for those with known coronary artery disease without ischemia or significant arrhythmias (14,144). In those with angina, the target heart rate should be 10 beats or more below the ischemic threshold (3). In patients without angina, the ischemic threshold should be determined by an exercise electrocardiogram.

Autonomic neuropathy interferes with heart rate regulation by depressing maximal heart rate and blood pressure, and increasing resting heart rate (92). Early warning signs of ischemia may also be absent in those with autonomic neuropathy. There is increased risk for exercise-induced hypotension after strenuous activity in persons with autonomic neuropathy (218). Moreover, persons with autonomic dysfunction exhibit a lower fitness level and fatigue at relatively low workloads (193,194). Consequently, physical activity for these persons should focus upon low-level daily activities, where mild changes in heart rate and blood pressure can be accommodated (69). Any physical activity program for persons with autonomic neuropathy should be viewed with caution and should have physician approval. Moreover, it is recommended that type 2 diabetic individuals with autonomic neuropathy undergo a diagnostic stress test to rule out the presence of cardiac function abnormalities and identify safe limits of physical activity (217).

Although weight-bearing activity is preferred (4), type 2 diabetic individuals with peripheral vascular disease (PVD) who have claudication may choose to perform low- or non-weight-bearing activity (e.g., swimming, aquacize, stationary cycling) or alternate between different types of weight-bearing versus non-weight-bearing activities. Physical activity must be performed to pain tolerance with intermittent rest during each session of activity (4). Moreover, peripheral neuropathy in the presence of PVD may preclude the use of weight-bearing activities, due to the possibility of foot trauma (193).

Peripheral neuropathy. This form of neuropathy affects the extremities, especially the lower legs and feet (70,71,162), and results in loss of distal sensation that can lead to musculoskeletal injury or to infection. Non-weight-bearing activities should be performed by persons with peripheral neuropathy in order to mitigate irritation and/or trauma to the lower legs and feet (69,193). As a pragmatic recommendation, proper footwear for all weight-bearing activities of daily living is very important to minimize the likelihood for undetected sores, which can evolve into an infection if unnoticed. The feet should be examined daily by the person with diabetes and at each physician visit.

Nephropathy. Increased blood pressure is a common precursor and is related to worsening kidney disease (69); however, it remains to be proven whether exercise-induced blood pressure changes exacerbate the progression of nephropathy. Although few studies have examined exercise-induced microalbuminuria in persons with type 2 diabetes (133), physical activity may assist in controlling factors (e.g., blood glucose and blood pressure) related to the progression of nephropathy in those with type 2 diabetes (88,103). Persons with nephropathy should avoid activities which cause the systolic blood pressure to rise to 180–200 mm Hg (e.g., performing Valsalva maneuver, high-intensity aerobic or strength exercises), as increases in systemic pressure could potentially worsen the progression of this disease. Those with later stages of renal disease should participate in lower intensity physical activities (~ 50% $\dot{V}O_{2max}$) with physician approval, as cardiorespiratory and health-related benefits are accrued at this lower level of training. It is recommended that exercise testing be conducted to identify safe intensity limits for those with type 2 diabetes who have advanced nephropathy (32).

Retinopathy. Although exercise increases systemic and retinal blood pressures, there is no evidence that physical activity acutely worsens the retinopathy present in diabetes (195). Bernbaum and associates (20) found that type 1 and type 2 diabetic individuals with proliferative retinopathy who engaged in a low-intensity training program improved cardiovascular function by 15%. Precautions were taken to limit systolic blood pressure to 20–30 mm Hg above baseline during each training session. Thus, in a well-supervised environment, low-intensity aerobic activity can be safely performed by persons with retinopathy (194,216). Those with type 2 diabetes should be evaluated to determine the degree of retinopathy. If retinopathy is present, they need to be cautioned about engaging in activities that cause blood pressure to increase dramatically, such as head-down or jarring activities or those with arms overhead (194,195,216).

Adoption and Maintenance of Exercise by Persons with Diabetes

In spite of substantial evidence showing health benefits of long-term exercise for persons with diabetes (195), it is rarely incorporated as an integral part of therapy (64). Moreover, adherence to prescribed exercise programs is frequently poor (63,129).

Little is known about factors likely to affect exercise adoption and maintenance. Two theoretical models are useful for understanding these factors: the transtheoretical model (i.e., stages of change theory) (146) and self-efficacy theory (17). The transtheoretical model postulates that persons are at different cognitive stages with regard to their readiness to adopt and maintain a particular behavior, such as exercise, ranging from precontemplation and contemplation to preparation, action, and maintenance. The implication of this stage-based model is that interventions designed to encourage adoption of an exercise regimen must be responsive to the stage of readiness that the individual is currently in and focus effort on moving the individual through the stages (121).

Self-efficacy theory postulates that adoption of exercise is a function of judgment concerning ability to do exercise in relation to the probable benefits and costs associated with the activity (16,17). In this context, research has demonstrated that persons with previous exercise experience (42,179), and particularly previous success (125,126), have substantially higher exercise efficacy expectations. In addition, physical status may be of equal importance to developing exercise efficacy expectations among older adults who are more likely to have type 2 diabetes and also suffer from more physical limitations generally associated with age (125,126,146).

Outcome expectations are viewed as an important element of models of health behavior and may contribute significantly to one's motivations to adopt a particular behavior. A person's confidence for adopting a behavior is influenced in part by the extent to which belief of rewards are associated with that behavior (1). Thus, outcome expectations will have important implications for the form of information and education delivered by health care providers (172). This is supported by research which found that having a physician discuss the benefits of physical activity was a strong predictor of exercise adoption among African-American women (118).

Factors influencing the contemplation stage. Several factors should be addressed to help motivate the person in the contemplation stage to initiate an exercise program (123). First, the program must be viewed by the person as desirable and intrinsically reinforcing. Second, the activities recommended must be perceived as realistic and feasible. Third, strategies for avoiding the potential negative consequences of exercise, particularly those associated with diabetes must be taught.

The failure of persons with diabetes to engage in regular exercise is due, in part, to their outcome expectations. Many are not familiar with the benefits of exercise on their diabetes. Even when the benefits are known, health care professionals often describe exercise to their patients using a negative reinforcement paradigm, i.e., exercise is done to avoid the onset of punishment in the form of complications, not as an intrinsically enjoyable activity with health benefits. Oftentimes, attempts at exercise have resulted in physical discomfort, injury, or hypoglycemia, thus "demonstrating" that the costs outweigh the potential long-term benefits.

To engender a better outcome expectation, the rationale for the prescription of exercise should include discussion of the social, psychological, and general health benefits in the population as a whole, as well as particular benefits in persons with diabetes. Social benefits include participation of family members, peers, and participation in organized, community-based activities (123). Psychological benefits include reduction in stress, anxiety, and depression and increased feelings of well-being (166). Health benefits include improvements in glucose regulation, weight control, lipid profiles, hypertension, and increased work capacity (36,37,194,198).

To help address efficacy expectations, several key points should be emphasized:

- To benefit diabetes control, exercise needs to be part of a lifelong management program that starts gradually and works up to higher intensity.
- To sustain an exercise program, help the patient select one that reflects their goals, desires, and the availability of appropriate support.
- Teach the person with diabetes how to perform the selected activity so that he/she avoids discomfort, injury, and problems with his/her diabetes.
- Assure those with diabetes that they do not have to figure out how to set up an exercise program alone. There are health care professionals who can help them accomplish these goals.

Factors influencing the action stage. An important component to increasing exercise adoption is providing patients with specific exercise prescriptions. Frequently, the recommendation to exercise is a generic prescription with no specific instructions about what to do or how to do it. As a result, most persons with diabetes do not have a clear idea about what type of exercise will work best for their particular situation. Moreover, they are not given much guidance concerning how to adjust their diabetes regimen to safely exercise. As a result, they often choose activities without any reflection as to their suitability or safety. Discussing patients' answers to two simple questions can help them to more critically consider factors that can help or hinder their selection of an exercise method they are likely to enjoy. These questions are:

What are your goals for exercise? Finding out patients' goals for exercising will help them identify a method to achieve those goals (180). Their rationale may not reflect what the health care professional feels is most important but may result in achieving the same endpoint.

What types of physical activity are you doing or think you would like to do? This question is designed to help guide patients in selecting an activity they are motivated to do. If they do not have preferences, ask them to indicate their preference between the following options: a) long or short duration; b) high versus low intensity; c) exercising by themselves or with others; d) exercising at home or at a facility; e) exercising indoors versus outdoors; and f) a competitive or cooperative sport.

Their responses to these types of preference trade-offs will help them to more critically consider what is truly reinforcing to them. It will also help to provide suggestions as the suitability of a given activity and how they may best adapt their diabetes regimen to its demands.

The Ease of Access and Ease of Performance Index. Once the person with diabetes has narrowed down the possibilities, or even selected a specific exercise method, the reasonableness of the activity given their personal situation should be considered by reviewing the "Ease of Access" and Ease of Performance" index (123). These are self-assessments of how realistic the activity is for them given their life style.

Ease of Access Index. The Ease of Access addresses the question *"how easily can I engage in my activity of choice where I live?"* Many people have a tendency to begin an exercise program only to find that it's simply too difficult to participate on a regular basis for a variety of reasons that were either ignored, rationalized, or simply not considered before the program was begun. To determine their "ease of access" index for a given activity, ask the person to consider the following questions:

- Does it require special facilities and are these facilities available?
- Does it require special equipment and is this equipment available and affordable?
- Does it require special training or instruction and is this instruction readily available, scheduled at convenient times, easy to get to, and affordable?
- Does it require others to do it and can these partners always be found?
- Is it seasonal, and what can be done other times of the year?

Ease of Performance Index. If the exercise activity has an acceptable ease of access index, encourage evaluation in terms of its ease of performance index. The ease of performance index is an assessment of how suitable the activity is in terms of the person's physical attributes and life style. To determine the ease of performance index, have the person consider the following questions:

- Does the activity suit their physical attributes?
- Can he/she realistically integrate the chosen activity into his/her current lifestyle?
- Can he/she afford any costs associated with it?
- Does he/she have a good support network if needed for the activity?

Factors influencing the maintenance stage. There are several factors that health care professionals can use to help persons with diabetes maintain an exercise program. These include:

1. *Appropriate exercise and equipment to avoid injury.* The individual with diabetes should engage in a proper warm-up and follow a gradual build-up training schedule. Equally important is the use of proper equipment, especially footwear.

2. *Set realistic exercise goals.* Exercise goals should be precisely defined and realistically attainable. Goals should be defined by exercise behavior (e.g., walk 30 min three times per week) rather than by a desired outcome (e.g., lose 20 pounds). Smaller, step-wise goals for which success and progress can be observed should be encouraged.

3. *Set an exercise schedule in advance and stick to it.* Long-term habits are developed through practice. Moreover, a regular schedule makes diabetes regimen adjustments easier to establish thereby improving glycemic control.

4. *Use an exercise partner.* An exercise partner can help encourage and motivate an individual to maintain a training schedule. In addition, they may be of assistance in the event of a hypoglycemic episode.

5. *Encourage self-rewards.* Progressive rewards for reaching exercise goals can increase motivation to stay with an exercise program.

6. *Identify alternative exercise activities to reduce boredom.* Individuals who become bored with a single activity should be encouraged to select alternative activities that will help them remain active. The goal is to do *some* form of physical activity.

7. *Understand the difference between failure and backsliding.* For some individuals, any deviation from a schedule or not meeting the expectations is viewed as failure. It is important to help such individuals understand and accept off days as part of any long-term exercise program. When off days do occur, the concept of a "backslide," i.e., a temporary state, should be reinforced, and return to the regular schedule encouraged.

CONCLUSION

Physical activity affords significant acute and chronic benefits for those with type 2 diabetes. The benefits of chronic physical activity are more numerous than those of acute physical activity, emphasizing the need for regular participation by those with type 2 diabetes and those at risk for this form of diabetes.

Unfortunately, physical activity is underutilized in the management of type 2 diabetes. This may be due to lack of understanding and/or motivation on the part of the person with diabetes and lack of clear recommendations, encouragement, and follow-up by health care professionals. Several factors including muscle fiber composition, low capillary density, obesity, and older age require that physical activity be initiated at lower intensity/duration and be increased gradually to reduce risks and contribute to maintenance of physical activity by those with type 2 diabetes.

Attention to the patient's stage of readiness and factors that will encourage adoption and maintenance of regular physical activity are extremely important for successful use of physical activity as a therapeutic intervention. Health care professionals must address physical activity more seriously in this patient population because most people with type 2 diabetes have the potential to derive benefits from regular, moderate levels of physical activity.

ACKNOWLEDGMENT

This pronouncement was reviewed for the American College of Sports Medicine by members-at-large, the Pronouncements Committee, and by Paula Harper, R.N., C.D.E.; Edward S. Horton, M.D.; Neil Ruderman, M.D., D.Phil.; Stephen Schneider, M.D.; and Bernard Zinman, M.D., FACP, FRCP.

REFERENCES

1. Ajzen, I., and M. Fishbein. *Understanding Attitudes and Predicting Social Behavior*. Englewood Cliffs, NJ: Prentice-Hall, 1980.
2. Albright, A. L. *American College of Sports Medicine: Exercise Management for Persons with Chronic Diseases and Disabilities*. Champaign, IL: Human Kinetics, 1997, pp. 94–98.
3. American College of Sports Medicine. *Guidelines for Exercise Testing and Prescription*, 4th Ed. Philadelphia: Lea & Febiger, 1991.
4. American College of Sports Medicine. *Guidelines to Exercise Testing and Exercise Prescription*, 5th Ed. Philadelphia: Williams & Wilkins, 1995, pp. 206–235.
5. American College of Sports Medicine. Position stand: Physical activity, physical fitness, and hypertension. *Med. Sci. Sports Exerc.* 25:i–x, 1993.
6. American Diabetes Association. *The Health Professional's Guide to Diabetes and Exercise*, N. Ruderman and J. Devlin (Eds.). Alexandria, VA: American Diabetes Association, Inc., 1995, pp. 133–197, 265–305.
7. American Diabetes Association. Implications of the diabetes control and complications trial (position statement). *Diabetes Care* 22(Suppl. 1):S24–S31, 1999.
8. American Diabetes Association. *Diabetes: 1996 Vital Statistics*. Alexandria, VA, 1996, pp. 11–74.
9. American Diabetes Association. Role of cardiovascular risk factors in prevention and treatment of macrovascular disease in diabetes (Consensus Statement). *Diabetes Care* 16:72–78, 1993.
10. American Diabetes Association. *Medical Management of Type 2 Diabetes*, 4th Ed. Alexandria, VA, 1998.
11. American Diabetes Association. Insulin resistance (Consensus Statement). *Diabetes Care* 21:310–314, 1998.
12. American Diabetes Association. Diabetes mellitus and exercise (Position Statement). *Diabetes Care* 22(Suppl. 1):S49–S53, 1999.
13. American Diabetes Association. Role of cardiovascular risk factors in prevention and treatment of macrovascular disease in diabetes (Consensus Statement). *Diabetes Care* 12:573–79, 1989.
14. American Heart Association Writing Group. Exercise standards: a statement for health professionals from the American Heart Association (Special Report). *Circulation* 91:580–615, 1995.
15. Ballor, D. L., J. R. Harvey-Berino, P. A. Ades, J. Cryan, and J. Calles-Escandon. Contrasting effects of resistance and aerobic training on body composition and metabolism after diet-induced weight loss. *Metabolism* 45:179–183, 1996.
16. Bandura, A. Self-efficacy mechanism in psychobiologic functioning. In: *Self-efficacy: Thought Control and Action*, R. Schwarzer, (Ed.). Washington, DC: Hemisphere Publishing Corporation, 1992, pp. 78–86.
17. Bandura, A. *Social Foundations of Thought and Action.* Englewood Cliffs, NJ: Prentice Hall, 1986.
18. Barnard, R. J. L. Lattimore, R. G. Holly, S. Cherny, and N. Pritikin. Response of non-insulin-dependent diabetic patients to an intensive program of diet and exercise. *Diabetes Care* 5:370–374, 1982.
19. Barnard, R. J., E. J. Ugianskis, and D. A. Martin. The effects of an intensive diet and exercise program on patients with non-insulin-dependent diabetes mellitus. *J. Cardiopulm. Rehabil.* 12: 194–201, 1992.
20. Bernbaum, M., S. G. Alber, J. D. Cohen, and A. Drimmer. Cardiovascular conditioning in individuals with diabetic retinopathy. *Diabetes Care* 12:740–742, 1989.
21. Berntorp, K., K. F. Eriksson, and F. Lindgarde. The importance of diabetes heredity in lean subjects on insulin secretion, blood lipids and oxygen uptake in the pathogenesis of glucose intolerance. *Diabetes Res.* 3:231–236, 1986.
22. Björntorp, P. Abdominal obesity and the development of non-insulin-dependent diabetes mellitus. *Diabetes Metab. Rev.* 4:615–622, 1988.
23. Björntorp, P. Metabolic implications of body fat distribution. *Diabetes Care* 14:1132–1143, 1991.
24. Björntorp, P. Portal adipose tissue as a generator of risk factors for cardiovascular disease and diabetes. *Arteriosclerosis* 10:493–496, 1990.
25. Björntorp, P. Regional fat distribution: implications for type II diabetes. *Int. J. Obes. Suppl.* 16:519–527, 1992.
26. Blair, S. N., H. W. Kohl, R. S. Paffenbarger, D. G. Clark, K. H. Cooper, and L. W., Gibbons. Physical fitness and all-cause mortality: a prospective study of healthy men and women. *JAMA* 262:2395–2401, 1989.
27. Blair, S. N., H. W. Kohl, N. F. Gordon, and R. S. Paffenbarger. How much physical activity is good for health? *Ann. Rev. Public Health* 13:99–126, 1992.
28. Blake, G. A., S. R. Levin, and S. N. Koyal. Exercise induced hypertension in normotensive patients with NIDDM. *Diabetes Care* 13:799–801, 1990.
29. Bogardus, C., E. Ravussin, D. C. Robbins, R. R. Wolfe, E. S. Horton, and E. A. H. Sims. Effects of physical training and diet therapy on carbohydrate metabolism in patients with glucose intolerance and non-insulin-dependent diabetes mellitus. *Diabetes* 33:311–8, 1984.
30. Bouchard, C, J. P. Deprés, and A. Tremblay. Exercise and obesity. *Obes. Res.* 1:133–147, 1993.
31. Bourn, D. M., J. I. Mann, B. J. McSkimming, M. A. Waldron, and J. D. Wishart. Impaired glucose tolerance and NIDDM: does a lifestyle intervention program have an effect? *Diabetes Care* 17:1311–1319, 1995.
32. Braden, C. Nephropathy: advanced. In: *The Health Professional's Guide to Diabetes and Exercise*. Alexandria, VA: American Diabetes Association, 1995, pp. 177–180.
33. Braun, B., M. B. Zimmermann, and N. Kretchmer. Effects of exercise intensity on insulin sensitivity in women with non-insulin-dependent diabetes mellitus. *J. Appl. Physiol.* 78:300–306, 1995.
34. Bryner, RW, I. H. Ullrich, J. Sauers, et al. Effects of resistance vs. aerobic training with an 800 calorie liquid diet on lean body mass and resting metabolic rate. *J. Am. Coll. Nutr.* 18:115–121, 1999.
35. Burstein, R., Y. Esptein, Y. Shapiro, I. Cahruzi, and E. Karnieli. Effect of an acute bout of exercise on glucose disposal in human obesity. *J. Appl. Physiol.* 69:299–304, 1990.
36. Campaign, B. N., and R. M. Lampman. *Exercise in the Clinical Management of Diabetes*. Champaign, IL: Human Kinetics, 1994.
37. Campaigne, B. N., and R. Gunnarsson. The effects of physical training in people with insulin dependent diabetes mellitus. *Diabetic Med.* 5:429–433, 1988.

38. Caro, J. F., L. G. Dohm, W. J. Pories, and M. K. Sinha. Cellular alterations in liver, skeletal muscle, and adipose tissue responsible for insulin resistance in obesity and type 2 diabetes. *Diabetes/Metab. Rev.* 5:665–689, 1989.
39. Cederholm, J., and L. Wibell. Glucose tolerance and physical activity in a health survey of middle-aged subjects. *Acta Med. Scand.* 217:373–378, 1985.
40. Centers for Disease Control and Prevention. *National Diabetes Fact Sheet: National estimates and general information on diabetes in the United States*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, 1999, pp. 1–8.
41. Centers for Disease Control and Prevention and American College of Sports Medicine. Summary statement: workshop on physical activity and public health. *Sports Med. Bull.* 28:7, 1993.
42. Clark, D. O., D. L. Patrick, D. Grembowski, and M. Durham. Socioeconomic status and exercise self-efficacy in late life. *J. Behav. Med.* 18:355–376, 1995.
43. Colman, E., L. I., Katzel, J. Sorkin, et al. The role of obesity and cardiovascular fitness in the impaired glucose tolerance of aging. *Exp. Gerontol.* 30:571–580, 1995.
44. Coyle, E. F., J. M. Hagberg, B. S. Hurley, W. H. Martin, A. A. Ehsani, and J. O. Holloszy. Carbohydrate feeding during prolonged strenuous exercise can delay fatigue. *J. Appl. Physiol.* 55:230–235, 1983.
45. Cruz-Vidal, M., R. Costas, M. Garcia-Palmieri, P. Sorlie, and E. Hertzmark. Factors related to diabetes mellitus in Puerto Rican men. *Diabetes* 28:300–307, 1979.
46. Defronzo, R. A., D. Simonson, and E. Ferrannini. Hepatic and peripheral insulin resistance: a common feature of type 2 (non-insulin dependent) and type 1 (insulin dependent) diabetes mellitus. *Diabetologia* 23:313–319, 1982.
47. Dela, F., T. Ploug, A. Handberg, et al. Physical training increases muscle GLUT4 protein and mRNA in patients with NIDDM. *Diabetes* 43:862–865, 1994.
48. Devlin, J. T., M. Hirshman, E. D. Horton, and E. S. Horton. Enhanced peripheral and splanchnic insulin sensitivity in NIDDM men after a single bout of exercise. *Diabetes* 36:434–39, 1987.
49. Diabetes Prevention Program Research Group. The Diabetes Prevention Program: design and methods for a clinical trial in the prevention of type 2 diabetes. *Diabetes Care* 22:623–634, 1999.
50. Dowse, G. K., H. Gareeboo, P. Z. Zimmet, et al. High prevalence of NIDDM and impaired glucose tolerance in Indian, Creole and Chinese Mauritians. *Diabetes* 39:390–396, 1990.
51. Dowse, G. K., P. Z. Zimmet, H. Gareeboo, et al. Abdominal obesity and physical inactivity are risk factors for NIDDM and impaired glucose tolerance in Indian, Creole, and Chinese Mauritians. *Diabetes Care* 14:271–282, 1991.
52. Eastman, R. C., R. Silverman, M. Harris, J. C. Javitt, Y. P. Chiang, and P. Gorden. Lessening the burden of diabetes: intervention strategies. *Diabetes Care* 16:1095–1102, 1993.
53. Endre T., I. Mattiasson, U. L. Hulthen, F. Lindgarde, and G. Berglund. Insulin resistance is coupled to low physical fitness in normotensive men with a family history of hypertension. *J. Hypertens.* 12:81–88, 1994.
54. Eriksson K, and F. Lindgarde. Poor physical fitness, and impaired early insulin response but late hyperinsulinaemia, as predictors of NIDDM in middle-aged Swedish men. *Diabetologia* 39:573–579, 1996.
55. Eriksson K, and F. Lindgarde. Prevention of type 2 (non-insulin-dependent) diabetes mellitus by diet and physical exercise. *Diabetologia* 34:891–898, 1991.
56. Eriksson J., S. Taimela, K. Eriksson, S. Parvianen, J. Peltonen, and U. Kujala. Resistance training in the treatment of non-insulin-dependent diabetes mellitus. *Int. J. Sports Med.* 18:242–246, 1997.
57. Estacio, R. O., J. G. Regensteiner, E. E. Wolfel, B. Jeffers, M. Dickenson, and R. W. Schrier. The association between diabetic complications and exercise capacity in NIDDM patients. *Diabetes Care* 21:291–295, 1998.
58. Estacio R. O., E. E. Wolfel, J. G. Regensteiner, B. Jeffers, E. P. Havranek, S. Savage, and Schrier. Effect of risk factors on exercise capacity in NIDDM. *Diabetes* 45:79–85, 1996.
59. Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. A report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes Care* 20: 1183–1197, 1997.
60. Feskens, E. J., J. G. Loeber, and D. Kromhout. Diet and physical activity as determinants of hyperinsulinemia: the Zutphen elderly study. *Am. J. Epidemiol.* 140:350–360, 1994.
61. Fletcher, G. F., S. N. Blair, J. Blumenthal, C. Caspersen, B. Chaitman, and S. Epstein. Statement on exercise: benefits and recommendations for physical activity programs for all Americans. *Circulation* 86:340–344, 1992.
62. Fluckey J. D., M. S. Hickey, J. K. Brambrink J. K., K. K. Hart, K. Alexander, and B. W. Craig. Effects of resistance exercise on glucose tolerance in normal and glucose-intolerant subjects. *J. Appl. Physiol.* 77:1087–1092, 1994.
63. Ford, E. S., and W. H. Herman. Leisure-time physical activity patterns in the U.S. diabetic population: findings from the 1990 national health interview survey–health promotion and disease prevention supplement. *Diabetes Care* 18:27–33, 1995.
64. Frisch, R. E., G. Wyshak, T. E. Albright, N. L. Albright, and I. Schiff. Lower prevalence of diabetes in female former college athletes compared with nonathletes. *Diabetes* 35:1101–1105, 1986.
65. Galbo, H. *Hormonal and Metabolic Adaptation to Exercise*. New York: Thieme-Stratton, 1983, pp. 30–39, 69–86.
66. Gavard, J. A., P. J. Lustman, and R. E. Clouse. Prevalence of depression in adults with diabetes. *Diabetes Care* 16:1167–1178, 1993.
67. Geliebter A, M. M. Maher, L. Gerace, B. Gutin, S. B. Heymsfield, and S. A. Hashim. Effects of strength or aerobic training on body composition, resting metabolic rate, and peak oxygen composition in obese dieting subjects. *Am. J. Clin. Nutr.* 66:557–563, 1997.
68. Gordon, N. The exercise prescription. In: *The Health Professional's Guide to Diabetes and Exercise*. Alexandria, VA: American Diabetes Association, 1995, pp. 71–82.
69. Graham, C., and P. Lasko-McCarthey. Exercise options for persons with diabetic complications. *Diabetes Educator* 16:212–20, 1990.
70. Greene, D. A., A. A. F. Sima, J. W. Albers, and M. A. Pfeiffer. Diabetic neuropathy. In: *Diabetes Mellitus*, 4th Ed., H. Rifkin and D. Porte (Eds.). New York: Elsevier, 1990, pp. 710–755.
71. Greene, D. A., and M. J. Stevens. Diabetes peripheral neuropathy: introduction. *Diabetes Spectrum* 6:234, 1993.
72. Haffner, S. M., M. P. Stern, H. P. Hazuda, M. Rosenthal, J. A. Knapp, and R. M. Malina. Role of obesity and fat distribution in non-insulin-dependent diabetes mellitus in Mexican Americans and non-Hispanic whites. *Diabetes Care* 9:153–161, 1986.
73. Hamburg, B. A., L. F. Lipsett, G. E. Inoff, and A. L. Drash (Eds.). Behavioral and psychological issues in diabetes: proceedings of the national conference. NIH Publication No. 80–1993. Washington, D.C.: U.S. Government Printing Office, 1980.
74. Hansen, B. C. Dietary considerations for obese diabetic subjects. *Diabetes Care* 11:183–188, 1988.
75. Hara, H., T. Kawate, M. Yamakido, and Y. Nishimoto. Comparative observation of micro- and macroangiopathies in Japanese diabetics in Japan and U.S.A. In: *Diabetic Microangiopathy*, H. Abe and M. Hoshi (Eds.). Tokyo: University of Tokyo Press, 1983, pp. 377–391.
76. Harris, M. I. Classification, diagnostic criteria, and screening for diabetes. In: *Diabetes in America*, sponsored by the National Diabetes Data Group. NIH, NIDDK, Publication No. 95–1468, 1995, p. 10.
77. Harris, M. I. Classification, diagnostic criteria, and screening for diabetes. In *Diabetes in America,* sponsored by the National Diabetes Data Group. NIH, NIDDK, Publication No. 95–1468, pp.15–36, 1995.
78. Hartz, A. J., D. C. Rupley, R. D. Kalkhoff, and A. A. Rimm. Relationship of obesity to diabetes: influence of obesity and body fat distribution. *Prev. Med.* 12:351–357, 1983.
79. Heath, G. W., J. R. Gavin, J. M. Hinderliter, J. M. Hagberg, S. A. Bloomfield, and J. O. Holloszy. Effects of exercise and

lack of exercise on glucose tolerance and insulin sensitivity. *J. Appl. Physiol.* 57:1857–1864, 1983.
80. Helmrich, S. P., D. R. Ragland, R. W. Leung, and R. S. Paffenbarger. Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. *N. Engl. J. Med.* 325:147–152, 1991.
81. Hickey, M. S., J. O. Carey, J. L. Azevedo, et al. Skeletal muscle fiber composition is related to adiposity and in vitro glucose transport rate in humans. *Am. J. Physiol.* 268(3 Pt 1):E453–E457, 1995.
82. Holloszy, J. O., J. Schultz, J. Kusnierkiewicz, J. M. Hagberg, and A. A. Ehsani. Effects of exercise on glucose tolerance and insulin resistance: brief review and some preliminary results. *Acta Med. Scand.* 711(Suppl.):55–65, 1987.
83. Hornsby, W. G. Resistance training. In: *The Health Professional's Guide to Diabetes and Exercise*, N. Ruderman and J. T. Devlin (Eds.). Alexandria, VA: American Diabetes Association, 1995, pp. 83–88.
84. Horton, E. S. Exercise and physical training: effects on insulin sensitivity and glucose metabolism. *Diabetes/Metab. Rev.* 2:1–17, 1986.
85. Hubinger, A., A. Franzen, and A. Gries. Hormonal and metabolic response to physical exercise in hyperinsulinemic and non-hyperinsulinemic type 2 diabetics. *Diabetes Res.* 4:57–61, 1987.
86. Hurley, B. F., D. R. Seals, A. A. Ehsani, et al. Effects of high-intensity strength training on cardiovascular function. *Med. Sci. Sports Exerc.* 16:483–488, 1984.
87. Jenkins, A. B., S. M. Furler, D. G. Bruce, and D. J. Chisholm. Regulation of hepatic glucose output during moderate exercise in non-insulin-dependent diabetes. *Metabolism* 37:966–972, 1988.
88. John, L., P. S. S. Sunder Rao, and A. S. Kanagasabapathy. Rate of progression of albuminuria in type 2 diabetes. *Diabetes Care* 17:888–890, 1994.
89. Ivy, J. L., S. L. Katz, C. L. Cutler, W. M. Sherman, and E. F. Coyle. Muscle glycogen synthesis after exercise: effects of time of carbohydrate ingestion. *J. Appl. Physiol.* 64:1480–1485, 1988.
90. Ivy, J. Exercise physiology and adaptations to training. In: *The Health Professional's Guide to Diabetes and Exercise*. Alexandria, VA: American Diabetes Association, 1995, pp. 7–26.
91. Kahn, B. B. Type 2 diabetes: when insulin secretion fails to compensate for insulin resistance. *Cell* 92:593–596, 1998.
92. Kahn, J. K., B. Zola, J. E. Zuni, and A. I. Vinik. Decreased exercise heart rate and blood pressure response in diabetic subjects with cardiac autonomic neuropathy. *Diabetes Care* 9:389–394, 1986.
93. Kannel, W. B., and D. L. McGee. Diabetes and cardiovascular disease: the Framingham Study. *JAMA* 241:2035–2038, 1979.
94. Katoh, J., Y. Harra, M. Kurusu, J. Miyaji, and K. Narutaki. Cardiorespiratory function as assessed by exercise testing in patients with non-insulin-dependent diabetes mellitus. *J. Int. Med. Res.* 24:209–213, 1996.
95. Kawate, R., M. Yamakido, Y. Nishimoto, P. H. Bennett, R. F. Hamman, and W. C. Knowler. Diabetes mellitus and its vascular complications in Japanese migrants on the island of Hawaii. *Diabetes Care* 2:161–170, 1979.
96. Kelley, S., and P. Seraganian. Physical fitness level and autonomic reactivity to psychosocial stress. *J. Psychosom. Res.* 28: 279–287, 1984.
97. Kemmer, F. W., and B. M. Berger. Exercise in therapy and the life of diabetic patients. *Clin. Sci.* 67:279–283, 1984.
98. Kemmer, F. W., and B. M. Berger. Therapy and better quality of life: the dichotomous role of exercise in diabetes mellitus. *Diabetes/Metab. Rev.* 2:53–68, 1986.
99. King, H., and M. Rewers. Global estimates for prevalence of diabetes mellitus and impaired glucose tolerance in adults. *Diabetes Care* 16:157–177, 1993.
100. King, H., P. Zimmet, L. Raper, and B. Balkau. Risk factors for diabetes in three pacific populations. *Am. J. Epidemiol.* 119:396–409, 1984.
101. Kissebah, A. H., and A. N. Peiris. Biology of regional body fat distribution: Relationship to non-insulin-dependent diabetes mellitus. *Diabetes Metab. Rev.* 5:83–109, 1989.
102. Kjaer, M., C. B. Hollenbeck, B. Frey-Hewitt, H. Galbo, W. Haskell, and G. M. Reaven. Glucoregulation and hormonal responses to maximal exercise in non-insulin-dependent diabetes. *J. Appl. Physiol.* 68:2067–2074, 1990.
103. Klein, R., B. E. K. Klein, and S. E. Moss. Prevalence of microalbuminuria in older-onset diabetes. *Diabetes Care* 16:1325–1330, 1993.
104. Klem, M. L., R. R. Wing, M. T. McGuire, H. M. Seagle, and J. O. Hill. A descriptive study of individuals successful at long-term maintenance of substantial weight loss. *Am. J. Clin. Nutr.* 66:239–246, 1997.
105. Klip, A. Minireview: regulation of glucose transport by insulin and non-hormonal factors. *Life Sci.* 31:2537–2548, 1982.
106. Kohl, H. W., N. F. Gordon, J. A. Villegas, and S. N. Blair. Cardiorespiratory fitness, glycemic status, and mortality risk in men. *Diabetes Care* 15:185–192, 1992.
107. Koivisto, V. A., and R. A. Defronzo. Exercise in the treatment of type 2 diabetes. *Acta Endocrinol.* 262(Suppl. 2):107–111, 1984.
108. Koivisto, V. A., H. Yki-Jarvinen, and R. A. Defronzo. Physical training and insulin sensitivity. *Diabetes/Metab. Rev.* 1:445–481, 1986.
109. Kriska, A., R. Laporte, D. Pettitt, et al. The association of physical activity with obesity, fat distribution and glucose intolerance in Pima Indians. *Diabetologia* 36:863–869, 1993.
110. Krotkiewski, M., P. Lonnroth, K. Mandroukas, et al. The effects of physical training on insulin secretion and effectiveness and on glucose metabolism in obesity and type 2 (non-insulin-dependent) diabetes mellitus. *Diabetologia* 28:881–890, 1985.
111. Lamb, D. R., and G. R. Brodowicz. Optimal use of fluids of varying formulations to minimize exercise-induced disturbances in homeostasis. *Sport Med.* 3:247–274, 1986.
112. Lampman, R. M., and D. E. Schteingart. Effects of exercise training on glucose control, lipid metabolism, and insulin sensitivity in hypertriglyceridemia and non-insulin dependent diabetes mellitus. *Med. Sci. Sports Exerc.* 23:703–712, 1991.
113. Laws, A., and G. M. Reaven. Physical activity, glucose tolerance, and diabetes in older adults. *Ann. Behav. Med.* 13:125–131, 1991.
114. Leon, A. S., J. C. Conrad, D. C. Casal, et al. Exercise for diabetics: effects of conditioning at constant body weight. *J. Cardiopulm. Rehabil.* 4:278–286, 1984.
115. Lindgärde, F., and B. Saltin. Daily physical activity, work capacity and glucose tolerance in lean and obese normoglycaemic middle-aged men. *Diabetologia* 20:134–138, 1981.
116. Long, B. Stress-management interventions: a 15-month follow-up of aerobic conditioning and stress inoculation training. *Cognit. Ther. Res.* 9:471–478, 1985.
117. Lustman, P., R. Carney, and H. Amado. Acute stress and metabolism in diabetes. *Diabetes Care* 4:658–659, 1981.
118. Macera, C. A., J. B. Croft, D. R. Brown, J. E. Ferguson, and M. J. Lane. Predictors of adopting leisure-time physical activity among a biracial community cohort. *Am. J. Epidemiol.* 142:629–635, 1995.
119. Manson, J. E., D. M. Nathan, A. S. Krolewski, M. J. Stampfer, W. C. Willett, and C. H. Hennekens. A prospective study of exercise and incidence of diabetes among US male physicians. *JAMA* 268:63–67, 1992.
120. Manson, J. E., E. B. Rimm, M. J. Stampfer, et al. Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. *Lancet* 338:774–778, 1991.
121. Marcus, B. H., V. C. Selby, R. S. Niaura, and J. S. Rossi. Self-efficacy and the stages of exercise behavior change. *Res. Q. Exerc. Sport* 63:60–66, 1992.
122. Marin, P., B. Andersson, M. Krotiewski, and P. Bjorntorp. Muscle fiber composition and capillary density in women and men with NIDDM. *Diabetes Care* 17:382–386, 1994.
123. Marrero, D. G., and J. M. Sizemore. Motivating patients with diabetes to exercise. In: *Practical Psychology for Diabetes Physicians: How to Deal with Key Behavioral Issues Faced by Health Care Teams,* B. J. Anderson and R. R. Rubin (Eds.). Alexandria, VA: American Diabetes Association, 1996, pp. 73–83.

124. Mayer-Davis, E. J., R. D'agostino, A. J. Karta, et al. Intensity and amount of physical activity in relation to insulin sensitivity. *JAMA* 279:669–674, 1998.
125. McAuley, E. Self-efficacy and the maintenance of exercise participation in older adults. *J. Behav. Med.* 16:103–112, 1993.
126. McAuley, E, C. Lox, and T. E. Duncan. Long-term maintenance of exercise, self-efficacy, and physiological change in older adults. *J. Gerontol. Psychol. Sci.* 48:218–224, 1993.
127. McCann, I. L., and D. S. Holmes. Influence of aerobic exercise on depression. *J. Pers. Soc. Psychol.* 46:1142–1147, 1984.
128. McKeigue, P. M., T. Pierpoint, J. E. Ferrie, and M. G. Marmot. Relationship of glucose intolerance and hyperinsulinaemia to body fat pattern in South Asians and Europeans. *Diabetologia* 35:785–791, 1992.
129. McNabb, W. L., M. T. Quinn, and L. Rosing. Weight loss programs for inner-city black women with non-insulin-dependent diabetes mellitus: PATHWAYS. *J. Am. Diet. Assoc.* 93:75–77, 1993.
130. Miller, W. J., W. M. Sherman, and J. L. Ivy. Effect of strength training on glucose tolerance and post-glucose insulin response. *Med. Sci. Sports Exerc.* 16:539–543, 1984.
131. Minuk, H. L., M. Vranic, E. B. Marliss, A. K. Hanna, A. M. Albisser, and B. Zinman. Glucoregulatory and metabolic response to exercise in obese non-insulin-dependent diabetes. *Am. J. Physiol.* 240:E458–E464, 1981.
132. Modan, M., A. Karasik, H. Halkin, et al. Effect of past and concurrent body mass index on prevalence of glucose intolerance and type 2 diabetes and on insulin response. *Diabetologia* 29: 82–89, 1986.
133. Mogensen, C. E. Nephropathy. Early. In: *The Health Professional's Guide to Diabetes and Exercise*. Alexandria, VA: American Diabetes Association, 1995, pp. 165–74.
134. Mourier A, J-F. Gautier, E. Dekerviler, et al. Mobilization of visceral adipose tissue related to the improvement in insulin sensitivity in response to physical training in NIDDM. *Diabetes Care* 20:385–392, 1997.
135. National Institutes of Health. Consensus development conference on diet and exercise in non-insulin-dependent diabetes mellitus. *Diabetes Care* 10:639–644, 1987.
136. Nyholm B., A. Mengal, S. Nielsen, et al. Insulin resistance in relatives of NIDDM patients: the role of physical fitness and muscle metabolism: *Diabetoligia* 39:813–822, 1996.
137. Nyholm B, Z. Qu, A. Kaal, et al. Evidence of an increased number of type IIb muscle fibers in insulin-resistant first-degree relatives of patients with NIDDM. *Diabetes* 46:1822–1828, 1997.
138. Ohlson, L. O., B. Larsson, K. Svärdsudd, et al. The influence of body fat distribution on the incidence of diabetes mellitus: thirteen and one-half years of follow-up of the participants in the study of men born in 1913. *Diabetes* 34:1055–1058, 1985.
139. Pan, X-P., G-W. Li, Y-H. Hu, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. *Diabetes Care* 20:537–544, 1997.
140. Paternostro-Bayles, M., R. R. Wing, and R. J. Robertson. Effect of life-style activity of varying duration on glycemic control in type 2 diabetic women. *Diabetes Care* 12:34–37, 1989.
141. Pereira, M., A. Kriska, M. Joswiak, et al. Physical inactivity and glucose intolerance in the multi-ethnic island of Mauritius. *Med. Sci. Sports Exerc.* 27:1626–1634, 1995.
142. Perry, I, S. Wannamethee, M. Walker, et al. Prospective study of risk factors for development of non-insulin-dependent diabetes in middle aged British men. *Br. Med. J.* 310:560–564, 1995.
143. Poehlman, E. T., A. W. Gardner, P. A. Ades, et al. Resting metabolism and cardiovascular disease risk in resistance-trained and aerobically-trained males. *Metabolism* 41:1351–1360, 1992.
144. Pollock, M. L., and J. H. Wilmore (Eds). *Exercise in Health and Disease: Evaluation and Prescription of Exercise for Prevention and Rehabilitation*, 2nd Ed. Philadelphia: Saunders, 1990, p. 105.
145. Porte, D. β cells in type II diabetes mellitus. *Diabetes* 40:166–180, 1991.
146. Prochaska, T. R., and M. Glasser. Older adult health behavior change in response to symptom experiences. *Adv. Med. Sociol.* 4:141–161, 1994.
147. Randle, P. J., P. B. Garland, C. N. Hales, and E. A. Newshone. The glucose fatty acid cycle: its role in insulin sensitivity and the metabolic-disturbances of diabetes mellitus. *Lancet* 21:785–789, 1963.
148. Ravussin, E., P. H. Bennett, M. E. Valencia, L. O. Schulz, and J. Esparza. Effects of a traditional lifestyle on obesity in Pima Indians. *Diabetes Care* 17:1067–1074, 1994.
149. Reaven, G. M. Syndrome X. *Clinical Diabetes* 12(4):32–36, 1994.
150. Regensteiner, J. G., S. M. Shetterly, E. J. Mayer, et al. Relationship between habitual physical activity and insulin area among individuals with impaired glucose tolerance. *Diabetes Care* 18:490–497, 1995.
151. Regensteiner, J., J. Sippel, E. McFarlang, E. Wolfel, and W. Hiatt. Effects of non-insulin dependent diabetes on oxygen consumption during treadmill exercise. *Med. Sci. Sports Exerc.* 27:875–881, 1995.
152. Reitman, J. S., B. Vasquez, I. Klimes, and M. Nagulusparan. Improvement of glucose homeostasis after exercise training in non-insulin-dependent diabetes. *Diabetes Care* 7:434–441, 1984.
153. Rice, B., I. Janssen, R. Hudson, and R. Ross. Effects of aerobic exercise and/or diet on glucose tolerance and plasma levels in obese men. *Diabetes Care* 22:684–691, 1999.
154. Rogers, M. A., C. Yamamoto, D. S. King, J. M. Hagberg, A. A. Ehsani, and J. O. Holloszy. Improvement in glucose tolerance after 1 wk of exercise in patients with mild NIDDM. *Diabetes Care* 11:613–618, 1988.
155. Ronnemaa, R., J. Marniemi, P. Puukka, and T. Kuusi. Effects of long-term physical exercise on serum lipids, lipoproteins and lipid metabolizing enzymes in type 2 (non-insulin-dependent) diabetic patients. *Diabetes Res.*7:79–84, 1988.
156. Rönnemaa, T., K. Mattila, A. Lehtonen, and V. Kallio. A controlled randomized study on the effect of long-term physical exercise on the metabolic control in type 2 diabetic patients. *Acta Med. Scand.* 220:219–224, 1986.
157. Ruderman, N. B., O. P. Ganda, and K. Johansen. The effect of physical training on glucose tolerance and plasma lipids in maturity-onset diabetes. *Diabetes* 28(Suppl. 1):89–92, 1979.
158. Ruderman, N., and C. Haudenschild. Diabetes as an atherogenic factor. *Prog. Cardiovasc. Dis.* 26:373–412, 1984.
159. Ruderman, N., D. Chisholm, X. Pi-Sunyer, and S. Schneider. The metabolically obese, normal weight individual revisited. *Diabetes* 47:699–713, 1998.
160. Ryan, A. S., R. E. Pratley, A. P. Goldberg, and D. Elahi. Resistive training increases insulin action in postmenopausal women. *J. Gerontol. A Biol. Sci. Med. Sci.* 51:M199–M205, 1996.
161. Saltin, B., F. Lindgarde, M. Houston, R. Jorlin, E. Hygaard, and Y. Gad. Physical training and glucose tolerance in middle-aged men with chemical diabetes. *Diabetes* 28:30–32, 1979.
162. Santiago, J. V. Overview of the complications of diabetes. *Clin. Chem.* 32:B48–B53, 1986.
163. Schneider, S. H. Long-term exercise programs. In: *The Health Professional's Guide to Diabetes and Exercise*. Alexandria, VA: American Diabetes Association, 1995, pp. 125–132.
164. Schneider, S. H., L. F. Amorosa, A. K. Khachadurian, and N. B. Ruderman. Studies on the mechanism of improved glucose control during regular exercise in type 2 (non-insulin-dependent) diabetes. *Diabetologia* 26:325–360, 1984.
165. Schneider, S. H., A. K. Khachadurian, L. F. Amorosa, L. Clemow, and N. B. Ruderman. Ten-year experience with an exercise-based outpatient lifestyle modification program in the treatment of diabetes mellitus. *Diabetes Care* 15(Suppl. 4):1800–1810, 1992.
166. Schneider S. H., and N. B. Ruderman. Exercise and NIDDM (Technical Review). *Diabetes Care* 17:924–937, 1990.
167. Schneider, S. H., A. Vitug, and N. B. Ruderman. Atherosclerosis and physical activity. *Diabetes/Metab. Rev.* 1:513–533, 1986.
168. Schranz, A., J. Tuomilehto, B. Marti, R. J. Jarrett, V. Grabauskas, and A. Vassallo. Low physical activity and worsening of glucose tolerance: results from a 2-year follow-up of a

population sample in Malta. *Diabetes Res. Clin. Pract.* 11:127–136, 1991.

169. Shaten, B. J., G. D. Smith, L. H. Kuller, and J. D. Neaton. Risk factors for the development of type 2 diabetes among men enrolled in the usual care group of the multiple risk factor intervention trial. *Diabetes Care* 16:1131–1339, 1993.
170. Shulman, G. I., D. L. Rothman, T. Jue, P. Stein, R. A. Defronzo, and R. G. Shulman. Quantitation of muscle glycogen synthesis in normal subjects and subjects with non-insulin-dependent diabetes by ^{13}C nuclear magnetic resonance spectroscopy. *N. Engl. J. Med.* 322:223–228, 1990.
171. Skarfors, E. T., T. A. Wegener, H. Lithell, and I. Selinus. Physical training as treatment for type 2 (non-insulin-dependent) diabetes in elderly men: a feasibility study over 2 years. *Diabetologia* 30:930–933, 1987.
172. Skinner, C. S., V. J. Strecher, and H. Hospers. Physicians' recommendations for mammography: do tailored messages make a difference? *Am. J. Public Health* 84:43–49, 1994.
173. Sonstroem, R. J., and W. P. Morgan. Exercise and self-esteem: rationale and model. *Med. Sci. Sports Exerc.* 21:329–337, 1989.
174. Sothmann, M. S., T. S. Horn, B. A. Hart, and A. B. Gustfson. Comparison of discrete cardiovascular fitness groups on plasma catecholamine and selected behavioral responses to psychological stress. *Psychophysiology* 24:47–54, 1987.
175. Soukup, J. T., and J. E. Kovaleski. A review of the effects of resistance training for individuals with diabetes mellitus. *Diabetes Educator* 19:307–312, 1993.
176. Stalonas, P. M., W. G. Johnson, and M. Christ. Behavior modification for obesity: the evaluation of exercise, contingency management, and programme adherence. *J. Consult. Clin. Psychol.* 46:463–469, 1978.
177. Stern, M. P. Kelly West Lecture: primary prevention of type II diabetes mellitus. *Diabetes Care* 14:399–410, 1991.
178. Stone, M. H., S. J. Fleck, N. T. Triplett, and W. J. Kraemer. Health- and performance-related potential of resistance training. *Sports Med* 11:210–231, 1991.
179. Strecher, V. J., B. McEvoy, M. H. Becker, and I. M. Rosenstock. The role of self-efficacy in achieving health behavior change. *Health Educ. Q.* 13:73–91, 1986.
180. Strecher, V. J., G. H. Seijts, G. J. Kok, et al. Goal setting as a strategy for health behavior change. *Health Educ. Q.* 22:190–200, 1995.
181. Smutok M. A., P. F. Kokkinos, C. Farmer, et al. Aerobic versus strength training for risk factor intervention in middle-aged men at high risk for coronary heart disease. *Metabolism* 42:177–184, 1993.
182. Surwit, R. S., and M. N. Feinglos. Stress and autonomic nervous system in type 2 diabetes: a hypothesis. *Diabetes Care* 11:83–85, 1988.
183. Taylor, R. J., P. H. Bennett, G. Legonidec, et al. The prevalence of diabetes mellitus in a traditional-living Polynesian population: the Wallis Island survey. *Diabetes Car*e 6:334–340, 1983.
184. Taylor, R. J., P. Ram, P. Zimmet, L. Raper, and H. Ringrose. Physical activity and prevalence of diabetes in Melanesian and Indian men in Fiji. *Diabetologia* 27:578–582, 1984.
185. Treuth, M. S., G. R. Hunter, T. Kekes-Szabo, R. L. Weinsier, M. I. Goran, and L. Berland. Reduction in intra-abdominal adipose tissue after strength training in older women. *J. Appl. Physiol* 78:1425–1431, 1995.
186. Trovati, M., Q. Carta, F. Cavalot, et al. Influence of physical training on blood glucose control, glucose tolerance, insulin secretion, and insulin action in non-insulin-dependent diabetic patients. *Diabetes Care* 7:416–420, 1984.
187. U. S. Department of Health and Human Services. Physical activity and health: a report of the Surgeon General. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, 1996.
188. Vaag, A., J. E. Henriksen, and H. Beck-Nielsen. Decreased insulin activation of glycogen synthase in skeletal muscles in young nonobese Caucasian first-degree relatives of patients with non-insulin-dependent diabetes mellitus. *J. Clin. Invest.* 89:782–788, 1992.
189. Van Gaal, L., E. Rillaerts, W. Creten, and I. De Leeuw. Relationship of body fat distribution pattern to atherogenic risk factors in NIDDM: preliminary results. *Diabetes Care* 11:103–106, 1988.
190. Vanninen, E., M. Uusitupa, O. SIItonen, J. Laitinen, and E. Lansimies. Habitual physical activity, aerobic capacity and metabolic control in patients with newly-diagnosed type 2 (non-insulin-dependent) diabetes mellitus: effect of 1-year diet and exercise intervention. *Diabetologia* 35:340–346, 1992.
191. Vasterling, J. J., M. E. Sementilli, and T. G. Burish. The role of aerobic exercise in reducing stress in diabetics patients. *Diabetes Educator* 14:197–201, 1988.
192. Verity, L. S., and A. H. Ismail. Effects of exercise on cardiovascular disease risk in women with NIDDM. *Diabetes Res. Clin. Pract.* 6:27–35, 1989.
193. Vinik, A. I. Neuropathy. In: *The Health Professional's Guide to Diabetes and Exercise*. Alexandria, VA: American Diabetes Association, 1995, pp. 183–197.
194. Vitug, A., S. H. Schneider, and N. B. Ruderman. Exercise and type I diabetes mellitus. In: *Exercise and Sports Science Reviews*, Vol. 16, K. Pandolph (Ed.). New York: MacMillan, 1988, pp. 285–304.
195. Vranic, M., and M. Berger. Exercise and diabetes mellitus. *Diabetes* 28:147–163, 1979.
196. Vranic, M., and D. Wasserman. Exercise, fitness, and diabetes. In: *Exercise, Fitness, and Health*, C. Bouchard, R. J. Shephard, T. Stephens, J. Sutton, and B. MacPherson (Eds.). Champaign, IL: Human Kinetics, 1990, pp. 467–490.
197. Wahren J, Felig, and P. L. Hagenfeldt. Physical exercise and fuel homeostasis in diabetes mellitus. *Diabetologia* 14:213–222, 1978.
198. Wallberg-Henricksson, H. Exercise and diabetes mellitus. In: *Exercise and Sport Science Reviews*, Vol. 22, J. O. Holloszy (Ed.). Philadelphia: Williams & Wilkins, 1992, pp. 339–368.
199. Wang, J. T., L. T. Ho, K. T. Tang, L. M. Wang, Y. D. I. Chen, and G. M. Reaven. Effect of habitual physical activity on age-related glucose intolerance. *J. Am. Geriatr. Soc.* 37:203–209, 1989.
200. Wasserman, D. H., and N. N. Abumrad. Physiological bases for the treatment of the physically active individual with diabetes. *Sports Med.* 7:376–392.
201. Wasserman, D. H., and B. Zinman. Exercise in individuals with IDDM (Technical Review). *Diabetes Care* 17:924–937, 1994.
202. Watts, N. B., R. G. Spanheimer, A. Digirolamo, et al. Prediction of glucose response to weight loss inpatients with non-insulin-dependent diabetes mellitus. *Arch. Intern. Med.* 150:803–806, 1990.
203. Waxman, S., and R. W. Nesto. Cardiovascular complications. In: *The Health Professional's Guide to Diabetes and Exercise.* Alexandria, VA: American Diabetes Association, 1995, pp. 155–162.
204. West, K. M. *Epidemiology of Diabetes and Its Vascular Lesions*. New York: Elsevier, 1978.
205. Wing, R. R. Behavioral strategies for weight reduction in obese type 2 diabetic patients. *Diabetes Care* 12:139–144, 1989.
206. Wing, R. R., L. H. Epstein, M. Paternostro-Bayles, A. Kriska, M. P. Norwalk, and W. Gooding. Exercise in a behavioural weight control programme for obese patients with type 2 (non-insulin-dependent) diabetes. *Diabetologia* 31:902–909, 1988.
207. Wing, R. R. Exercise and weight control. In: *The Health Professional's Guide to Diabetes and Exercise.* Alexandria, VA: American Diabetes Association, 1995, pp. 111–114.
208. Wing, R. R., L. H. Epstein, M. P. Nowalk, R. Koeske R., and S. Hagg. Behavior change, weight loss, and physiological improvements in type II diabetic patients. *J. Consult Clin. Psychol.* 53:111–122, 1985.
209. Wing R. R., R. Koeske, L. H. Epstein, M. P. Nowalk, W. Gooding, and D. Becker. Long-term effects of modest weight loss in type II diabetic patients. *Arch Intern Med.* 147:1749–1753, 1987.

210. Wulsin, L. R., A. M. Jacobsen, and L. I. Rand. Psychosocial adjustment to advanced proliferative diabetic retinopathy. *Diabetes Care* 16:1061–1066, 1993.
211. Yamanouchi, K., T. Shinozaka, K. Chikada, et al. Daily walking combined with diet therapy is a useful means for obese NIDDM patients not only to reduce body weight but also to improve insulin sensitivity. *Diabetes Care* 18:775–778, 1995.
212. Yki-Jarvinen, H., and V. A. Koivisto. Effects of body composition on insulin sensitivity. *Diabetes* 32:965–969, 1983.
213. Yki-Jarvinen, H. Glucose toxicity. *Endocr. Rev.* 13:415–431, 1992.
214. Zamboni, M., Armellini, F., Turcato, et al. Effect of weight loss on regional body fat distribution in premenopausal women. *Am. J. Clin. Nutr.* 58:29–34, 1993.
215. Zierath, J. R., and H. Wallberg-Henricksson. Exercise training in obese diabetic patients: special considerations. *Sports Med.* 14:171–189, 1992.
216. Zimmet, P. Z., S. Faauiso, S. Ainuu, S. Whitehouse, B. Milne, and W. Deboer. The prevalence of diabetes in the rural and urban Polynesian population of Western Samoa. *Diabetes* 30:45–51, 1981.
217. Zimmet, P. Z., R. Taylor, P. Ram, et al. Prevalence of diabetes and impaired glucose tolerance in the biracial population of Fiji: a rural-urban comparison. *Am. J. Epidemiol.* 118:673–688, 1983.
218. Zola, B., J. Kahn, J. Juni, and A. I. Vinik. Abnormal cardiac function in diabetics with autonomic neuropathy in the absence of ischemic heart disease. *J. Clin. Endocrinol. Metab.* 63:208–214, 1986.