

Review

Prevention of Type 2 Diabetes by Diet and Lifestyle Modification

Lydia A. Bazzano, MD, PhD, Mary Serdula, MD, MPH, and Simin Liu, MD, ScD, FACN

Department of Medicine, Beth Israel Deaconess Hospital (L.A.B.), Division of Preventive Medicine, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School and Department of Epidemiology, Harvard School of Public Health (S.L.), Boston, Massachusetts, Division of Nutrition and Physical Activity, Centers for Disease Control and Prevention (M.S.), Atlanta, Georgia

Key words: type 2 diabetes mellitus, coronary heart disease, lifestyle, prevention

Diabetes mellitus is an epidemic of our time. This disease affects nearly 150 million adults worldwide and nearly 11 million in the United States in 2000. Because of the prevalence of obesity and diabetes and associated vascular complications, preventing even a small proportion of cases would save thousands of lives and billions of dollars in healthcare costs and lost productivity. Researchers have made great strides in identifying many lifestyle and dietary factors associated with diabetes, but solidifying the scientific basis for prevention and control of this disease as well as implementation at a national level remains a difficult challenge. The literature on the influence of diet and lifestyle in the development of diabetes is reviewed here, with emphasis on epidemiologic data. We outline a systematic approach to primary and secondary prevention of this disease by evaluating and prioritizing risk factors for which intervention is effective and developing a framework for application of intervention strategies. Effective interventions must target not only the affected individuals but also families, workplaces, schools and communities. Prevention of this devastating disease calls for the identification of culture-sensitive measures that can be applied to the population in general and some high-risk minority groups in particular.

Key teaching points

- Prevalence of diabetes is rising to epidemic proportions and costs US society billions of dollars per year.
- Modifiable risk factors for diabetes include obesity, alcohol intake, cigarette smoking, physical inactivity and dietary factors, such as glycemic load, intake of fat, fiber, and whole-grain foods.
- Disease prevention must identify culturally sensitive measures that can be applied to the population in general and to specific high-risk minority groups in particular.

INTRODUCTION

Diabetes mellitus has become one of the great epidemics of our time, affecting nearly 150 million adults worldwide and nearly 11 million adults in the United States in 2000 [1]. In 2002, diabetes cost approximately 132 billion dollars in direct medical costs and lost productivity [2]. The prevalence of diagnosed diabetes in US adults has increased by 61% in the past decade, from 4.9% in 1990 to 7.9% in 2001 [3–5]. By the year 2050, diabetes is expected to affect approximately 29 million adults in the United States, an

increase of 165% [1]. Cumulative lifetime risk for developing diabetes for individuals born in 2000 in the United States is 32.8% for males and 38.5% for females [6]. Moreover, diabetes is not only costly and increasing in prevalence, it is the sixth leading cause of death in the United States, accounting for 71,372 deaths in 2001 [7]. Individuals diagnosed with diabetes sustain a significant reduction in life expectancy and quality of life. It has been estimated that diagnosis with diabetes at 40 years of age will result in the loss of approximately 11.6 life-years for males and 14.3 life-years for females [6].

Address correspondence to: Simin Liu, MD, ScD, FACN, Associate Professor of Medicine, Director of Nutrition Research, Division of Preventive Medicine, Harvard Medical School and Brigham and Women's Hospital, 900 Commonwealth Avenue, Boston, MA 02215. E-mail: siminliu@hsph.harvard.edu

Type 2 diabetes mellitus accounts for approximately 95% of all diabetes and is associated with both macrovascular complications (e.g. myocardial infarction and stroke), and microvascular complications (e.g. nephropathy and retinopathy). Complications such as diabetic neuropathy reduce quality of life for those affected. Macrovascular complications comprise both the most costly and life-threatening conditions. They account for 85% of the cumulative costs in the first 5 years after diagnosis and are implicated in 75% of diabetes-related deaths [8,9]. The average cost of diabetic complications over 30 years was estimated at \$47,240 per patient [9]. In addition, the risk of both macrovascular and microvascular complications may be greater with type 2 diabetes mellitus than type 1 diabetes mellitus [10].

Insulin resistance and progressive dysfunction of pancreatic beta cells are well-established fundamental steps in the pathogenesis of type 2 diabetes mellitus. Risk factors for the conversion from an insulin-sensitive state to insulin resistance include obesity, diet, sedentary lifestyle, and genetics. Obesity and weight gain, which have also grown to epidemic proportions in the United States, may be the most potent catalysts of this transformation [5,11]. Compensatory hyperinsulinemia tends to lead to [1] dyslipidemia; [2] decreases in vasodilatation mediated by nitric oxide; and [3] increases in blood pressure, clotting factors, and inflammatory risk factors for coronary artery disease (e.g., fibrinogen and plasminogen activator inhibitor-1) [12,13]. Because of the prevalence of obesity and diabetes and their associated vascular complications, preventing even a small proportion of cases would save thousands of lives and billions of dollars in health care costs and lost productivity. Researchers have made great strides in identifying many lifestyle and dietary factors associated with diabetes, but solidifying the scientific basis for prevention and control of this disease remains a difficult challenge.

The literature on the influence of diet and lifestyle factors in the development of diabetes is summarized here with emphasis on prospective epidemiologic data where available. However, it is important to note that this is not an exclusive review of the entirety of the literature on this topic. The majority of studies included in this review were identified through a MEDLINE search using the MeSH terms “diabetes mellitus type 2” with exploded focus, combined with either of the MeSH terms “primary prevention” or “lifestyle” also with exploded focus. In addition, we outline a systematic approach to primary and secondary prevention of type 2 diabetes by evaluating and prioritizing risk factors for which intervention is effective and by developing a framework for application of intervention strategies.

Dietary Factors

Uncertainties abound even though the complex interrelationships between nutrients and foods in our diet and risk of developing of diabetes and hyperglycemia have been examined by many investigators. Decades of research have indicated that

diets with high saturated fat content and low fiber content may increase the risk of insulin resistance and lead to development of type 2 diabetes, but epidemiologic data have been relatively inconsistent [14]. More recently, investigations of specific types of foods, glycemic loads of carbohydrates contained in foods, and dietary patterns have led to a clearer picture of those dietary components that may affect development of diabetes [14–16].

Dietary Fat Intake

The type and amount of dietary fat intake has been associated with insulin sensitivity in animal studies [17]. Monounsaturated or polyunsaturated fats appear to have beneficial effects on insulin action, whereas saturated fats and diets with high total-fat content appear to decrease insulin sensitivity in animal studies [17,18]. It is hypothesized that dietary fats affect the phospholipid composition of cell membranes in skeletal muscle and other tissues. Several clinical studies showed a decrease in insulin sensitivity with high fat diets [14,19]. However, many aspects of these studies diminish the strength of their conclusions, including large differences in diets, the non-randomized assignment of diets, and lack of standardized methods to measure insulin sensitivity. A few studies using more standard measures reported a relationship between fat content and insulin sensitivity [20]. One reason may be the relatively short duration of intervention in many of these studies. A recent multicenter, 3-month investigation found that a diet high in saturated fat (18% of energy) decreased insulin sensitivity more than a diet high in monounsaturated fat (21% of energy) among 162 healthy men and women [21]. Many cross-sectional epidemiologic studies also demonstrated positive associations between intake of saturated fat and hyperinsulinemia, after adjustment for measures of body fat [22,23], but, at least one large, well-designed study showed no association [24]. Prospective studies including the Nurses’ Health Study [25] suggest the role of specific types of fat in the development of type 2 diabetes mellitus. In the Nurses Health Study, investigators reported an inverse association between development of diabetes and intake of vegetable fat and polyunsaturated fat, a positive association for *trans*-fatty acids, but no association for total fat in the diet.

Dietary Fiber

Non-starch polysaccharides or dietary fiber have been shown to delay absorption of carbohydrates after a meal and thereby decrease the insulinemic response to other dietary carbohydrates [26]. In one multicenter study of 2,909 healthy young adults ages 18 to 30 years old, dietary fiber intake was strongly associated with body weight, waist-to-hip ratio, fasting blood insulin concentrations, and concentrations at 2 hours after glucose intake, after adjustment for confounding factors [27]. In addition, several large prospective cohort studies showed inverse associations between dietary fiber intake and

risk of developing type 2 diabetes mellitus [22,23,28–31]. Data from the Health Professional's Follow-up Study and from the Nurses Health Study support an inverse association between cereal fiber and development of diabetes [28,29]. In these two studies, investigators found a stronger association for cereal fiber than for fiber from other sources (e.g. fruits and vegetables). Meyer et al. found a similar inverse association in the Iowa Women's Health Study [30]. In that prospective cohort study, 35,988 older women in Iowa who were initially free of diabetes had followed-up for 6 years. Multivariate-adjusted relative risks of diabetes were 1.0, 1.09, 1.00, 0.94, and 0.78 across quintiles for intake of total dietary fiber (p for trend <0.01). Associations with type 2 diabetes were stronger for cereal fiber than for sources of fruit and vegetable fiber. More recently, two large prospective cohort studies have also reported similar findings [31,32].

Glycemic Index and Glycemic Load

Few epidemiologic studies have examined the role of type and amount of carbohydrates in relation to the development of hyperglycemia or type 2 diabetes mellitus. Those that have, generally found little association between total carbohydrate intake or intake of simple sugars and the development of diabetes [28,30,33–36]. One exception, the Iowa Women's Health Study, reported that intake of glucose or fructose was significantly and positively related to risk of developing type 2 diabetes mellitus [30].

Since the introduction of the glycemic index in 1981, the role of carbohydrates in the development of type 2 diabetes has been thought to depend less on the size and structure of the carbohydrate molecule and more on the body's glycemic response to different carbohydrates [37]. The glycemic index compares the glucose-raising potential of equal amounts of carbohydrate but the glycemic index does not capture the quantity of carbohydrate in a food serving. The glycemic load, the product of the glycemic index value of a food and its total carbohydrate content, captures both aspects of the glucogenic potential of a food [38]. Several large prospective cohort studies have examined the relationship between glycemic index or load and risk of developing type 2 diabetes mellitus [28–30,39]. Overall, persons with a diet at the highest level of the glycemic index or glycemic load were significantly more likely to develop type 2 diabetes mellitus than those at the lowest levels. For example, in the Nurses Health Study, the relative risk of developing type 2 diabetes mellitus was 1.47 (95% confidence interval [CI], 1.16 to 1.86) for the highest versus the lowest quintile of dietary glycemic load [29]. Similarly, in the Health Professionals' Follow-up Study, the relative risk of developing type 2 diabetes mellitus was 1.37 (95% CI, 1.02 to 1.83) for comparison of the extreme quintiles of dietary glycemic load [28]. However, two large prospective studies found no relationship between dietary glycemic index or glycemic load and risk of developing type 2 diabetes mellitus [30,31]. The

lack of association in these studies may have been related to the methods of diet assessment used.

The American Diabetes Association (ADA) recommends eating foods containing carbohydrate from whole-grains, fruits, vegetables, and low-fat milk as part of a healthy diet for persons with diabetes [40]. On the whole carbohydrates derived from these foods are likely to have a low glycemic index. While the ADA does not comment on the potential role of the glycemic index or load in prevention of diabetes amongst normoglycemic persons, they do state that diabetics should regard the total amount of carbohydrate in meals or snacks as more important for blood glucose regulation than the source or type [40].

Whole-Grain Intake

Few prospective studies have examined the relationship between intake of whole-grain foods and risk of type 2 diabetes mellitus. In both the Iowa Women's Health Study and the Nurses Health Study, whole-grain consumption was significantly and inversely associated with development of diabetes [30,41]. For Iowa women, the relative risks of developing diabetes were 1.0, 0.99, 0.98, 0.92, and 0.79 (p for trend <0.01), across quintiles of whole-grain intake, after multivariate adjustment. Those in the highest quintile consumed more than 33 servings of whole-grain foods per week; those in the lowest quintile consumed less than 13 servings per week [30]. In another study, U.S. nurses in the highest quintile of whole-grain consumption (median, 2.7 servings per day) had a 27% lower risk of developing diabetes (relative risk, 0.73; 95% CI, 0.63 to 0.85) than those in the lowest quintile of whole-grain intake (median, 0.13 servings per day), after multivariate adjustment [41]. In addition, Fung et al. reported similar results by using data from the Health Professionals' Follow-up Study [42]. Two more recent studies in other large cohorts reported an inverse association between whole-grain intake and development of hyperglycemia and diabetes [32,43]. This finding lends strong support to the hypothesis that increasing whole-grain intake may decrease the development of diabetes in the general population.

Overweight and Obesity

Obesity has long been recognized as one of the strongest risk factors for development of diabetes. It has been estimated to account for 60% to 90% of the risk variance [44,45]. Several prospective studies in widely different populations demonstrated a strongly positive association between body mass index (BMI) and weight gain and subsequent development of diabetes [46]. Colditz and colleagues found that risk of diabetes was increased by nearly 90-fold among female nurses who were morbidly obese (BMI ≥ 35) at ages 30 to 55 years and had normal weight at age 18 (BMI <22) [46]. In many studies, weight loss improved glycemic control in individuals with diabetes and led to remission of diabetes in a few [11,47,48]. In

studies examining energy-restricted diets in persons with type 2 diabetes mellitus, fasting hyperglycemia declined rapidly within the first week and reductions in hepatic glucose production were observed even before significant weight loss occurred [49,50]. Anderson and colleagues combined the results of 10 studies of obese persons with type 2 diabetes who were treated for 4 to 6 weeks with very low energy diets [11]. Subjects lost approximately 10% of body weight, and fasting plasma glucose values decreased to about one-half of initial values after 2 weeks. These values remained stable for the duration of these studies, but weight loss on these diets was often not sustained and the quality of glycemic control decreased as weight was regained. There is also increasing evidence that waist circumference is an important risk factor for diabetes. Prospective studies of sustained weight loss and development of diabetes suggest that even modest weight loss is associated with significantly reduced risk of diabetes [51,52]. This type of evidence suggests that obesity and excess energy consumption are perhaps the most important factors contributing to risk of diabetes. Hence, even minor weight reductions may have major beneficial effects on subsequent diabetes risk of overweight persons.

Physical Activity

The importance of sedentary lifestyle as a risk factor for diabetes and of the protective effects of physical activity have also been studied. It is hypothesized that physical activity results in a higher rate of insulin-stimulated glucose disposal at a defined insulin dose [53–56]. In addition, physically trained persons may have a smaller increase in plasma insulin concentrations in response to a glucose load than do sedentary persons [57,58]. These results suggest that exercise training increases tissue sensitivity to insulin.

In prospective cohort studies, persons who maintain a physically active lifestyle develop impaired glucose tolerance and type 2 diabetes mellitus less often than do those with a sedentary lifestyle [59–63]. Helmrich and colleagues examined leisure-time physical activity and development of diabetes among 5,990 male alumni of the University of Pennsylvania over 14 years [63]. They discovered that men who exercised regularly, at moderate or vigorous intensity, had a 35% lower risk of developing type 2 diabetes mellitus than men who were sedentary [63]. Manson and colleagues observed similar findings by analyzing data from the Nurses Health Study and the Health Professionals' Follow-up Study [60,61]. More recently, walking was compared to vigorous activity among 70,102 participants in the Nurses Health Study cohort [59]. After 8 years of follow-up, participants who performed the most physical activity were 26% less likely to develop diabetes than those who were sedentary (p for trend <0.01). Even among women who did not perform vigorous physical activity, those who walked most also were 26% less likely to develop diabetes than those who walked least (p for trend = 0.01). This finding suggests that both walking and more vigorous physical activity are

associated with reductions in risk of diabetes. However, more studies are needed to determine the intensity, frequency, and duration of exercise that is most effective in prevention of diabetes. At least one study suggests that cardiopulmonary fitness may play a lesser role than overall energy expenditure in prevention of glucose intolerance [64].

Alcohol Consumption

Epidemiologic studies of alcohol intake and risk of type 2 diabetes mellitus have produced conflicting results. A recent review of 18 prospective cohort studies evaluated the association between alcohol consumption and the incidence of diabetes [65]. Eight of these studies found a U-shaped relationship between alcohol consumption and diabetes incidence; moderate drinkers had the lowest risk for diabetes, and nondrinkers and heavy drinkers had a higher risk. However, heavy drinking was significantly associated with type 2 diabetes mellitus in only two of these studies [66,67]. Alcohol consumption and diabetes incidence were inversely related in three studies, but the prevalence of heavy drinking was low (1%–3%), hence these studies had limited power to detect a relationship between heavy alcohol use and diabetes [39,68,69].

Mechanisms by which alcohol may act to increase or decrease risk of diabetes are multifold. Several studies suggest that low-to-moderate amounts of alcohol intake may decrease development of diabetes by increasing insulin sensitivity and slowing glucose uptake from a meal [70,71]. Excessive alcohol intake may contribute to excess energy intake and obesity, induction of pancreatitis, disturbance of carbohydrate and glucose metabolism, and impairment of liver function [72–74]. Although further studies of alcohol intake and development of type 2 diabetes mellitus are needed, the burden of evidence suggests that a moderate alcohol intake may reduce incidence of diabetes.

Cigarette Smoking

Cigarette smoking may increase risk of diabetes in several ways. Smoking has been shown to cause elevations in blood glucose concentration and may increase insulin resistance [75,76]. Current smokers also tend to have higher blood concentrations of glycosylated hemoglobin (HbA_{1c}) than do nonsmokers [77,78]. One recent population-based cross-sectional study showed an independent positive association between cigarette smoking and HbA_{1c} concentration in men and women [78]. In addition, although many studies have shown that smokers, on average, have lower BMI than nonsmokers, they also tend to have more central fat deposition which is associated with insulin resistance [79,80].

Several large prospective cohort studies suggest that smoking is associated with development of diabetes. Among participants of the Nurses Health Study, women who smoked more than 25 cigarettes per day had a 42% greater risk (95% CI, 1.18 to 1.72) of developing diabetes than those who had never

smoked, after adjustment for obesity and other risk factors [81]. A prospective study of 7,124 British men who had follow-up for an average of 16 years reported similar findings [82]. In that study, men who smoked cigarettes had a 74% higher risk (95% CI, 1.24 to 2.43) of developing diabetes than those who had never-smoked, after adjustment for age and BMI. In the Physicians' Health Study cohort of 21,068 men ages 40 to 84 years, smokers of at least 20 cigarettes daily had a 70% greater risk (95% CI, 1.3 to 2.3) of developing diabetes than participants who never smoked, after adjustment for multiple risk factors [83]. Among 1,266 Japanese men from 35 to 59 years of age, the number of cigarettes smoked daily and pack-year history of smoking were positively associated with development of impaired fasting glucose and type 2 diabetes mellitus [84]. Not every study shows this association [85], but overall, the data suggest that cigarette smoking may be an independent modifiable risk factor for development of diabetes. National policy changes in recent years have helped reduce rates of cigarette smoking and have played a crucial role in recent gains in tobacco control efforts.

Multiple risk factors for development of diabetes were examined simultaneously using data from the Nurses Health Study cohort [39]. Hu et al. examined risk of diabetes based on multiple factors defining a low-risk group: BMI, a diet high in cereal fiber and polyunsaturated fat and low in *trans* fat and glycemic load, moderate-to-vigorous physical activity for at least half an hour per day, not smoking, and consumption of at least 4 ounces of an alcoholic drink per day. The investigators reported that overweight or obesity was the single most important predictor of diabetes. Moreover, physical activity, diet score, smoking and abstinence from alcohol were also associated with the development of diabetes after adjustment for BMI. Women in the low-risk cohort defined by these habits were at significantly lower risk of developing diabetes (relative risk, 0.09; 95% CI, 0.05 to 0.17). These findings lend important support to the notion that type 2 diabetes mellitus is a preventable disease.

Diabetes Prevention Trials

Four major studies of lifestyle change in diabetes prevention demonstrated that it is possible to prevent diabetes in high-risk persons [86–89]. The first study was conducted in Malmo, Sweden, among 415 men ages 47 to 49 years who participated in a large population-based screening program [86]. Persons with diabetes, impaired glucose tolerance or normal glucose tolerance were nonrandomly assigned to lifestyle intervention or usual care groups. After 6 years, 10.6% of the men with impaired glucose tolerance in the lifestyle intervention group had developed diabetes, whereas 28.6% in the usual care group had developed diabetes [86].

In China, the Da Qing study showed similar results among 557 participants with impaired glucose tolerance who were randomly assigned by a clinic to a control group or to one of

three lifestyle-intervention groups: diet, exercise, or diet and exercise [87]. After 6 years, the cumulative incidence of diabetes in the control group was 67.7% compared with 43.8% in the diet group, 41.1% in the exercise group, and 46.0% in the combination diet and exercise group [87].

More recently, two large randomized controlled trials of lifestyle changes in the prevention of diabetes were conducted in the United States and Finland [88,89]. The Finnish Diabetes Prevention Study [88] was conducted among 522 participants who were 40 to 65 years of age, were overweight (BMI >25), and had impaired glucose tolerance. Subjects were randomly assigned to receive either lifestyle intervention consisting of advice and supervision related to diet and exercise or usual care (control group). This trial, with an average follow-up of 3.2 years, was terminated early, after the first analysis of interim results showed that the cumulative incidence of diabetes in the intervention group was 58% lower than in the control group even though a large portion of subjects in the intervention group did not achieve their weight loss or diet goals [88].

The largest prevention trial was conducted in the United States among 3,819 subjects who were at least 25 years of age and had BMI of at least 24 and impaired glucose tolerance [89]. The Diabetes Prevention Program randomly assigned participants to one of four groups: intensive lifestyle intervention, metformin therapy with standard lifestyle advice, troglitazone therapy with standard advice on lifestyle, and standard lifestyle advice with placebo medication (control group). The troglitazone arm of the study was discontinued early because of concerns about drug safety. Incidence of diabetes in the group with intensive lifestyle intervention was 58% lower than that in the group receiving standard advice plus placebo, and the intensive lifestyle intervention was more effective than metformin plus standard advice [89].

Given the evidence to date, the World Health Organization published recommendations for the prevention of diabetes in a 2003 technical report [90]. The strength of evidence regarding overweight, obesity, central adiposity, and physical inactivity as risk factors for diabetes type 2 was deemed convincing. Dietary intake of saturated fat was considered a probable risk factor for developing diabetes, while intake of dietary fiber or non-starch polysaccharides was considered a probable protective factor for developing diabetes. Intake of *trans*-fatty acids and total fat were considered possibly risk factors for diabetes, while consumption of a low glycemic index diet was considered possibly protective. The data on alcohol consumption in relation to diabetes were considered insufficient to render a judgment. Neither cigarette smoking nor whole-grain intake was addressed in the latter report. Our conclusions in this review agree strongly with those of the World Health Organization with the expansion that we consider cigarette smoking to be a possible risk factor and whole-grain intake to be a probable protective factor in the development of diabetes. Overall, scientific evidence strongly supports the role of dietary and

lifestyle factors in the prevention of type 2 diabetes. Thus individuals can adopt healthy lifestyle and diets in efforts to prevent development of type 2 diabetes mellitus.

Toward a Framework for Prevention

On the basis of evidence from these trials and from the analyses of multiple risk factors, early identification of risk factors and intervention may contribute to the prevention of diabetes. A high BMI is one of the most potent risk factors for the development of diabetes. Therefore, persons should be targeted for intensive lifestyle prevention if they have BMI \geq 25 plus two or more of the following risk factors: family history of diabetes, ethnicity of American Indian, African American, Hispanic or Asian/Pacific Islander, and/or insulin resistance. For these persons, an individualized strategy that focuses on losing weight, improving dietary composition, increasing physical activity, and avoiding smoking should be pursued. Weight loss alone may reverse the course of insulin resistance and normalize blood glucose concentrations. Sustaining weight loss is a difficult task, but even modest weight loss may confer substantial benefits for diabetes prevention. Dietary change should include a reduction in saturated and *trans* fats and an increase in fruits, vegetables, and whole-grain foods, as well as maintenance of a low glycemic load. Lifestyle change should also focus on increasing physical activity, which improves insulin sensitivity independent of the effect on BMI. Moreover, although even modest physical activity decreases risk of type 2 diabetes mellitus, a sedentary lifestyle promotes obesity and increases risk of type 2 diabetes mellitus.

Applying Research in Communities

Prevention programs should target communities, as well as individuals. Approaches that focus on individuals at risk work well for those who are motivated, but community-based prevention programs can benefit more people by facilitating the spread of culturally-relevant messages and providing access to social support systems [91]. Although community-based programs for diabetes prevention described in the literature are still in the early phases, most have been initiated by communities disproportionately affected by diabetes, driven by the concern of community leaders who have witnessed the effects of diabetes [91]. In some communities where the deleterious effects of diabetes are well known, community program planners find they must counter a sense of fatalism about the inevitability of diabetes understanding the community's history as context for contemporary action [92,93]. Some communities also tend to view diabetes more as a disease of personal responsibility, that ranks as a lower priority for community interventions than other threats [92]. For these communities, awareness of risk and of potential for prevention first need to be heightened [91,93]. The campaign of the National Diabetes Education Program, co-directed by the Centers for Disease Control and Prevention and the National Institutes of Health, "Small Steps,

Big Rewards" can serve to promote awareness of the benefit of modest adaptations in lifestyle to prevent or delay the onset of diabetes [94].

Translating interventions such as the Diabetes Prevention Program from research settings into real-world community settings is challenging. Compared with research settings, community settings are more complex and have fewer resources [95]. Challenges include designing multifaceted programs that are culturally relevant and that incorporate traditional community preferences in food and physical activity, as well as beliefs about gender role and health in diverse populations [91]. Understanding barriers to communication, which is key, can be facilitated by peers in talking circles [96] or focus groups. It is also critical to understand the context of increased predisposition to risk factors (e.g. obesity and physical inactivity) and to diabetes, in terms of a community's social and economic background, and history. Barriers to action may include [1] a community's environmental challenges such as fear of impure water, which can promote the substitution of packaged, sweetened beverages; [2] lack of safety for community walks; or [3] cultural dissonance with medical recommendations. Additional barriers include personal factors such as lack of insurance, lack of time, and inadequate social support at home. Community-based prevention programs tend [1] to enlist the engagement of formal and informal community leaders; [2] to reinforce social support and the connection to the community and [3] to use meaningful methods for communication such as culturally-based stories, and for social support, such as talking circles and liaisons with community health workers [96,97].

The successful experience with comprehensive tobacco control provides a framework for future diabetes interventions that also address environmental conditions in communities and in the larger society [98]. In addition to clinical intervention and management and educational strategies, the tobacco-cessation model includes economic approaches, policy and environmental changes. Without environmental and policy support, individual lifestyle change is likely to dissipate over time [99]. A wide variety of policy and environmental opportunities exist to promote protective health behaviors, including prevention of obesity and promotion of physical activity. These opportunities include changes in education, food-assistance and feeding programs, food labeling and advertising, transportation, and urban development [100]. Health policy and legislation play an important role in implementing lifestyle changes on a broad scale. Many of the potential challenges in this approach to the primary prevention of diabetes type 2 are currently being explored by national authorities [101].

Conclusions

The preponderance of evidence reviewed here on risk factors for and prevention of diabetes demonstrates that type 2 diabetes mellitus which comprises 95% of diabetes cases, is a preventable disease. Because of the cost and morbidity of

diabetes and the rampant increase in overweight and obesity and in incidence of diabetes in the U.S. population, prevention of even a small part of the growing number of cases would save thousands of lives and billions of health care dollars. One inescapable conclusion is that the public health importance of diabetes prevention is indisputable. To reduce the burden of this devastating disease, prevention programs must target not only the affected individuals but also families, workplaces, schools, and communities.

REFERENCES

1. Boyle JP, Honeycutt AA, Narayan KM, Hoerger TJ, Geiss LS, Chen H, Thompson TJ: Projection of diabetes burden through 2050: impact of changing demography and disease prevalence in the U.S. *Diabetes Care* 24:1936–1940, 2001.
2. Hogan P, Dall T, Nikolov P: American Diabetes Association: Economic costs of diabetes in the US in 2002. *Diabetes Care* 26:917–932, 2003.
3. Mokdad AH, Ford ES, Bowman BA, Nelson DE, Engelgau MM, Vinicor F, Marks JS: Diabetes trends in the U.S.: 1990–1998. *Diabetes Care* 23:1278–1283, 2000.
4. Mokdad AH, Ford ES, Bowman BA, Nelson DE, Engelgau MM, Vinicor F, Marks JS: The continuing increase of diabetes in the U.S. *Diabetes Care* 24:412, 2001.
5. Mokdad AH, Ford ES, Bowman BA, Dietz WH, Vinicor F, Bales VS, Marks JS: Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. *JAMA* 289:76–79, 2003.
6. Narayan KM, Boyle JP, Thompson TJ, Sorensen SW, Williamson DF: Lifetime risk for diabetes mellitus in the United States. *JAMA* 290:1884–1890, 2003.
7. Anderson RN, Smith BL: Deaths: leading causes for 2001. *Nat'l Vital Stat Rep* 52:1–85, 2003.
8. O'Brien JA, Patrick AR, Caro J: Estimates of direct medical costs for microvascular and macrovascular complications resulting from type 2 diabetes mellitus in the United States in 2000. *Clin Ther* 25:1017–1038, 2003.
9. Caro JJ, Ward AJ, O'Brien JA: Lifetime costs of complications resulting from type 2 diabetes in the U.S. *Diabetes Care* 25:476–481, 2002.
10. Nazimek-Siewniak B, Moczulski D, Grzeszczak W: Risk of macrovascular and microvascular complications in Type 2 diabetes: Results of longitudinal study design. *J Diabetes Complications* 16:271–276, 2002.
11. Anderson JW, Kendall CWC, Jenkins DJA: Importance of weight management in type 2 diabetes: Review with meta-analysis of clinical studies. *J Am Coll Nutr* 22:331–339, 2003.
12. Williams K, Sniderman AD, Sattar N, D'Agostino R, Wagenknecht LE, Haffner SM: Comparison of the associations of apolipoprotein B and low density lipoprotein cholesterol with other cardiovascular risk factors in the Insulin Resistance Atherosclerosis Study (IRAS). *Circulation* 108:2312–2316, 2003.
13. Reaven GM: Pathophysiology of insulin resistance in human disease. *Physiol Rev* 75:473–486, 1995.
14. Hu FB, van Dam RM, Liu S: Diet and risk of type II diabetes: the role of types of fat and carbohydrate. *Diabetologia* 44:805–817, 2001.
15. Liu S: Intake of refined carbohydrates and whole-grain foods in relation to risk of type 2 diabetes mellitus and coronary heart disease. *J Am Coll Nutr* 21:298–306, 2002.
16. van Dam RM, Rimm EB, Willett WC, Stampfer MJ, Hu FB: Dietary patterns and risk for type 2 diabetes mellitus in U.S. men. *Ann Intern Med* 136:201–209, 2002.
17. Storlien LH, Jenkins AB, Chisholm DJ, Pascoe WS, Khouri S, Kraegen EW: Influence of dietary fat composition on development of insulin resistance in rats. Relationship to muscle triglyceride and omega-3 fatty acids in muscle phospholipid. *Diabetes* 40:280–289, 1991.
18. Lardinois CK, Starich GH: Polyunsaturated fats enhance peripheral glucose utilization in rats. *J Am Coll Nutr* 10:340–345, 1991.
19. Swinburn BA: Effect of dietary lipid on insulin action. *Clinical studies. Ann N Y Acad Sci* 683:102–109, 1993.
20. Uusitupa M, Schwab U, Makimattila S, Karhapaa P, Sarkkinen E, Maliranta H, Agren J, Penttila I: Effects of two high-fat diets with different fatty acid compositions on glucose and lipid metabolism in healthy young women. *Am J Clin Nutr* 59:1310–1316, 1994.
21. Vessby B, Uusitupa M, Hermansen K, Riccardi G, Rivelles AA, Tapsell LC, Nalsen C, Berglund L, Louheranta A, Rasmussen BM, Calvert GD, Maffetone A, Pedersen E, Gustafsson IB, Storlien LH: Substituting dietary saturated for monounsaturated fat impairs insulin sensitivity in healthy men and women: The KANWU Study. *Diabetologia* 44:312–319, 2001.
22. Marshall J, Bessesen D, Hamman R: High saturated fat and low starch and fibre are associated with hyperinsulinaemia in a non-diabetic population: The San Luis Valley Diabetes Study. *Diabetologia* 40:430–438, 1997.
23. Feskens EJ, Loeber JG, Kromhout D: Diet and physical activity as determinants of hyperinsulinemia: the Zutphen Elderly Study. *Am J Epidemiol* 140:350–360, 1994.
24. Mayer-Davis EJ, Monaco JH, Hoen HM, Carmichael S, Vitolins MZ, Rewers MJ, Haffner SM, Ayad MF, Bergman RN, Karter AJ: Dietary fat and insulin sensitivity in a triethnic population: the role of obesity. The Insulin Resistance Atherosclerosis Study (IRAS). *Am J Clin Nutr* 65:79–87, 1997.
25. Salmeron J, Hu FB, Manson JE, Stampfer MJ, Colditz GA, Rimm EB, Willett WC: Dietary fat intake and risk of type 2 diabetes in women. *Am J Clin Nutr* 73:1019–1026, 2001.
26. Anderson JW, O'Neal DS, Riddell-Mason S, Floore TL, Dillon DW, Oeltgen PR: Postprandial serum glucose, insulin, and lipoprotein responses to high- and low-fiber diets. *Metabolism* 44:848–854, 1995.
27. Ludwig DS, Pereira MA, Kroenke CH, Hilner JE, Van Horn L, Slattery ML, Jacobs DR: Dietary fiber, weight gain, and cardiovascular disease risk factors in young adults. *JAMA* 282:1539–1546, 1999.
28. Salmeron J, Ascherio A, Rimm EB, Colditz GA, Spiegelman D, Jenkins DJ, Stampfer MJ, Wing AL, Willett WC: Dietary fiber, glycemic load, and risk of NIDDM in men. *Diabetes Care* 20:545–550, 1997.
29. Salmeron J, Manson JE, Stampfer MJ, Colditz GA, Wing AL, Willett WC: Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. *JAMA* 277:472–477, 1997.
30. Meyer KA, Kushi LH, Jacobs DR Jr, Slavin J, Sellers TA, Folsom

- AR: Carbohydrates, dietary fiber, and incident type 2 diabetes in older women. *Am J Clin Nutr* 71:921–930, 2000.
31. Stevens J, Ahn K, Juhaeri, Houston D, Steffan L, Couper D: Dietary fiber intake and glycemic index and incidence of diabetes in African-American and white adults: the ARIC study. *Diabetes Care* 25:1715–1721, 2002.
 32. Montonen J, Knekt P, Jarvinen R, Aromaa A, Reunanen A: Whole-grain and fiber intake and the incidence of type 2 diabetes. *Am J Clin Nutr* 77:622–629, 2003.
 33. Feskens E, Kromhout D: Cardiovascular risk factors and the 25-year incidence of diabetes mellitus in middle-aged men. The Zutphen Study. *Am J Epidemiol* 130:1101–1108, 1989.
 34. Colditz GA, Manson JE, Stampfer MJ, Rosner B, Willett WC, Speizer FE: Diet and risk of clinical diabetes in women. *Am J Clin Nutr* 55:1018–1023, 1992.
 35. Feskens E, Bowles C, Kromhout D: Carbohydrate intake and body mass index in relation to the risk of glucose intolerance in an elderly population. *Am J Clin Nutr* 54:136–140, 1991.
 36. Janket SJ, Manson JE, Sesso H, Buring JE, Liu S: A prospective study of sugar intake and risk of type 2 diabetes in women. *Diabetes Care* 26:1008–1015, 2003.
 37. Jenkins D, Wolever TM, Taylor RH, Barker H, Fielden H, Baldwin JM, Bowling AC, Newman HC, Jenkins AL, Goff DV: Glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr* 34:362–366, 1981.
 38. Liu S, Willett WC, Stampfer MJ, Hu FB, Franz M, Sampson L, Hennekens CH, Manson JE: A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. *Am J Clin Nutr* 71:1455–1461, 2000.
 39. Hu FB, Manson JE, Stampfer MJ, Colditz G, Liu S, Solomon CG, Willett WC: Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. *N Engl J Med* 345:790–797, 2001.
 40. Franz MJ, Bantle JP, Beebe CA, Brunzell JD, Chiasson JL, Garg A, Holzmeister LA, Hoogwerf B, Mayer-Davis E, Mooradian AD, Purnell JQ, Wheeler M: Evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications. *Diabetes Care* 26(Suppl 1):S51–S61, 2003.
 41. Liu S, Manson JE, Stampfer MJ, Hu FB, Giovannucci E, Colditz GA, Hennekens CH, Willett WC: A prospective study of whole-grain intake and risk of type 2 diabetes mellitus in US women. *Am J Public Health* 90:1409–1415, 2000.
 42. Fung TT, Hu FB, Pereira MA, Liu S, Stampfer MJ, Colditz GA, Willett WC: Whole-grain intake and the risk of type 2 diabetes: a prospective study in men. *Am J Clin Nutr* 76:535–540, 2002.
 43. McKeown NM, Meigs JB, Liu S, Wilson PW, Jacques PF: Whole-grain intake is favorably associated with metabolic risk factors for type 2 diabetes and cardiovascular disease in the Framingham Offspring Study. *Am J Clin Nutr* 76:390–398, 2002.
 44. Wolf AM, Colditz GA: Current estimates of the economic cost of obesity in the United States. *Obes Res* 6:97–106, 1998.
 45. Colditz GA, Willett WC, Stampfer MJ, Manson JE, Hennekens CH, Arky RA, Speizer FE: Weight as a risk factor for clinical diabetes in women. *Am J Epidemiol* 132:501–513, 1990.
 46. Colditz GA, Willett WC, Rotnitzky A, Manson JE: Weight gain as a risk factor for clinical diabetes mellitus in women. *Ann Intern Med* 122:481–486, 1995.
 47. Sjostrom CD, Lissner L, Wedel H, Sjostrom L: Reduction in incidence of diabetes, hypertension and lipid disturbances after intentional weight loss induced by bariatric surgery: the SOS Intervention Study. *Obes Res* 7:477–484, 1999.
 48. Pories WJ, MacDonald KG Jr, Morgan EJ, Sinha MK, Dohm GL, Swanson MS, Barakat HA, Khazanie PG, Leggett-Frazier N, Long SD, et al.: Surgical treatment of obesity and its effect on diabetes: 10-y follow-up. *Am J Clin Nutr* 55(2 Suppl):582S–585S, 1992.
 49. Henry RR, Gumbiner B: Benefits and limitations of very-low-calorie diet therapy in obese NIDDM. *Diabetes Care* 14:802–823, 1991.
 50. Wing RR, Blair EH, Bononi P, Marcus MD, Watanabe R, Bergman RN: Caloric restriction per se is a significant factor in improvements in glycemic control and insulin sensitivity during weight loss in obese NIDDM patients. *Diabetes Care* 17:30–36, 1994.
 51. Resnick HE, Valsania P, Halter JB, Lin X: Relation of weight gain and weight loss on subsequent diabetes risk in overweight adults. *J Epidemiol Community Health* 54:596–602, 2000.
 52. Moore LL, Vioni AJ, Wilson PW, D'Agostino RB, Finkle WD, Ellison RC: Can sustained weight loss in overweight individuals reduce the risk of diabetes mellitus? *Epidemiology* 11:269–273, 2000.
 53. Mikines KJ, Sonne B, Tronier B, Galbo H: Effects of acute exercise and detraining on insulin action in trained men. *J Appl Physiol* 66:704–711, 1989.
 54. Rosenthal M, Haskell WL, Solomon R, Widstrom A, Reaven GM: Demonstration of a relationship between level of physical training and insulin-stimulated glucose utilization in normal humans. *Diabetes* 32:408–411, 1983.
 55. King DS, Dalsky GP, Staten MA, Clutter WE, Van Houten DR, Holloszy JO: Insulin action and secretion in endurance-trained and untrained humans. *J Appl Physiol* 63:2247–2252, 1987.
 56. Hollenbeck CB, Haskell W, Rosenthal M, Reaven GM: Effect of habitual physical activity on regulation of insulin-stimulated glucose disposal in older males. *J Am Geriatr Soc* 33:273–277, 1985.
 57. Seals DR, Hagberg JM, Allen WK, Hurley BF, Dalsky GP, Ehsani AA, Holloszy JO: Glucose tolerance in young and older athletes and sedentary men. *J Appl Physiol* 56:1521–1525, 1984.
 58. LeBlanc J, Nadeau A, Richard D, Tremblay A: Variations in plasma glucose, insulin, growth hormone and catecholamines in response to insulin in trained and non-trained subjects. *Metabolism* 31:453–456, 1982.
 59. Hu FB, Sigal RJ, Rich-Edwards JW, Colditz GA, Solomon CG, Willett WC, Speizer FE, Manson JE: Walking compared with vigorous physical activity and risk of type 2 diabetes in women: a prospective study. *JAMA* 282:1433–1439, 1999.
 60. Manson JE, Nathan DM, Krolewski AS, Stampfer MJ, Willett WC, Hennekens CH: A prospective study of exercise and incidence of diabetes among US male physicians. *JAMA* 268:63–67, 1992.
 61. Manson JE, Rimm EB, Stampfer MJ, Colditz GA, Willett WC, Krolewski AS, Rosner B, Hennekens CH, Speizer FE: Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. *Lancet* 338:774–778, 1991.
 62. Burchfiel CM, Sharp DS, Curb JD, Rodriguez BL, Hwang LJ, Marcus EB, Yano K: Physical activity and incidence of diabetes:

- the Honolulu Heart Program. *Am J Epidemiol* 141:360–368, 1995.
63. Helmrich SP, Ragland DR, Leung RW, Paffenbarger RS: Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. *N Engl J Med* 325:147–152, 1991.
 64. Wareham NJ, Wong MY, Day NE: Glucose intolerance and physical inactivity: the relative importance of low habitual energy expenditure and cardiorespiratory fitness. *Am J Epidemiol* 152:132–139, 2000.
 65. Howard AA, Arnsten JH, Gourevitch MN: Effect of alcohol consumption on diabetes mellitus: a systematic review. *Ann Intern Med* 140:211–219, 2004.
 66. Wei M, Gibbons LW, Mitchell TL, Kampert JB, Blair SN: Alcohol intake and incidence of type 2 diabetes in men. *Diabetes Care* 23:18–22, 2000.
 67. Nakanishi N, Suzuki K, Tatara K: Alcohol consumption and risk for development of impaired fasting glucose or type 2 diabetes in middle-aged Japanese men. *Diabetes Care* 26:48–54, 2003.
 68. Ajani UA, Hennekens CH, Spelsberg A, Manson JE: Alcohol consumption and risk of type 2 diabetes mellitus among US male physicians. *Arch Intern Med* 160:1025–1030, 2000.
 69. Conigrave KM, Hu BF, Camargo CA Jr, Stampfer MJ, Willett WC, Rimm EB: A prospective study of drinking patterns in relation to risk of type 2 diabetes among men. *Diabetes* 50:2390–2395, 2001.
 70. Facchini F, Chen YD, Reaven GM: Light-to-moderate alcohol intake is associated with enhanced insulin sensitivity. *Diabetes Care* 17:115–119, 1994.
 71. Mayer EJ, Newman B, Quesenberry CP, Friedman GD, Selby JV: Alcohol consumption and insulin concentrations. Role of insulin in associations of alcohol intake with high-density lipoprotein cholesterol and triglycerides. *Circulation* 88:2190–2197, 1993.
 72. Perry IJ, Wannamethee SG, Shaper AG: Prospective study of serum gamma-glutamyltransferase and risk of NIDDM. *Diabetes Care* 21:732–737, 1998.
 73. Manolio TA, Savage PJ, Burke GL, Liu KA, Wagenknecht LE, Sidney S, Jacobs DR Jr, Roseman JM, Donahue RP, Oberman A: Association of fasting insulin with blood pressure and lipids in young adults. The CARDIA study. *Arteriosclerosis* 10:430–436, 1990.
 74. Avogaro A, Tiengo A: Alcohol, glucose metabolism and diabetes. *Diabetes Metab Rev* 9:129–146, 1993.
 75. Ronnema T, Ronnema EM, Puukka P, Pyorala K, Laakso M: Smoking is independently associated with high plasma insulin levels in nondiabetic men. *Diabetes Care* 19:1229–1232, 1996.
 76. Facchini FS, Hollenbeck CB, Jeppesen J, Chen YD, Reaven GM: Insulin resistance and cigarette smoking. *Lancet* 339:1128–1130, 1992.
 77. Nilsson PM, Lind L, Pollare T, Berne C, Lithell HO: Increased level of hemoglobin A1c, but not impaired insulin sensitivity, found in hypertensive and normotensive smokers. *Metabolism* 44:557–561, 1995.
 78. Sargeant LA, Khaw KT, Bingham S, Day NE, Luben RN, Oakes S, Welch A, Wareham NJ: Cigarette smoking and glycaemia: the EPIC-Norfolk Study. *European Prospective Investigation into Cancer. Int J Epidemiol* 30:547–554, 2001.
 79. Barrett-Connor E, Khaw KT: Cigarette smoking and increased central adiposity. *Ann Intern Med* 111:783–787, 1989.
 80. Shimokata H, Muller DC, Andres R: Studies in the distribution of body fat. III. Effects of cigarette smoking. *JAMA* 261:1169–1173, 1989.
 81. Rimm EB, Manson JE, Stampfer MJ, Colditz GA, Willett WC, Rosner B, Hennekens CH, Speizer FE: Cigarette smoking and the risk of diabetes in women. *Am J Public Health* 83:211–214, 1993.
 82. Wannamethee SG, Shaper AG, Perry IJ: Smoking as a modifiable risk factor for type 2 diabetes in middle-aged men. *Diabetes Care* 24:1590–1595, 2001.
 83. Manson JE, Ajani UA, Liu S, Nathan DM, Hennekens CH: A prospective study of cigarette smoking and the incidence of diabetes mellitus among US male physicians. *Am J Med* 109:538–542, 2000.
 84. Nakanishi N, Nakamura K, Matsuo Y, Suzuki K, Tatara K: Cigarette smoking and risk for impaired fasting glucose and type 2 diabetes in middle-aged Japanese men. *Ann Intern Med* 133:183–191, 2000.
 85. Qiao Q, Valle T, Nissinen A, Tuomilehto J: Smoking and the risk of diabetes in elderly Finnish men. Retrospective analysis of data from a 30-year follow-up study. *Diabetes Care* 22:1821–1826, 1999.
 86. Eriksson KF, Lindgarde F: Prevention of type 2 (non-insulin-dependent) diabetes mellitus by diet and physical exercise. The 6-year Malmo feasibility study. *Diabetologia* 34:891–898, 1991.
 87. Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, Hu ZX, Lin J, Xiao JZ, Cao HB, Liu PA, Jiang XG, Jiang YY, Wang JP, Zheng H, Zhang H, Bennett PH, Howard BV: Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. *Diabetes Care* 20:537–544, 1997.
 88. Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hamalainen H, Ilanne-Parikka P, Keinanen-Kiukkaanniemi S, Laakso M, Louheranta A, Rastas M, Salminen V, Uusitupa M: Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 344:1343–1350, 2001.
 89. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, Nathan DM; Diabetes Prevention Program Research Group: Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 346:393–403, 2002.
 90. WHO/FAO: “Diet, Nutrition and the Prevention of Chronic Diseases: Report of a Joint WHO/FAO Expert Consultation.” Geneva: WHO/FAO, pp 72–79, 2003.
 91. Satterfield DW, Volansky M, Caspersen CJ, Engelgau MM, Bowman BA, Gregg EW, Geiss LS, Hoesy GM, May J, Vinicor F: Community-based lifestyle interventions to prevent type 2 diabetes. *Diabetes Care* 26:2643–2652, 2003.
 92. Goodman RM, Speers MA, McLeroy K, Fawcett S, Kegler M, Parker E, Smith SR, Sterling TD, Wallerstein N: Identifying and defining the dimensions of community capacity to provide a basis for measurement. *Health Educ Behav* 25:258–278, 1998.
 93. Satterfield DW, Lofton T, May JE, Bowman BA, Alfaro-Correa A, Benjamin C, Stankus M: Learning from listening: common concerns and perceptions about diabetes prevention among diverse American populations. *J Public Health Manage Pract* 9:S56–S63, 2003.

94. DHHS, NIH, NIDDK, First Gov, CDC: National Diabetes Education Program. Accessed July 2005. <http://ndep.nih.gov/>
95. Garfield SA, Malozowski S, Chin MH, Narayan KM, Glasgow RE, Green LW, Hiss RG, Krumholz HM: Considerations for diabetes translational research in real-world settings. *Diabetes Care* 26:2670–2674, 2003.
96. Struthers R, Hodge FS, De Cora L, Geishirt-Cantrell B: The experience of native peer facilitators in the campaign against type 2 diabetes. *J Rural Health* 19:174–180, 2003.
97. Harwell TS, Dettori N, Flook BN, Priest L, Williamson DF, Helgerson SD, Gohdes D: Preventing type 2 diabetes: perceptions about risk and prevention in a population-based sample of adults \geq 45 years of age. *Diabetes Care* 24:2007–2008, 2001.
98. Mercer SL, Green LW, Rosenthal AC, Husten CG, Khan LK, Dietz WH: Possible lessons from the tobacco experience for obesity control. *Am J Clin Nutr* 77(4 Suppl):1073S–1082S, 2003.
99. Schmid TL, Pratt M, Howze E: Policy as intervention: environmental and policy approaches to the prevention of cardiovascular disease. *Am J Public Health* 85:1207–1211, 1995.
100. Nestle M, Jacobson MF: Halting the obesity epidemic: a public health policy approach. *Public Health Rep* 115:12–24, 2000.
101. Williamson DF, Vinicor F, Bowman BA: Primary prevention of type 2 diabetes mellitus by lifestyle intervention: implications for health policy. *Ann Intern Med* 140:951–957, 2004.

Received August 6, 2004; revision accepted January 29, 2005