Pediatric Diabetes 2003: 4: 38–56
Printed in Denmark. All rights reserved

Pediatric Diabetes
ISSN 1399-543X

Review Article

Prevention of type 2 diabetes in young people: a theoretical perspective

Huang TT-K, Goran MI. Prevention of type 2 diabetes in young people: a theoretical perspective.
Pediatric Diabetes 2003: 4: 38–56. © Blackwell Munksgaard, 2003

Abstract: Type 2 diabetes in youth is an increasing public health concern, especially in certain minority populations. The current paper consists of four sections. First, we establish the significance of the problem by presenting an overview of epidemiological and physiological evidence. Second, we discuss behavioral issues relevant to the prevention of type 2 diabetes in youth. Third, a qualitative review of existing prevention interventions specific to type 2 diabetes in youth is presented. Results suggest that modest improvements in social cognitive, dietary, and exercise outcomes are possible with diabetes intervention studies, although beneficial changes are difficult to sustain over the long term. Although theoretical frameworks are not always explicit, most studies have utilized elements of the social cognitive theory. Less attention has been paid to sociocultural and community organization variables. Finally, the paper discusses issues of risk definition and intervention sustainability, and presents a comprehensive, theoretically diverse model for the prevention of type 2 diabetes in youth. In summary, we suggest that theories of the natural history and pathophysiology of type 2 diabetes are important to identify modifiable risk factors, while theories of behavioral change are essential to modify the risk factors identified. The combination of sound physiological and behavioral theories should form the basis of prevention intervention design. In addition, an ecologic approach that takes into consideration the dynamic interactions of personal, social, and environmental factors would best promote the long-term adoption of healthful behaviors in a supportive, meaningful, and personally enjoyable context.

Terry T-K. Huang[1] and Michael I. Goran[2]

[1]Energy Metabolism Laboratory, Jean Mayer USDA Human Nutrition Research center, Tufts University, Boston, USA ; and [2]Institute for Health Promotion & Disease Prevention Research, Departments of Preventive Medicine and Physiology and Biophysics, Keck School of Medicine, University of Southern California, USA

Key words: adherence – pathophysiology – prevention – type 2 diabetes – youth

Corresponding author: Michael I. Goran PhD, Department of Preventive Medicine, University of Southern California, 1540 Alcazar Street, CHP208D, Los Angeles, CA 90089, USA.
Tel: 323 442-3027; fax: 323 442-3048;
e-mail: goran@hsc.usc.edu

Submitted 23 August 2002. Accepted for publication 23 December 2002

Epidemiological and physiological overview

Type 2 diabetes mellitus as an increasing health problem in young people

Type 2 diabetes is a serious condition that can have a detrimental impact on health, quality of life, and life expectancy. Due to its chronic nature, which necessitates lifelong maintenance, it is also a tremendous economic burden on the health care system. Type 2 diabetes is diagnosed based on multiple measures of elevated fasting plasma glucose (>126 mg/dL) or an abnormal plasma or serum glucose following an (up to) 75 g oral glucose tolerance test (>200 mg/dL at 2 h). Approximately 15 million Americans are estimated to suffer from type 2 diabetes, which constitutes approximately 5% of the total population. However, due to the lack of symptoms in the early stages of disease pathology, only half of them are currently diagnosed. In adults, onset of type 2 diabetes frequently occurs 7 yr or more before diagnosis (1). In young people, most are referred because of incidental findings of glucosuria or hyperglycemia (1–3). Demographically, the prevalence of type 2 diabetes is particularly high in Native

American, African-American, Hispanic, and South Pacific Islander populations (4).

Although type 2 diabetes has historically been characterized as adult-onset diabetes, it has been shown to be on the rise in young people in recent years, comprising some 30% of new cases of diabetes in the second decade of life (5). The mean age at diagnosis of type 2 diabetes in young people is 12–14 yr (1, 3). The recent increase in the incidence of type 2 diabetes is thought to be related to the increasing prevalence of obesity in young people (1), which has reached epidemic proportions in the US and is disproportionately high in young people of ethnic minority background (6). Alongside a positive family history and ethnicity, obesity is a major risk factor for the development of type 2 diabetes in youth (7).

The incidence of type 2 diabetes in young people is particularly high in minority populations (1, 5, 8). Pima Indians have the highest documented prevalence of type 2 diabetes in the world. Pima youth in the age range 5–14 yr have an incidence rate of type 2 diabetes of 1 in 1000 person-years and those aged 15–24 yr have an incidence rate of 9 in 1000 person-years (9). Type 2 diabetes also has been documented in First Nation youth in Manitoba, with a prevalence of at least 0.53 per 1000 in the age range 7–14 yr (2). Among Japanese young people, type 2 diabetes is seven times more frequent than type 1 diabetes, showing a 30-fold increase in incidence over the past 20 yr which is concomitant with changing food patterns and increasing obesity (10, 11). Similar alarming statistics have also been obtained recently in other parts of the world (12).

Furthermore, a review of one Southern California pediatric diabetes clinic population demonstrated that 21% of diabetic patients of Mexican-American descent had type 2 diabetes, compared with only 3% of their white counterparts (1). In another study, 69% of pediatric patients with type 2 diabetes in the Greater Cincinnati area were African-American where the African-American population constituted only 14.5% of the general population. The odds ratio for the development of type 2 diabetes in African-American compared with white youths was 3.5 in boys and 6.1 in girls (3). Interestingly, African-American girls have been shown to be at particularly high risk for obesity, compared with boys and white girls (13). In San Antonio, Texas, 123 youth with type 2 diabetes were diagnosed between 1990 and 1998, accounting for 18% of all new diabetes cases (14). Type 2 diabetes now comprises some 30% of new cases of diabetes in the second decade of life (5) and may be higher among minority youth and adolescents.

Recently, the American Diabetes Association (ADA) defined 'prediabetes' as a condition of serious health threat (ADA Website – Pre-Diabetes, 19 April 2002: http://www.diabetes.org/main/info/prediabetes.jsp). Long-term damages to the body may occur during prediabetes, before the actual onset of type 2 diabetes. Prediabetes is defined as a fasting plasma glucose level greater than or equal to 110 mg/dL and under 126 mg/dL (i.e., impaired fasting glucose) or a 2- h post-oral glucose tolerance test (OGTT) plasma glucose level greater or equal to 140 mg/dL and under 200 mg/dL (i.e., impaired glucose tolerance). In high-risk young people, prediabetes may be quite prevalent. For instance, Sinha et al. (15) showed that 25% of 55 obese children (4–10 yr of age) and 21% of 112 obese adolescents (11–18 yr of age) suffered from impaired glucose tolerance. Among the children and adolescents with impaired glucose tolerance, 51% were white, 30% were African-American, and 19% were Hispanic. In addition, 4% of the obese adolescents had type 2 diabetes, and were either African-American or Hispanic. If left untreated, young people who suffer from prediabetes are likely to develop diabetes and diabetes-related complications at a young age, posing a serious public health burden. Therefore, early identification of youth with prediabetes is imperative.

Pathophysiology of type 2 diabetes in young people

Type 2 diabetes predominantly results from a combination of insulin resistance and an inability of the pancreas to maintain adequate compensatory insulin secretion (16–18). In brief, three major physiological abnormalities contribute to the development of hyperglycemia: (i) peripheral insulin resistance in muscle and adipose tissue; (ii) excessive hepatic glucose production; and (iii) impaired insulin secretion (16–20).

Although the pathophysiology of type 2 diabetes is fairly well known in adults, in young people, it is still not entirely clear. The pubertal transition is a time during which rapid and dynamic changes occur in various metabolic systems, including hormonal regulation, body fat and fat distribution, and insulin resistance. The time of greatest risk during adolescence appears to be mid-puberty, when insulin resistance worsens and further increases risk (21) (Fig. 1). We have recently studied 33 boys and 27 girls (32 white, 28 African-American) to determine the effect of puberty on insulin resistance (22). All were at Tanner stage 1 at baseline, and after 2 yr of follow-up, 29 remained at Tanner stage 1, whereas 31 had progressed to Tanner stage 3 or 4. In those progressing to Tanner stage 3 or 4, insulin sensitivity fell significantly by 32%, and disposition index, a measure of beta-cell functioning, fell by 27% at 2-yr follow-up. These longitudinal findings are consistent with previous cross-sectional studies (21, 23–26), suggesting that failure to adapt to pubertal insulin resistance or to recover following pubertal insulin resistance may lead to sustained insulin resistance and beta-cell failure, eventually leading to the onset of diabetes.

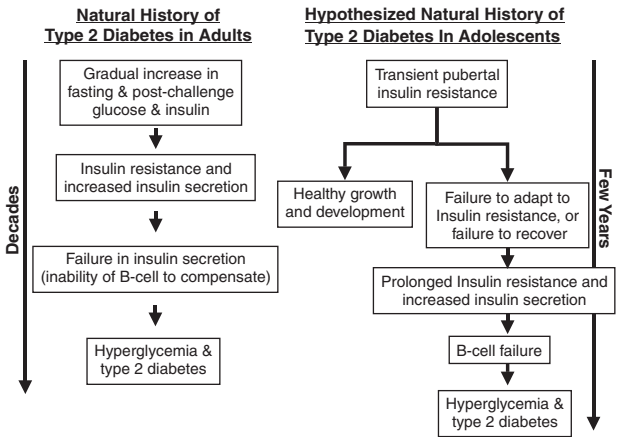

Fig. 1. Natural history of type 2 diabetes in young people and adults.

The presence of polycystic ovary syndrome (PCOS) may contribute in part to the increased risk of insulin resistance and type 2 diabetes among females. PCOS affects approximately 4–12% of reproductive-age women (27) and is increasingly recognized in adolescent girls with hyperandrogenism (28–30). Lewy et al. (31) reported a 50% reduction in peripheral tissue insulin sensitivity, increased hepatic insulin resistance, and compensatory hyperinsulinemia among 12-yr-old girls with PCOS. Therefore, PCOS-associated metabolic abnormalities occur early in life and appear to play a role in the female preponderance to type 2 diabetes.

It is clear that type 2 diabetes is a very different disease when expressed during adolescence over the course of just several years (7). The unique physiological course of type 2 diabetes in young people underscores the important need for childhood prevention interventions that target prepubertal or early pubertal children.

Obesity and type 2 diabetes

Obesity has reached epidemic proportions in the United States (32) and is a serious risk factor for the development of type 2 diabetes, mediated by the high rate of insulin resistance in obese individuals (33–37). From 1976 to 1987, the prevalence of obesity in young people aged 6–11 yr increased by 54% and the prevalence of superobesity and obesity in those aged 12–21 yr increased by 64% (38, 39). The latest report suggests that overweight prevalence in 1998 was 21.5% among African-Americans, 21.8% among Hispanics, and 12.3% among Whites (6). Although the definitions of obesity and overweight may vary, the increased trend is consistent across many studies. The increase in obesity, particularly in minorities, may be an important reason for the increase in type 2 diabetes in youth (8).

Increasing evidence suggests that obesity and type 2 diabetes are now a worldwide issue, affecting both developed and developing countries (8, 40–42). As countries undergo swift socioeconomic change, a shift from communicable diseases to non-communicable diseases is apparent. The coexistence of childhood under-nutrition and overweight is also common (43, 44). Whereas in developed countries, the poor suffer disproportionately from obesity and type 2 diabetes, in developing countries, the more affluent are usually the first to experience drastic lifestyle changes and more susceptible to obesity and obesity-related diseases. However, this is expected to change as the socioeconomic distribution changes in developing countries.

Obesity seems to precede type 2 diabetes and may provoke diabetes in genetically predisposed individuals (33, 45, 46). In healthy young people, increased adiposity is associated with increased fasting insulin and decreased insulin sensitivity, suggesting that body fat may be related to risk of type 2 diabetes (13). In addition to the degree of obesity, the duration of obesity and location of body fat are also important risk factors for type 2 diabetes (47). Maximum lifetime body mass index (BMI) is associated cross-sectionally with type 2 diabetes, independent of current BMI (48). The risk of type 2 diabetes is positively related to the number of years of being obese (49). Furthermore, the site of body fat, particularly central fat, is a predictor of type 2 diabetes, independent of the presence of obesity (50–58). In children, the relative roles of different compartments of adiposity are less clear (59), and their relation to type 2 diabetes still needs to be further investigated.

Finally, we note briefly the several adipose tissue-related hormones, such as leptin, adiponectin, and resistin, which have been studied in relation to fat deposition or insulin resistance. Leptin has been shown to be inversely related to adiposity in children (60, 61), but the relationship is very inconsistent across studies. We have found that leptin resistance may be present early in life in some young people (62), which may in part explain why the relationship between leptin and adiposity is unclear across studies. Adiponectin has been shown to be inversely related to adiposity and insulin resistance in adults (63–66). In children, one recent study suggests that hypoadiponectinemia may be a consequence of childhood obesity (67). In the same study, no relationship was found between adiponectin and insulin resistance, though a crude measure of insulin sensitivity was used. Resistin is thought to link obesity to type 2 diabetes by interfering with insulin signaling and therefore decreasing glucose uptake and insulin sensitivity (68). Its role, however, remains very controversial as resistin gene expression is virtually absent in adipose tissue of obese or diabetic individuals (69, 70). To our knowledge, the role of resistin has not been examined in youth.

Fetal origins of type 2 diabetes

Growth retardation in the intrauterine environment may predispose individuals to be at increased risk for

obesity, hypertension, metabolic syndrome, type 2 diabetes, and cardiovascular disease (CVD) later on in life (17, 71–74). Fetal malnutrition is thought to relate to metabolic syndrome via negative effects on functioning of the kidney, pancreas, hypothalamic–pituitary–adrenal axis, sympathetic system, and muscle, liver, and adipose tissue (75). Though there is mounting evidence to support this hypothesis as a potential mechanism for increased susceptibility to type 2 diabetes, what is less clear is how fetal growth retardation interacts with early postnatal and childhood development to increase susceptibility to disease. If this hypothesis were true, implications for the developing world could be tremendous.

Exposure to maternal gestational diabetes has also been noted as a predictor for childhood obesity and type 2 diabetes (76–78). Gestational diabetes creates a hyperglycemic intrauterine environment and may alter glucose and insulin metabolism in the offspring. Maternal antepartum glucose levels and fetal and neonatal insulin levels are predictive of obesity and diabetes risk in childhood (79). However, no cut-offs are available to identify offspring of highest risk. There is also no information on the effect of management strategies on long-term offspring health outcomes. Prospective studies on mother–child pairs exposed to gestational diabetes are warranted.

Behavioral risk factors

Diet, physical activity, and physical inactivity are the most modifiable risk factors in the prevention of type 2 diabetes. Recent intervention studies in adults have shown that intensive lifestyle training promoting physical activity and healthy eating can reduce diabetes risks and incidence (80–82) across different age, racial, and ethnic groups, and that the effect of lifestyle intervention is greater than pharmacological therapy. For instance, the Diabetes Prevention Program Research Group (82) published results in adults that suggested a 58% reduction in type 2 diabetes incidence in those undergoing dietary and physical activity intervention, and a 31% reduction in those taking metformin, compared with placebo. In young people, lifestyle factors are particularly worthy of emphasis because the nurturing of a habitual healthy way of life must start young and could yield tremendous long-term benefits. Replication of the Diabetes Prevention Program is currently underway in young people.

Diet

Poor diet has long been considered a major behavioral factor in the development of both obesity and type 2 diabetes (50). Both total caloric intake and certain dietary components such as carbohydrates and fat have been implicated (47). Ecological studies of severe food shortages during wars indicated that diabetes mortality and morbidity declined abruptly with decreased caloric intake (83–85). However, such studies do not offer sufficient information to determine which dietary components may be most relevant.

Although relatively few data are available for young people, diet may also play a role in increasing obesity, insulin resistance, and glucose intolerance in that group, especially in minority populations where these risk factors of type 2 diabetes are most prevalent. For instance, one study found that a sample of Mexican-American 9-yr-olds had higher fat and protein intake and lower daily fruit and vegetable intake than non-Hispanic white youths. The authors concluded that these factors might explain why Mexican-American youths are more likely to be obese than white youths, exacerbating their risk for type 2 diabetes (86).

Recently, research into the significance of junk food and soda pop consumption in relation to childhood obesity has been emerging (87, 88). In general, consumption of junk/fast foods and soda pop is inversely related to consumption of more nutritious foods such as milk, fruits, and vegetables (87, 89–91). However, neither the correlation nor the causal relationship between junk food/soda pop consumption and obesity has been consistently identified (92). One study suggests that glucose and insulin response may be a function of the interaction between obesity and soda pop consumption (93). In the short term, however, healthy non-obese children and adolescents adapt very quickly and efficiently to drastic changes in fat and carbohydrate intakes (94).

The role of diet remains unclear and controversial. Dietary factors may be only marginally associated with body fat in youth (88, 95) and do not seem to account for the difference in insulin sensitivity between African-American and Caucasian youth (96). The diversity of dietary behavior in the population and difficulty in measuring it may also contribute to the weak correlations between diet and disease risks.

Physical activity

Besides diet, lack of physical activity is considered another major behavioral risk factor for the development of obesity and type 2 diabetes. Both observational (97–116) and ecologic studies (97–100) suggest that type 2 diabetes prevalence is consistently lower in populations with higher levels of habitual physical activity. In large longitudinal studies in adults, the benefit of regular physical activity is well documented and suggests a strong dose–response (113, 116).

It has been postulated that the protective effect of physical activity on the development of type 2 diabetes may be due to the improvement of insulin resistance (117, 118), particularly among those with high insulin levels to begin with, as commonly seen in

obese individuals. At least two studies showed that patients who start an exercise program with high insulin levels respond with a drop in insulin levels (119, 120). In addition to the improvement of insulin resistance, a decrease in intra-abdominal fat, fasting glucose, free fatty acids, and an increase in insulin sensitivity and glucose tolerance are all potential benefits of exercise training (121–123).

In young people, at least two studies have shown a positive effect of physical training on decreasing fasting insulin concentrations (124) and stabilizing visceral fat accumulation (125). In addition, a recent review by Goran et al. (126) on the role of physical activity in the prevention of obesity in young people revealed that studies collectively suggest that aerobic exercise can reduce body fat in young people independently of dietary modification and that strength training may also yield metabolically beneficial effects on insulin action. Nevertheless, it was also noted that the beneficial effects of physical training often dissipated during postintervention follow-ups, suggesting a dire need for research into strategies that facilitate the maintenance of healthy activity and dietary behavior changes in free-living situations over the long term.

Physical inactivity

In addition to physical activity, behaviors that result in physical inactivity increase the risk of becoming overweight, and minority youth demonstrate higher levels of inactivity (127). Three mechanisms are suggested to link sedentary activity and obesity (128): (i) reduced energy expenditure; (ii) increased dietary energy intake from eating during sedentary activities or from the effects of food advertising; and (iii) decreased metabolic rate during sedentary activities. For instance, TV-viewing has been associated with obesity in young people and adolescents (129) and a reduction of the amount of time young people spend in such physical inactivity has been shown to reduce obesity (130, 131). However, we have found that energy expenditure does not relate independently to gain in adiposity in children (132) and the effects of diet and decreased metabolic rate currently lack conclusive evidence (133). We have found that when recreational activity time, physical fitness, and sedentary activity time (TV/computer/video usage) are examined simultaneously, only recreational activity time is independently related to total body fat (Neville K, Huang TT-K, Ball G, Spruijt-Metz D, Goran MI, unpublished data). The most promising finding on the importance of sedentary activity to date comes from a randomized controlled trial, which found that manipulation of TV-viewing time alone results in decreasing adiposity (134). No studies have examined physical inactivity in relation to type 2 diabetes in youth specifically.

Maternal influences

Recently, emerging evidence suggests that several maternal factors may be associated with childhood obesity, including maternal smoking during pregnancy (135–139), breastfeeding behavior (140–143), and parental dietary behavior and control of feeding (144). Several mechanisms may be possible linking each of the maternal factors to childhood obesity. However, there is currently no evidence to directly link these factors to type 2 diabetes in youth. Therefore, whether obesity is always a mediator in these cases is not clear.

Maternal smoking may be related to poor diet during pregnancy (145) and suboptimal nutritional environment *in utero* could result in fetal growth retardation (146, 147). Intrauterine exposure to nicotine may also alter cholinergic and catecholaminergic neurotransmitter systems of the brain (148), thus resulting in behavioral deficits such as poor impulse control.

Breastfeeding could affect child and adolescent susceptibility to obesity through lowered energy per volume of breast milk compared with formula (149) and the potential ability of breast milk to modify growth factors that inhibit adipocyte differentiation (150). Overfeeding may also be a problem when not breastfeeding, resulting in early programming of child eating behavior (149).

Finally, parental control and limitation can override children's natural appetite signals, resulting in lowered self-regulation of energy intake and hence increased susceptibility to obesity (144, 151). Gender, cultural, and socioeconomic variations would likely modify these associations, however, and need to be further studied.

Qualitative review of prevention interventions of type 2 diabetes in young people (Table 1)

Given the relatively recent emergence of type 2 diabetes in young people, there have been few interventions designed to help prevent or delay its onset. In order to begin seriously designing effective prevention interventions, it is important to consider the natural history and pathophysiology of type 2 diabetes to identify modifiable risk factors, as well as theories of behavioral change to modify the risk factors identified. The combination of sound physiological and behavioral theories should form the basis of prevention intervention design (152). In this section, we review prevention intervention studies aimed at reducing diabetes-related risk factors among healthy non-diabetic young people.

Selection of studies

Seven articles on prevention interventions of type 2 diabetes in young people, identified through Medline (1966 through present), PsychInfo (1887 through

Table 1. Design and major findings of secondary prevention interventions of type 2 diabetes in young people

Study	Subjects	Intervention							Duration	Major Findings	Theoretical Approach
		NE	BM	PA	FS	PI	CI	MM			
Cook et al. (153)	>200 youths in grades K-2	X		X	X				2 yr	N/A	Didactic teaching, skill learning
Holcomb et al. (154, 155)	1114 5th grade Hispanics (mostly) and others	X	X						2 months, 1 month follow-up	Increased diabetes-related knowledge, self-efficacy, and exercise behavior, decreased fat intake	Social cognitive, sociocultural
Macaulay et al. (161)	458 6–12 yr-old Mohawks	X	X	X		X	X	X	3 yr	N/A	Social cognitive, social support, Precede-Proceed, community organization
Marlow et al. (157)	24 13–18 yr-olds	X		X					2 months	Increased diabetes-related knowledge	Modeling, social influence, transtheoretical, sociocultural
McKenzie et al. (158)	35 7–12 yr-old Mexican Americans	X				X			4 months	Trend to better health behavior, increased awareness of need for lifestyle change	Knowledge-based, behavior-modeling, sociocultural
Trevino et al. (159)	102 4th grade Mexican-Americans	X		X	X				9 months and ongoing	Increased diabetes-related knowledge and decreased fat intake	Social cognitive
Teufel and Ritenbaugh (160)	370–440 Zuni 9th-12th graders	X		X	X		X		2 yr and ongoing	Decreased BMI, decreased sugared beverage intake, decreased sitting pulse rate, increased glucose/insulin ratio (reduced hyperinsulinemia)	Social cognitive, social support, Precede-Proceed, community organization

NE, nutritional education; BM, behavior modification; PA, physical activity; FS, food service; PI, parental involvement; CI, community involvement; MM, mass media.

present), and Eric (1966 through present), were published between 1997 and 1999. These studies targeted specifically Hispanic and Native young people living in Canada and the US (153–160). Table 1 summarizes the characteristics and major findings of these studies.

Study subjects and design

Of the seven studies addressing type 2 diabetes specifically, five were targeted at primary school children (153, 155, 159, 161, 162) and two were designed for adolescents (157, 160). Three studies were implemented mostly among Mexican-American youths (155, 159, 162), while the rest involved Native American or First Nations Canadian youths (153, 157, 161). Samples of all seven studies were based on self-selection. The duration of intervention ranged from 2 months to 3 yr, with follow-up periods up to 2 yr. One study compared intervention effects between one group with teacher training and another without teacher training (153).

Intervention components

In all seven studies under review, nutrition education was included as part of the intervention. Nutrition education typically took place as a combination of dietetic counseling and/or basic nutritional knowledge, reading food labels, and determining proper portion sizes. Some studies also included the use of some form of behavior modification, particularly contingency management. Physical activity was also included as an active component in five interventions (153, 156, 157, 159, 160). Merely encouraging young people or their families to exercise is not considered an active component of program in the current review. Two studies involved parents in their intervention (161, 162). Two studies (diabetes specific) involved food service delivery (153, 159), and one involved modification of food supply and access from less healthy to more healthy choices in the schools (160). In addition, one intervention involved the participation of either the whole community and/or mass media (161). The Zuni program saw the school as a community and received strong support from school administration (160). A very strong top-down approach is thus evident in this scenario (160).

Major findings

In general, though effects on long-term morbidity are not yet known from type 2 diabetes prevention interventions in young people, results of social cognitive, behavioral, and selected anthropometric/physiological outcomes appear to be positive. Target outcomes in these published studies have usually included diabetes knowledge, social cognitive adjustment (e.g. self-efficacy), and change in dietary/physical activity behavior. The Zuni program reported positive mid-project BMI, pulse rate, and glucose/insulin outcomes (160). Four of the type 2 diabetes prevention interventions showed an increase in awareness and knowledge regarding type 2 diabetes (155, 157–159). Though the change in absolute scores was small, a large proportion of the study participants seemed to have benefitted from the interventions. One study also suggested an increase in diabetes self-efficacy and exercise behavior (155). Two studies rendered some evidence for the intervention effect on lowered fat intake (155, 159). Unfortunately, because several of these type 2 diabetes-specific interventions are still new and ongoing, no results were reported in two of the six type 2 diabetes studies (153, 161).

Theoretical approaches

Several theoretical frameworks have been used, explicitly or implicitly, by the reviewed studies as the basis of intervention design and implementation. Although most articles failed to give a clear description of the theoretical framework behind the intervention design, based on the description of program procedure, we have identified the use of the following theoretical approaches: social cognitive (154–156, 159, 160), social support (156, 160), social influence (157), Precede-Proceed (156, 160), sociocultural (154, 155, 157, 158), community organization (160, 161), transtheoretical (157), and didactic teaching (153).

Social cognitive theory posits that behavioral change is a result of the interaction of incremental mastery and achievement, outcome expectation, individual coping responses, and various social reinforcements (163). In addition, a gradual development of internal (as opposed to external) gratification for healthy behavior is important to sustain any behavioral change. The social cognitive approach is appropriate in a variety of settings since it focuses on the learning of skills rather than the acquisition of knowledge alone. Several studies in young people have shown that effective programs are skill-based rather than knowledge-based only (164–170).

The Precede-Proceed model addresses predisposing, reinforcing, and enabling factors, as well as environmental and organizational factors, that effect health behaviors (171). It is a robust model that emphasizes comprehensive planning. Working through Precede-Proceed requires the use of deductive thinking, starting with the final consequences and working back to the original causes. In other words, one begins with the desired outcome and determines what causes or precedes it. In order to achieve that objective, diagnosis of the problem should be made in various dimensions, including social, epidemiological, behavioral, environmental, educational, organizational, and administrative dimensions. Once the diagnosis is complete, one may

proceed to the implementation and evaluation of the intervention (171). This model is important for prevention of type 2 diabetes because it attempts to simultaneously take into account diabetes knowledge, behavioral skills, social reinforcement, the availability of healthy foods, and the opportunity for physical activity.

The use of sociocultural models in diabetes prevention studies can be attributed greatly to the fact that the target populations consist of mainly minority youths. Different cultures have different beliefs about illnesses, different languages, and different lifestyle patterns, compared with the mainstream, white culture. In order for these interventions to be effective, cultural differences must be considered and incorporated into the intervention design. Moreover, one of the intervention studies, targeted at young Mohawks (161), involved community organizations and the employment of mass media as a vehicle of health promotion. Community organization models have been suggested as an important component of health promotion and disease prevention. Interventions that incorporate community involvement are more likely to address the health problem with a top-down approach, in addition to the bottom-up health education curriculum. The combination of top-down and bottom-up elements results in more comprehensive and more effective interventions. Mass media is important in the diffusion of intervention. In the one study that utilized mass media, we see that this modality is more of an active intervention component when community organizations are involved.

In order for interventions to achieve sustained success, they must empower the target population to possess a sense of ownership of the process of adopting healthier lifestyle behaviors. When interwoven, social cognitive theory, the Precede-Proceed model, sociocultural models, and ecologic models render a holistic approach to solving the complex and dynamic interaction of personal, situational, and environmental factors. We propose that such an integrated approach may be key to effective prevention of type 2 diabetes in the pediatric population and this area of research deserves further attention.

Limitations

A few noteworthy limitations are inherent in the studies under review. First, many of the studies utilized measures that may not be sensitive enough to detect individual differences in social cognitive variables. Most studies also failed to examine potential mediators of the intervention effect, such as personal meanings of behavior or perceived barriers to behavioral change. In addition, because studies often used inconsistent outcome measures, comparison of results across studies is difficult. Only one study reported physiological or disease-related outcomes such as glucose and insulin concentrations, making it hard to ascertain the clinical benefit of the interventions. Also, most studies suffered from lack of randomization and control. Overall, information is extremely limited in young people with regard to effective prevention strategies. There is insufficient evidence to conclude the relative effectiveness of any program component alone. A combination of intervention elements including dietary education/counseling, physical activity, diabetes knowledge, competence building, social support, and community support will have to be considered concurrently, but future studies are needed to clearly identify effective program elements.

Definition of risk and sustainable intervention

Definition of high risk in young people

In addition to the increased risk associated with pubertal insulin resistance, higher rates of obesity and type 2 diabetes have been shown in ethnic minorities in industrialized countries or distinct populations in developing countries. Therefore, high-risk young people have typically been defined based on their self-reported ethnic background. The disproportionate rate of incidence in different ethnic groups may imply different underlying disease mechanisms (biological and/or environmental) in different segments of the population. However, longitudinal studies in different ethnic groups are required to elucidate this further. Moreover, using ethnicity as a definition of 'high risk' may not be sufficiently specific, since even in heavily affected populations, the odds of developing diabetes are still smaller than that of not developing the condition.

The determination of overweight or obesity has also been used as a way to identify high-risk young people. Although this method may be more specific than a simple ethnic categorization, defining overweight and obesity in young people can be problematic itself. Most anthropometric measures of overweight and obesity, such as body mass index and skinfolds, are arguably more appropriate for comparison research between populations and less appropriate for identifying individuals at risk. More accurate measures of an individual's adiposity, such as computed tomography, magnetic resonance imaging, or dual-energy X-ray absorptiometry, are usually less available in a clinical situation and information from research is not yet sufficient to establish a risk index for young people using these measures. In addition, overweight status as a criterion of risk also suffers from the lack of sensitivity since there are many more overweight individuals compared with those who are actually diabetic. Weight gain is also associated with the onset of puberty. Thus, at least in young people, overweight status alone may not be a sufficient definition of high risk for type 2 diabetes.

A third definition of high risk involves the examination of family history. The presence of type 2 diabetes in a first-degree or second-degree family member is an established risk factor for type 2 diabetes. Pima Indians (172) and Caucasians (173, 174) with at least one diabetic parent have a much higher incidence of type 2 diabetes than those who are equally obese but who do not have a diabetic parent. However, a positive family history of type 2 diabetes may represent a host of risk factors that are genetic, physiologic, demographic, and/or sociocultural in nature. Indeed, in the first edition of *Diabetes in America* (175), numerous family studies were reviewed, with the conclusion that they were limited in value for differentiating the genetic and environmental factors shared by family members.

Finally, high risk can also be construed as possessing clinical or metabolic indicators associated with syndrome X. Lowered insulin sensitivity and insulin secretion are most proximal to the onset of diabetes. Therefore, such measures may be more sensitive and specific than others in identifying high-risk individuals. However, precise measures of insulin resistance are often expensive, time-consuming, and inaccessible. Thus, simple measures of fasting insulin and glucose concentrations have been suggested as proxy measures. In adults, prospective studies have consistently shown that hyperglycemia and hyperinsulinemia strongly predict the development of type 2 diabetes (176–182). Although the predictive utility of fasting insulin and/or glucose concentrations for type 2 diabetes is not entirely clear in young people, recent studies suggest that higher fasting insulin levels are associated with lower insulin sensitivity early on in age (13) and may be the most significant single predictor of insulin sensitivity (183).

As a result of the difficulty in defining high risk of type 2 diabetes using any one criterion, the ADA recommends the use of a combination of criteria (7). Testing recommendations take into account overweight status (defined as BMI >85th percentile for age and sex, weight for height >85th percentile, or weight >120% of ideal [50th percentile] for height) and two of the following risk factors: positive family history of type 2 diabetes, ethnicity (American Indians, African-Americans, Hispanic-Americans, Asians/South Pacific Islanders), or signs of insulin resistance or conditions associated with insulin resistance (acanthosis nigricans, hypertension, dyslipidemia, PCOS). Youth who fit these criteria should be tested every two yr starting at age 10 or at onset of puberty if it occurs earlier in age (7).

Designing type 2 diabetes prevention interventions

Sustained lifestyle changes are essential in order to reverse the clinical risks (particularly insulin resistance) of type 2 diabetes (Fig. 2). However, these changes have been shown generally to be very difficult to maintain over a long period of time, particularly among minority youths, because of a lack of adherence to prescribed regimens (153, 184, 185). Part of the difficulty for individuals in maintaining lifestyle behavior changes may be due to the fact that there are few culturally sensitive and family-based interventions available for minority populations who are at high risk for type 2 diabetes. For example, young African-Americans with diabetes have been found to be less adherent than their white counterparts, because of the lack of emphasis on the needs of African-American communities (186). Similar issues can be extended to Hispanic and Native populations. In addition to cultural considerations, family involvement and age-appropriate contexts are also important underlying elements in an intervention for youth.

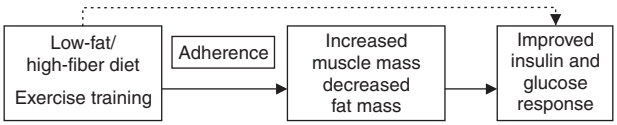

Fig. 2. Predicted change in overweight status and insulin resistance with a controlled diet and exercise training intervention. Dashed line represents the potential pathway.

When designing a prevention intervention for type 2 diabetes, particular emphasis should be given to the theoretical constructs that engage in interactions of personal, social, and environmental factors. When designed based on sound theory, an intervention should promote the adoption of healthful behaviors in a supportive, meaningful, and personally enjoyable context. In addition, in a diabetes prevention study, it is also important to monitor changes in glucose tolerance and insulin resistance. Simply monitoring weight or body fat changes may not be sufficient for detecting benefits on metabolic outcomes.

A comprehensive, theoretical model for prevention of type 2 diabetes in young people

Figure 3 illustrates a comprehensive, theoretical model for the prevention of type 2 diabetes in young people. This model is conceptualized to address diet, physical activity, and glucose monitoring as the principal behavioral prescriptions for young people at high risk for type 2 diabetes. The current model takes into consideration the promotion of adherence to prescribed regimens in individuals' free-living daily situations so as to ensure the long-term benefits of a diet and exercise training program. In addition, given the age group of the target population, adherence issues must be addressed in both young people and their parents.

The proposed model of intervention is designed to promote better adherence in both young people and their parents, so that the true effect of diabetes education, dietary counseling, and exercise training can be observed over time. It incorporates the strengths of

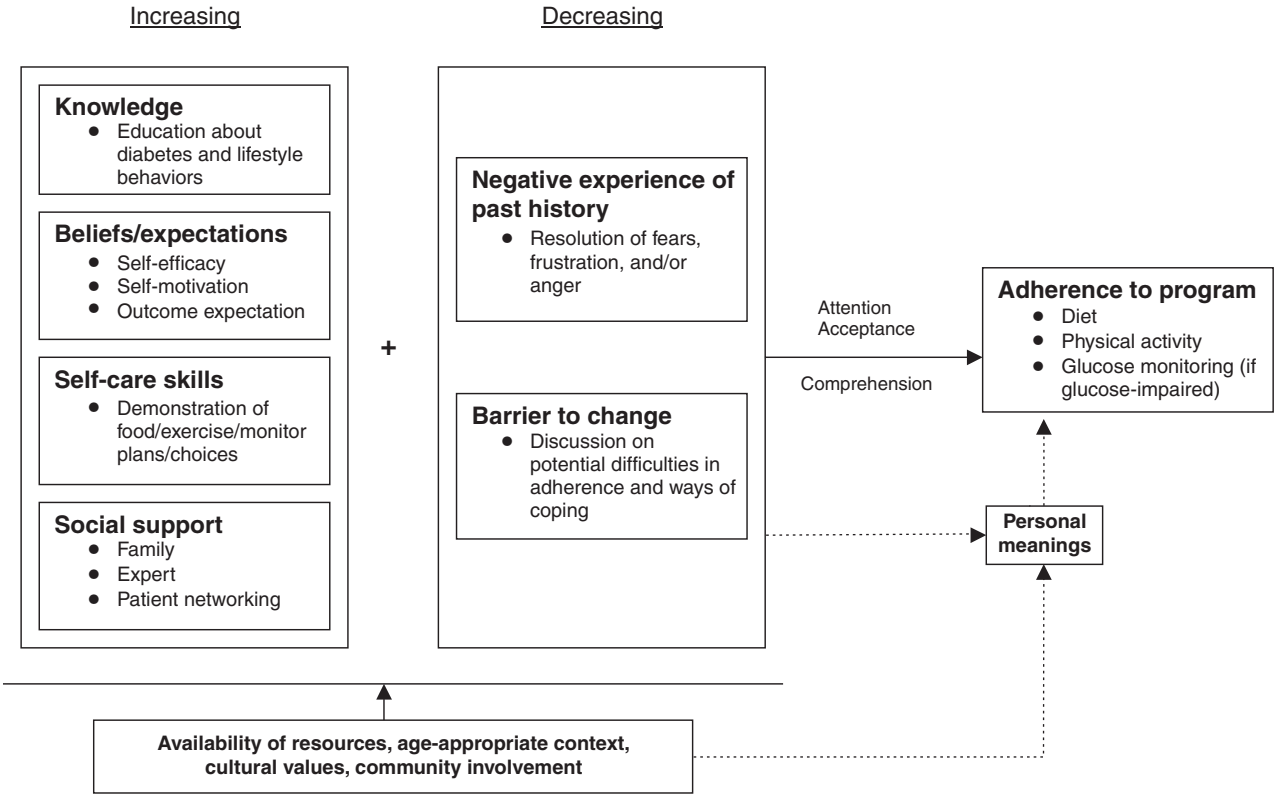

Fig. 3. A theoretical model for prevention of type 2 diabetes in young people. Dashed lines represent alternative paths.

social cognitive theory (163), protection motivation theory (187), social influence theory (188), the Precede-Proceed model (171), sociocultural theories (188–190), and community organization theory (191). It emphasizes an increase in knowledge, realistic beliefs/expectations, self-care skills, and social support with regard to diabetes, while attempting to help individuals overcome potential obstacles in making behavioral changes. Availability of resources (including family involvement), an age-appropriate and culturally sensitive context, and involvement of community organizations serve as underlying moderating constructs that accompany all aspects of an intervention; this approach makes the current model particularly unique. As such, a comprehensive structured intervention can help increase internal mediating processes such as the attention, comprehension, and acceptance of regimen demands (192), therefore leading to better clinical outcomes.

Social cognitive variables

Knowledge

Education about diabetes and its related complications is essential in helping families of young people at risk to understand what diabetes is, why their children are at risk, and the severity of diabetes and its complications. Knowledge has been shown to be related to diabetic self-care behaviors in adults (193). In addition, at least one study attributes poor adherence in African-American youths with type 2 diabetes to lack of understanding about the disease (194). Young people at risk, due to their age, may not fully understand the ramifications of their medical condition; however, they may be informed of the importance of taking care of themselves in order to stay healthy. Young people need to be taught how to analyze their behavior choices in the context of their health (5). Parents, on the other hand, must be given more detailed information with regard to their child's risk for type 2 diabetes. Parents can play an important role in ensuring that their children are living a healthy lifestyle.

Beliefs/expectations

Self-efficacy, self-motivation, and accurate outcome expectations form the core of this construct. Self-efficacy has been shown to be important in inducing behavioral changes (163, 195) and has been shown to be related to diabetes-specific health behaviors (196). Self-motivation is related to adherence and other health-related behaviors (197). In addition, outcome expectations have been shown to be associated with diabetes-specific self-care behaviors such as diet and exercise (196, 198). Outcome expectations or beliefs about the effectiveness of the intervention are important because they serve as positive feedback for someone to be motivated to continue to engage in healthy

behaviors. Young people need to be empowered, i.e., given a sense of control over their own health, so that they do not feel there is nothing they can do to keep themselves healthy. Parents, on the other hand, require self-efficacy and self-motivation in helping their children to stay healthy. Both young people and their parents require positive and realistic outcome expectations.

Self-care skills

This construct refers to teaching young people at risk and their families how to build physical activity and healthy diets into their existing lives. It has been suggested that peer pressure to consume high-caloric foods and beverages high in sugar content may contribute to non-adherence in the young (5). The ability to take care of oneself in the context of one's health is crucial in behavioral adherence. Therefore, demonstrations of exercise plans and food choices should form part of an intervention. For glucose-impaired young people, the important steps in glucose monitoring need to be taught to families. Self-care skills also include general problem-solving skills that may help young people overcome the social pressure to engage in unhealthy behaviors that often take place in peer groups.

Social support

Social support plays an important role in health (199). The benefit of social support on physical health has been demonstrated in the care of other chronic illnesses (200). A supportive environment is particularly useful in encouraging behavioral adherence, and should include both familial support and expert support. The latter entails both informational support and emotional support. The needs and concerns of young people and their families need to be understood and empathy shown. Establishing an open channel of communication and rapport can promote a sense of cooperation between the researchers/clinicians and the target families, hence ensuring better adherence. In addition to the two forms of support mentioned above, patient networking may also be important. It is important for families to know that there are other families out there who experience similar issues. This may be especially helpful for single parent families. Patient networking not only provides encouragement for young people and their families to maintain behavior changes, but also serves as a monitoring process, whereby they can model after others with regard to healthy behavior practices.

Negative experience of past history

This aspect of the proposed model aims to reduce the impact of fears, frustration, and/or anger that stem from past experiences (201). These experiences can include negative interactions with health care professionals, social pressures from communities or peer groups, and/or a false sense of uniqueness. The idea is to help young people and their families gain confidence in themselves and in an intervention by eliminating psychological obstacles accumulated from the past.

Barriers to change

This construct focuses on the costs of adherent response. Barriers to change have been shown to have significant associations with non-adherence (198, 202). This element of the intervention aims to discuss, *a priori*, potential difficulties in adherence, including the degree of physical (e.g. pain), intrapersonal (e.g. dislike, inconvenience), and interpersonal (e.g. embarrassment, interference with social activities) discomfort that may be caused by the program prescription, and how to come up with creative ways to overcome these difficulties. Emphasis should be given to the appraisal of adherence rather than the risks of non-adherence, as the former may be more influential in promoting actual adherence (202).

Underlying moderating constructs

Figure 4 illustrates the theoretical determinants of the four moderators in the proposed intervention model: availability of resources, cultural values, age appropriateness, and community involvement. An interaction effect between these moderators and social cognitive or motivational constructs may be mediated by personal meanings of health behavior (203). In the current model, meanings of behavior are indicated by affective reinforcement for healthy behavioral change and selective preference for certain behaviors. Meanings of behavior are purely personal and affective, and often in opposition to knowledge and beliefs. In contrast to conventional values-expectancy theories, the theory of meanings of behavior takes developmental issues adequately into account (204–207). Previous research has demonstrated that the meanings that adolescents attach to health-related behaviors are highly predictive of those behaviors (204, 208).

In addition to the proposed interaction effects, availability of resources, cultural values, age-appropriateness, and community involvement may also exert significant main effects. The theory and intervention application behind each moderator is further discussed below.

Availability of resources

The availability of resources in the context of prevention of type 2 diabetes in young people includes three parameters: family involvement, cost, and access. The availability of resources can be conceptualized as reference to the enabling factors from the Precede-Proceed

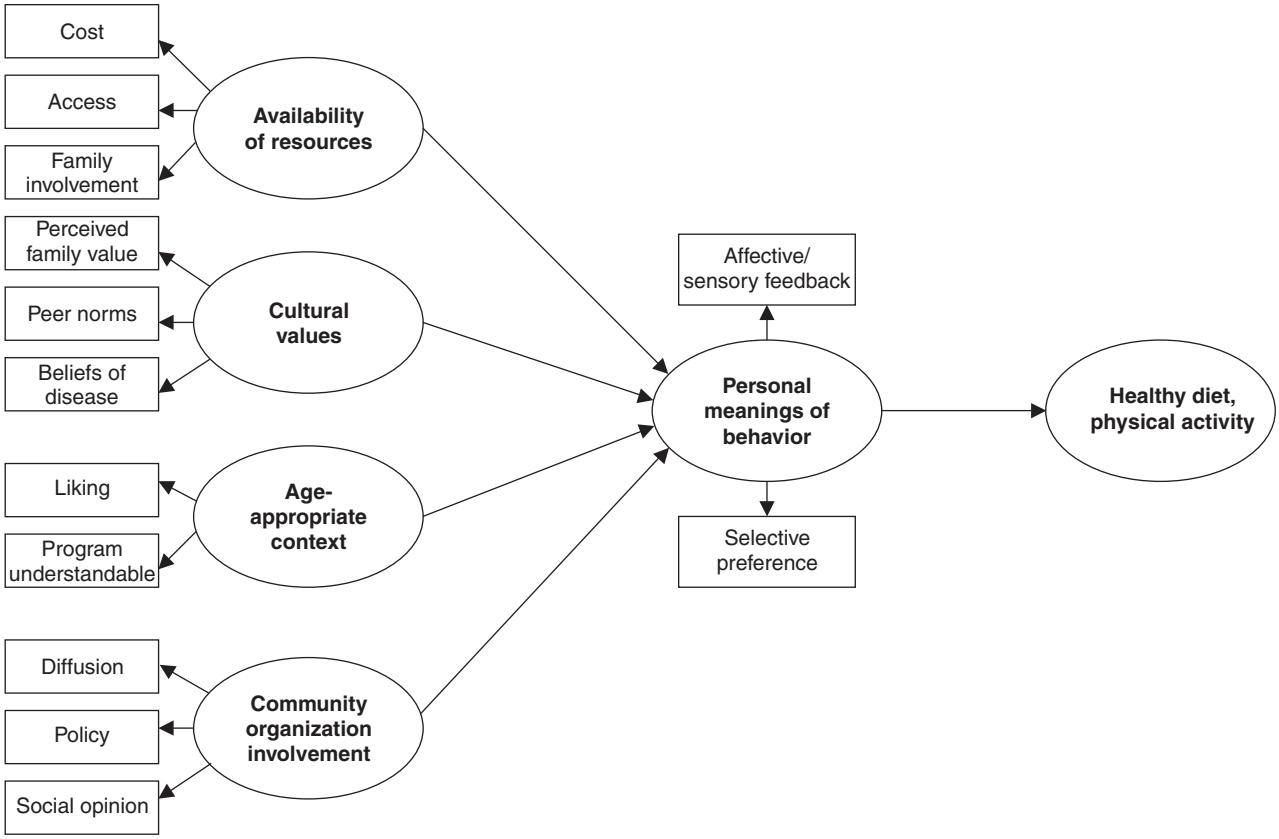

Fig. 4. Factors associated with proposed intervention moderators.

model (171). Family involvement is characterized by the family-based nature of the current model. Parents have to take on the responsibility to encourage their child to practice healthy behaviors, to monitor such behaviors, and to model such behaviors themselves. Lifestyle changes are most successful if the whole family is mobilized to engage in these changes. Long-term adherence requires that a new lifestyle habit be shaped early on in a child's life. The immediate environment of the child is most important in shaping the new habit. The relative cost as perceived by young people and their families also directly influences the availability of healthy foods and the opportunity to be physically active. Cost can be financial, temporal, and/or social. Finally, the accessibility of healthy foods and physical activity must be addressed. Access may be limited due to geography, culture, or environmental safety. Thus, interventions that help identify regular opportunities to be physically active and to access fresh and healthy food choices are much needed.

Age-appropriate context

In order for an intervention to be successful, it must be tailored to the liking of the age group being addressed. Young people are much more likely to be self-motivated to practice healthy behaviors when they like what they are doing. It has been shown that adolescents with type 1 diabetes engaged in self-motivated exercise training at home upon receiving an intervention that capitalized on the popularity of aerobic exercises and popular music (209). The language and techniques used must be understandable and acceptable for a young audience. Developmental stage influences level of comprehension in children and adolescents (210). Special attention should thus be paid to developmental issues of communication, cognitive, and behavioral strategies.

Cultural values

Given that type 2 diabetes is most prevalent in minority populations, cultural sensitivity would appear to be most important when designing a prevention intervention for such populations. Cultural sensitivity refers to an understanding of the different health belief systems and varying socioeconomic status. Cultural values can be assessed by perceived family values, normative beliefs and practices among friends, and cultural beliefs of disease. For instance, one study of Indian young people with diabetic family members reported that, although subjects had some knowledge about the disease, they did not relate complications, such as retinopathy or amputation, to diabetes. Furthermore, over half of the study population expressed the belief that diabetes was contagious or was caused by 'bad blood', and over a third of the group attributed the disease to

'weakness' (211). Indeed, cultures vary greatly in their beliefs about illnesses and such beliefs may significantly impact behavioral practices. In addition, when dealing with different cultures, there may be a lack of familiarity with recommended food items, which may be costly or difficult for families to obtain, and require special preparation (185, 212). Given the different needs of minority populations, it is necessary, then, to develop culturally relevant prevention intervention programs, in order to obtain positive outcomes.

Community involvement

Based on the community organizational theory (191), community involvement enables people to become self-reliant in addressing lifestyles related to health. Empowerment, at the core of the community organization approach, is defined as access to, control of, and ownership of valued resources and the process of behavioral change (213, 214). Empowerment at the community-level helps to build a strong social norm of change and systems support for change in the individual. In the practical setting, community involvement can be indicated by the level of diffusion of health education, policy setting, and health-related social opinions. In many of the type 2 diabetes prevention efforts for Mexican-Americans or Native Americans, community involvement refers to both the geographic community and the diabetes-affected community because of the relatively small size of these communities and the relatively large proportion of affected individuals. In more heterogeneous, urban settings, involvement of the entire geographic community may not be feasible or cost-effective; therefore, diabetes-affected communities may serve more appropriately as the unit of involvement.

Model implementation

Given that community involvement is indicated as an essential moderator in the proposed model, the community would be the most ideal setting for implementation. However, community is defined broadly. For instance, a school or school district could be defined as a community on its own. The idea is that within a defined community, the mobilization and partnership of all sectors, such as the board of education, school administration, teachers, families, and students, should be optimized.

The basic framework of the proposed model could be potentially diffused to non-US settings. All the moderating variables are conceptualized with the flexibility of different circumstances in different cultures and/or countries. Therefore, we do not suggest a universal model for all populations but rather creating programs that are specific to the needs, resources, and limitations of each target population using the proposed general framework as a guideline. The proposed model should be put through rigorous empiric testing in the future, to determine and refine the most effective elements. Cost of an intervention program based on the proposed model is difficult to estimate and would require empiric testing and analysis as well.

Conclusions

The rapidly emerging health problem of type 2 diabetes in minority populations indicates a strong need for prevention interventions designed to address the issues of minority cultures. To date, few empiric studies have been conducted to prevent the onset of type 2 diabetes or its related complications in young people. However, based on the results from dietary and physical activity studies in adults, and obesity interventions in young people, prevention interventions of type 2 diabetes may also merit from programs that address lifestyle-related changes in young people. More importantly, though, the design of prevention interventions of type 2 diabetes should be based on sound theoretical reasoning. We propose a model that is theoretically diverse, incorporating elements of several social, cognitive, behavioral, and environmental theories. The model aims to intervene on variables such as diabetes knowledge; self-efficacy, self-motivation, and outcome expectation of behavioral change; mastery of diabetes self-care skills; familial and expert support; and reducing the impact of negative past experiences and overcoming potential barriers to change. Because high adherence to intervention recommendations is typically hard to achieve, particularly in young people, moderating factors such as the availability of resources, cultural values, age-appropriateness, and community involvement, are considered in the proposed model to ensure sustained, long-term health benefits.

Acknowledgements

We thank Drs Mary Ann Pentz, Donna Spruijt-Metz, and Geoff Ball for their insightful comments.

References

1. Glaser NS. Non-insulin-dependent diabetes mellitus in childhood and adolescence. Pediatr Clin North Am 1997: 44: 307–337.
2. Dean HJ, Mundy RL, Moffatt M. Non-insulin-dependent diabetes mellitus in Indian children in Manitoba. CMAJ 1992: 147: 52–57.
3. Pinhas-Hamiel O, Dolan LM, Daniels SR et al. Increased incidence of non-insulin-dependent diabetes mellitus among adolescents. J Pediatr 1996: 128: 608–615.
4. American Diabetes Association. Diabetes 2001. Vital Statistics. American Diabetes Association, Alexandria, VA, 2001.
5. Rosenbloom AL, Joe JR, Young RS, Winter WE. Emerging epidemic of type 2 diabetes in youth. Diabetes Care 1999: 22: 345–354.

6. Strauss RS, Pollack HA. Epidemic increase in childhood overweight, 1986–98. JAMA 2001: 286: 2845–2848.
7. Anonymous. Type 2 diabetes in children and adolescents. American Diabetes Association Pediatrics 2000: 105: 671–680.
8. Zimmet P, Alberti KGMM, Shaw J. Global and societal implications of the diabetes epidemic. Nature 2001: 414: 782–787.
9. Knowler WC, Pettitt DJ, Savage PJ, Bennett PH. Diabetes incidence in Pima indians: contributions of obesity and parental diabetes. Am J Epidemiol 1981: 113: 144–156.
10. Kitagawa T, Owada M, Urakami T, Tajima N. Epidemiology of type 1 (insulin-dependent) and type 2 (non-insulin-dependent) diabetes mellitus in Japanese children. Diabetes Res Clin Prac 1994: 24 (Suppl.): S7–S13.
11. Kitagawa T, Owada M, Urakami T, Yamauchi K. Increased incidence of non-insulin dependent diabetes mellitus among Japanese schoolchildren correlates with an increased intake of animal protein and fat. Clin Pediatr 1998: 37: 111–115.
12. Kadiki OA, Reddy MR, Marzouk AA. Incidence of insulin-dependent diabetes (IDDM) and non-insulin-dependent diabetes (NIDDM) (0–34 years at onset) in Benghazi, Libya. Diabetes Res Clin Prac 1996: 32: 165–173.
13. Gower BA, Nagy TR, Goran MI. Visceral fat, insulin sensitivity, and lipids in prepubertal children. Diabetes 1999: 48: 1515–1521.
14. Hale DE, Danney MM. Non-insulin dependent diabetes in Hispanic youth (Type 2Y). Diabetes 1998: 47: A82.
15. Sinha R, Fisch G, Teague B et al. Prevalence of impaired glucose tolerance among children and adolescents with marked obesity. N Engl J Med 2002: 346: 802–810.
16. Defronzo RA. Pathogenesis of type 2 (non-insulin dependent) diabetes mellitus: a balanced overview. Diabetologia 1992: 35: 389–397.
17. Hales CN. The pathogenesis of NIDDM. Diabetologia 1994: 37(Suppl. 2): S162–168.
18. Turner R, Hattersley A, Cook J. Type II diabetes: search for primary defects. Ann Med 1992: 24: 511–516.
19. Henry RR. Glucose control and insulin resistance in non-insulin-dependent diabetes mellitus. Ann Intern Med 1996: 124: 97–103.
20. Genuth S. Insulin use in NIDDM. Diabetes Care 1990: 13: 1240–1264.
21. Moran A, Jacobs DR Jr, Steinberger J et al. Insulin resistance during puberty: results from clamp studies in 357 children. Diabetes 1999: 48: 2039–2044.
22. Goran MI, Gower BA. Longitudinal study on pubertal insulin resistance. Diabetes 2001: 50: 2444–2450.
23. Amiel SA, Sherwin RS, Simonson DC, Lauritano AA, Tamborlane WV. Impaired insulin action in puberty: a contributing factor to poor glycemic control in adolescents with diabetes. N Engl J Med 1986: 315: 215–219.
24. Caprio S, Tamborlane WV. Effect of puberty on insulin action and secretion. Semin Reprod Endocrinol 1994: 12: 90–96.
25. Caprio S, Cline G, Boulware S et al. Effects of puberty and diabetes on metabolism of insulin-sensitive fuels. Am J Physiol 1994: 266: E885–E891.
26. Travers SH, Jeffers BW, Bloch CA, Hill JO, Eckel RH. Gender and Tanner stage differences in body composition and insulin sensitivity in early pubertal children. J Clin Endocrinol Metab 1995: 80: 172–178.
27. Knochenhauer ES, Key TJ, Kahsar-Miller M et al. Prevalence of the polycystic ovary syndrome in unselected black and white women of the southeastern United States. A Prospective Study J Clin Endocrinol Metab 1998: 83: 3078–3082.
28. Ibanez L, Potau N, Carrascosa A. Insulin resistance, premature adrenarche, and a risk of the polycystic ovary syndrome (PCOS). Trends Endocrinol Metabolism 1998: 9: 72–77.
29. Ibanez L, Potau N, Zampolli M et al. Hyperinsulinemia in postpubertal girls with a history of premature pubarche and functional ovarian hyperandrogenism. J Clin Endocrinol Metab 1996: 81: 1237–1243.
30. Apter D. Endocrine and metabolic abnormalities in adolescents with a PCOS-like condition: consequences for adult reproduction. Trends Endocrinol Metab 1998: 9: 58–61.
31. Lewy VD, Danadian K, Witchel SF et al. Early metabolic abnormalities in adolescent girls with polycystic ovarian syndrome. J Pediatr 2001: 138: 38–44.
32. Schonfeld-Warden N, Warden CH. Pediatric obesity. An overview of etiology and treatment. Pediatr Clin North Am 1997: 44: 339–361.
33. Beck-Nielsen H, Hother-Nielsen O. Obesity in non-insulin-dependent diabetes mellitus. In: Leroith D, Taylor SI, Olefsky JM, eds. Diabetes Mellitus. Lippincott-Raven Publishers, Philadelphia, 1996, pp 475–484.
34. Barrett-Connor E. Epidemiology, obesity, and non-insulin-dependent diabetes mellitus. Epidemiol Rev 1989: 11: 172–181.
35. Caro JF, Sinha MK, Raju SM et al. Insulin receptor kinase in human skeletal muscle from obese subjects with and without noninsulin dependent diabetes. J Clin Invest 1987: 79: 1330–1337.
36. Kolterman OG, Insel J, Saekow M, Olefsky JM. Mechanisms of insulin resistance in human obesity: evidence for receptor and postreceptor defects. J Clin Invest 1980: 65: 1272–1284.
37. Lillioja S, Young AA, Culter CL et al. Skeletal muscle capillary density and fiber type are possible determinants of in vivo insulin resistance in man. J Clin Invest 1987: 80: 415–424.
38. Gortmaker SL, Dietz WH Jr, Sobol AM, Wehler CA. Increasing pediatric obesity in the United States. Am J Dis Child 1987: 141: 535–540.
39. Troiano RP, Flegal KM, Kuczmarski RJ, Campbell SM, Johnson CL. Overweight prevalence and trends for children and adolescents. The National Health and Nutrition Examination Surveys, 1963–91. Arch Pediatr Adolesc Med 1995: 149: 1085–1091.
40. Fall CH. Mrc Environmental Epidemiology Unit UoSUK. Non-industrialised countries and affluence. Br Med Bull 2001, 60.
41. Uauy R, Albala C, Kain J. Obesity trends in Latin America: transiting from under- to overweight. J Nutr 2001: 131: 893S–899S.
42. Seidell JC. Obesity, insulin resistance and diabetes – a worldwide epidemic. Br J Nutr 2000: 83(Suppl. 1): S5–8.
43. Kain J, Uauy R, Vio F, Albala C. Trends in overweight and obesity prevalence in Chilean children: comparison of three definitions. Eur J Clin Nutr 2002: 56: 200–204.
44. UNICEF. The State of the World's Children 2002. United Nations Publications, New York, 2002.
45. Olivarius ND, Andreasen AH. Weight history and its impact on risk factors and dibetic complications of newly diagnosed diabetic patients 40 years of age and over. 15th IDF Congress, Kobe, Japan. International Diabetes Federation, Brussels, Belgium, 1994.
46. Bennett PH, Knowler WC, Rushforth NB. The Role of Obesity in the Development of Diabetes in the Pima Indians. In: Vague J, Vague PH, eds. Diabetes and Obesity. Exerta Medica, Oxford, 1979, p 117.

47. Rewers M, Hamman RF. Risk Factors for Non-insulin-Dependent Diabetes. National Diabetes Data Group. Diabetes in America. National Institute of Diabetes and Digestive and Kidney Diseases, Bethesda, MD, 1995, pp 179–220.
48. Modan M, Karasik A, Halkin H et al. Effect of past and concurrent body mass index on prevalence of glucose intolerance and type 2 (non-insulin-dependent) diabetes and on insulin response. The Israel study of glucose intolerance, obesity and hypertension. Diabetologia 1986: 29: 82–89.
49. Everhart JE, Pettitt DJ, Bennett PH, Knowler WC. Duration of obesity increases the incidence of NIDDM. Diabetes 1992: 41: 235–240.
50. Bennett PH, Bogardus C, Tuomilehto J. Epidemiology and natural history of NIDDM. non-obese and obese. In: Alberti KG, Defronzo RA, Keen H, eds. International Textbook of Diabetes. John Wiley, Chichester, 1992, pp 148–176.
51. Bjorntorp P. Abdominal obesity and the development of noninsulin-dependent diabetes mellitus. Diab Metab Rev 1988: 4: 615–622.
52. Cowie CC, Harris MI, Silverman RE, Johnson EW, Rust KF. Effect of multiple risk factors on differences between blacks and whites in the prevalence of non-insulin-dependent diabetes mellitus in the United States. Am J Epidemiol 1993: 137: 719–732.
53. Hartz AJ, Rupley DC, Rimm AA. The association of girth measurements with disease in 32,856 women. Am J Epidemiol 1984: 119: 71–80.
54. van Noord PA, Seidell JC, den Tonkelaar I, Baanders-van Halewijn EA, Ouwehand IJ. The relationship between fat distribution and some chronic diseases in 11,825 women participating in the DOM-project. Int J Epidemiol 1990: 19: 564–570.
55. Haffner SM, Stern MP, Mitchell BD, Hazuda HP, Patterson JK. Incidence of type II diabetes in Mexican Americans predicted by fasting insulin and glucose levels, obesity, and body-fat distribution. Diabetes 1990: 39: 283–288.
56. Ohlson LO, Larsson B, Svardsudd K et al. The influence of body fat distribution on the incidence of diabetes mellitus: 13.5 years of follow-up of the participants in the study of men born in 1913. Diabetes 1985: 34: 1055–1058.
57. Seidell JC, Deurenberg P, Hautvast JG. Obesity and fat distribution in relation to health – current insights and recommendations. World Rev Nutr Dietet 1987: 50: 57–91.
58. Carey DG, Jenkins AB, Campbell LV, Freund J, Chisholm DJ. Abdominal fat and insulin resistance in normal and overweight women. Direct measurements reveal a strong relationship in subjects at both low and high risk of NIDDM. Diabetes 1996: 45: 633–638.
59. Huang TT, Johnson MS, Gower BA, Goran MI. Effect of changes in fat distribution on the rates of change of insulin response in children. Obesity Res 2002: 10: 978–984.
60. Ahmed ML, Ong KK, Morrell DJ et al. Longitudinal study of leptin concentrations during puberty: sex differences and relationship to changes in body composition. J Clin Endocrinol Metab 1999: 84: 899–905.
61. Byrnes SE, Baur LA, Bermingham M, Brock K, Steinbeck K. Leptin and total cholesterol are predictors of weight gain in pre-pubertal children. Int J Obes Relat Metab Disord 1999: 23: 146–150.
62. Johnson MS, Huang TT, Figueroa-Colon R, Dwyer JH, Goran MI. Influence of leptin on changes in body fat during growth in African American and white children. Obes Res 2001: 9: 593–598.
63. Yang WS, Lee WJ, Funahashi T et al. Weight reduction increases plasma levels of an adipose-derived anti-inflammatory protein, adiponectin. J Clin Endocrinol Metab 2001: 86: 3815–3819.
64. Weyer C, Funahashi T, Tanaka S et al. Hypoadiponectinemia in obesity and type 2 diabetes: close association with insulin resistance and hyperinsulinemia. J Clin Endocrinol Metab 2001: 86: 1930–1935.
65. Hotta K, Funahashi T, Arita Y et al. Plasma concentrations of a novel, adipose-specific protein, adiponectin, in type 2 diabetic patients. Arterioscler Thromb Vasc Biol 2000: 20: 1595–1599.
66. Arita Y, Kihara S, Ouchi N et al. Paradoxical decrease of an adipose-specific protein, adiponectin, in obesity. Biochem Biophys Res Commun 1999: 257: 79–83.
67. Stefan N, Bunt JC, Salbe AD et al. Plasma adiponectin concentrations in children: relationships with obesity and insulinemia. J Clin Endocrinol Metab 2002: 87: 4652–4656.
68. Steppan CM, Bailey ST, Bhat S et al. The hormone resistin links obesity to diabetes. Nature 2001: 409: 307–312.
69. Nagaev I, Smith U. Insulin resistance and type 2 diabetes are not related to resistin expression in human fat cells or skeletal muscle. Biochem Biophys Res Commun 2001: 285: 561–564.
70. Sentinelli F, Romeo S, Arca M et al. Human resistin gene, obesity, and type 2 diabetes: mutation analysis and population study. Diabetes 2002: 51: 860–862.
71. Barker DJ.The fetal origins of adult hypertension. J Hypertens 1992: 10: S39–44.
72. Hales CN, Barker DJ. Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. Diabetologia 1992: 35: 595–601.
73. Hales CN. The role of poor fetal and infant growth in non-insulin-dependent diabetes. Bull Mem Acad R Med Belg 1993: 148: 281–285.
74. Leon DA. Fetal growth and adult disease. Eur J Clin Nutr 1998: 52: S72–S78.
75. Hales CN, Barker DJ., The thrifty phenotype hypothesis. Br Med Bull 2001: 60: 5–20.
76. Pettitt DJ, Bennett PH, Saad MF et al. Abnormal glucose tolerance during pregnancy in Pima Indian women. Long-term effects on offspring. Diabetes 1991: 40(Suppl. 2): 126–130.
77. Pettitt DJ, Nelson RG, Saad MF, Bennett PH, Knowler WC. Diabetes and obesity in the offspring of Pima Indian women with diabetes during pregnancy. Diabetes Care 1993: 16: 310–314.
78. Silverman BL, Rizzo TA, Cho NH, Metzger BE. Long-term effects of the intrauterine environment. The Northwestern University Diabetes Pregnancy Center. Diabetes Care 1998: 21(Suppl. 2): B142–149.
79. Buchanan TA, Kjos SL. Gestational diabetes: risk or myth? J Clin Endocrinol Metab 1999: 84: 1854–1857.
80. Pan XR, Li GW, Hu YH et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT Diabetes Study. Diabetes Care 1997: 20: 537–544.
81. Tuomilehto J, Lindstrom J, Eriksson JG et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. N Engl J Med 2001: 344: 1343–1350.
82. Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med 2002: 346: 393–403.
83. Westlund K. Incidence of diabetes mellitus in Oslo, Norway, 1925–54. Br J Prev Soc Med 1966: 20: 105.
84. Mann JI, Houston A. The aetiology of non-insulin-dependent diabetes mellitus. In: Mann JI, Pyorala K,

Teuscher A, eds. Diabetes. Churchill Livingstone, Edinbugh, 1983, pp 122–164.
85. Himsworth HP. Diet and the incidence of diabetes mellitus. Clin Sci 1935: 2: 117–148.
86. Trevino RP, Marshall RM Jr, Hale DE et al. Diabetes risk factors in low-income Mexican-American children. Diabetes Care 1999: 22: 202–207.
87. French SA, Story M, Neumark-Sztainer D et al. Fast food restaurant use among adolescents: associations with nutrient intake, food choices and behavioral and psychosocial variables. Int J Obes Relat Metab Disord 2001: 251823–1833.
88. Troiano RP, Briefel RR, Carroll MD et al. Energy and fat intakes of children and adolescents in the united states: data from the national health and nutrition examination surveys. Am J Clin Nutr 2000: 72(Suppl.): 1343S–1353S.
89. Jiménez-Cruz A, Bacardâi-Gascâon M, Jones EG. Consumption of fruits, vegetables, soft drinks, and high-fat-containing snacks among Mexican children on the Mexico-U.S. border. Arch Med Res 2002: 33: 74–80.
90. Harnack L, Stang J, Story M. Soft drink consumption among US children and adolescents: nutritional consequences. J Am Diet Assoc 1999: 99: 436–441.
91. Lytle LA, Seifert S, Greenstein J et al. How do children's eating patterns and food choices change over time? Results from a cohort study. Am J Health Promot 2000: 14: 222–228.
92. Bandini LG, Vu D, Must A et al. Comparison of high-calorie, low-nutrient-dense food consumption among obese and non-obese adolescents. Obes Res 1999: 7: 438–443.
93. Janssens JP, Shapira N, Debeuf P et al. Effects of soft drink and table beer consumption on insulin response in normal teenagers and carbohydrate drink in youngsters. Eur J Cancer Prev 1999: 8: 289–295.
94. Sunehag AL, Toffolo G, Treuth MS et al. Effects of dietary macronutrient content on glucose metabolism in children. J Clin Endocrinol Metabol 2002: 87: 5168–5178.
95. Ku CY, Gower BA, Nagy TR, Goran MI. Relationships between dietary fat, body fat, and serum lipid profile in prepubertal children. Obes Res 1998: 6: 400–407.
96. Lindquist CH, Gower BA, Goran MI. Role of dietary factors in ethnic differences in early risk of cardiovascular disease and type 2 diabetes. Am J Clin Nutr 2000: 71: 725–732.
97. Stern MP, Gonzalez C, Mitchell BD et al. Genetic and environmental determinants of type II diabetes in Mexico City and San Antonio. Diabetes 1992: 41: 484–492.
98. Zimmet P, Seluka A, Collins J et al. Diabetes mellitus in an urbanized, isolated Polynesian population. The Funafuti survey. Diabetes 1977: 26: 1101–1108.
99. Zimmet P, Faaiuso S, Ainuu J et al. The prevalence of diabetes in the rural and urban Polynesian population of Western Samoa. Diabetes 1981: 30: 45–51.
100. Wicking J, Ringrose H, Whitehouse S, Zimmet P. Nutrient intake in a partly westernized isolated Polynesian population: the Funafuti survey. Diabetes Care 1981: 4: 92–95.
101. Taylor R, Zimmet P. The influence of variation in obesity in the sex difference in the prevalence of abnormal glucose tolerance in Tuvalu. NZ Med J 1981: 94: 176–178.
102. Taylor RJ, Bennett PH, Legonidec G et al. The prevalence of diabetes mellitus in a traditional-living Polynesian population: the Wallis Island survey. Diabetes Care 1983: 6: 334–340.
103. Taylor R, Ram P, Zimmet P, Raper LR, Ringrose H. Physical activity and prevalence of diabetes in Melanesian and Indian men in Fiji. Diabetologia 1984: 27: 578–582.
104. Uusitupa M, Siitonen O, Pyorala K et al. The relationship of cardiovascular risk factors to the prevalence of coronary heart disease in newly diagnosed type 2 (non-insulin-dependent) diabetes. Diabetologia 1985: 28: 653–659.
105. King H, Zimmet P, Raper LR, Balkau B. Risk factors for diabetes in three Pacific populations. Am J Epidemiol 1984: 119: 396–409.
106. Chen MK, Lowenstein FW. Epidemiology of factors related to self-reported diabetes among adults. Am J Prev Med 1986: 2: 14–19.
107. Frisch RE, Wyshak G, Albright TE, Albright NL, Schiff I. Lower prevalence of diabetes in female former college athletes compared with nonathletes. Diabetes 1986: 35: 1101–1105.
108. Holbrook TL, Barrett-Connor E, Wingard DL. The association of lifetime weight and weight control patterns with diabetes among men and women in an adult community. Int J Obes 1989: 13: 723–729.
109. Cederholm J, Wibell L. Impaired glucose tolerance: influence by environmental and hereditary factors. Diabete Metab 1991: 17: 295–299.
110. Leonetti DL, Fujimoto WY, Wahl PW. Early-life background and the development of non-insulin-dependent diabetes mellitus. Am J Phys Anthropol 1989: 79: 345–355.
111. Dowse GK, Zimmet PZ, Gareeboo H et al. Abdominal obesity and physical inactivity as risk factors for NIDDM and impaired glucose tolerance in Indian, Creole, and Chinese Mauritians. Diabetes Care 1991: 14: 271–282.
112. Ramaiya KL, Swai AB, McLarty DG, Alberti KG. Impaired glucose tolerance and diabetes mellitus in Hindu Indian immigrants in Dar es Salaam. Diabet Med 1991: 8: 738–744.
113. Manson JE, Rimm EB, Stampfer MJ et al. Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. Lancet 1991: 338: 774–778.
114. Harris MI. Epidemiological correlates of NIDDM in Hispanics, whites, and blacks in the US population. Diabetes Care 1991: 14: 639–648.
115. Helmrich SP, Ragland DR, Leung RW, Paffenbarger RS Jr. Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. N Engl J Med 1991: 325: 147–152.
116. Manson JE, Nathan DM, Krolewski AS et al. A prospective study of exercise and incidence of diabetes among US male physicians. JAMA 1992: 268: 63–67.
117. Bouchard C, Tremblay A, Nadeau A et al. Long-term exercise training with constant energy intake. 1: Effect on body composition and selected metabolic variables [published erratum appears in Int J Obes 1990; 14: 987]. Int J Obes 1990: 14: 57–73.
118. Lampman RM, Schteingart DE. Effects of exercise training on glucose control, lipid metabolism, and insulin sensitivity in hypertriglyceridemia and non-insulin dependent diabetes mellitus. Med Sci Sports Exerc 1991: 23: 703–712.
119. Krotkiewski M, Lonnroth P, Mandroukas K 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 1985: 28: 881–890.

120. Trovati M, Carta Q, Cavalot F 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 1984: 7: 416–420.
121. Anonymous. Physical Activity and Health: A Report of the Surgeon General. US Department of Health and Human Services, Rockville, MD, 1996.
122. Ruderman NB, Schneider SH. Diabetes, exercise, and atherosclerosis. Diabetes Care 1992: 15: 1787–1793.
123. Gudat U, Berger M, Lefebvre PJ. Phsyical activity, fitness, and non-insulin-dependent diabetes mellitus. In: Bouchard C, Shephard RJ, Stephens T, eds. Physical Activity, Fitness, and Health. International,. Proceedings and Consensus Statement. Human Kinetics, Champaign, IL, 1994 pp 669–683.
124. Ferguson MA, Gutin B, Le NA, et al. Effects of exercise training and its cessation on components of the insulin resistance syndrome in obese children. Int J Obes Relat Metab Disord 1999: 23: 889–895.
125. Treuth MS, Hunter GR, Figueroa-Colon R, Goran MI. Effects of strength training on intra-abdominal adipose tissue in obese prepubertal girls. Med Sci Sports Exerc 1998: 30: 1738–1743.
126. Goran MI, Reynolds KD, Lindquist CH. Role of physical activity in the prevention of obesity in children. Int J Obes Relat Metab Disord 1999: 23(Suppl. 3): S18–S33.
127. Gordon-Larsen P, McMurray RG, Popkin BM. Adolescent physical activity and inactivity vary by ethnicity. The National Longitudinal Study of Adolescent Health. J Pediatr 1999: 135: 301–306.
128. Robinson TN. Television viewing and childhood obesity. Pediatr Clin North Am 2001: 48: 1017–1025.
129. Gortmaker SL, Must A, Sobol AM et al. Television viewing as a cause of increasing obesity among children in the United States, 1986–90. Arch Pediatr Adolesc Med 1996: 150: 356–362.
130. Epstein LH, Smith JA, Vara LS, Rodefer JS. Behavioral economic analysis of activity choice in obese children. Health Psychol 1991: 10: 311–316.
131. Epstein LH, Valoski AM, Vara LS et al. Effects of decreasing sedentary behavior and increasing activity on weight change in obese children. Health Psychol 1995: 14: 109–115.
132. Johnson MS, Figueroa-Colon R, Herd SL et al. Aerobic fitness, not energy expenditure, influences subsequent increase in adiposity in black and white children. Pediatrics 2000: 106: E50.
133. Dietz WH, Bandini LG, Morelli JA et al. Effect of sedentary activities on resting metabolic rate. Am J Clin Nutrition 1994: 59: 556–559.
134. Robinson TN. Reducing children's television viewing to prevent obesity: a randomized controlled trial. JAMA 1999: 282: 1561–1567.
135. Von Kries R, Toschke AM, Koletzko B, Slikker W Jr. Maternal smoking during pregnancy and childhood obesity. Am J Epidemiol 2002: 156: 954–961.
136. Toschke AM, Koletzko B, Slikker W Jr, Hermann M, von Kries R. Childhood obesity is associated with maternal smoking in pregnancy. Eur J Pediatr 2002: 161: 445–448.
137. Power C, Jefferis BJ. Fetal environment and subsequent obesity: a study of maternal smoking. Int J Epidemiol 2002: 31: 413–419.
138. Vik T, Jacobsen G, Vatten L, Bakketeig LS. Pre- and post-natal growth in children of women who smoked in pregnancy. Early Hum Dev 1996: 45: 245–255.
139. Montgomery SM, Ekbom A. Smoking during pregnancy and diabetes mellitus in a British longitudinal birth cohort. BMJ 2002: 324: 26–27.
140. von Kries R, Koletzko B, Sauerwald T et al. Breast feeding and obesity: cross sectional study. Bmj 1999: 319: 147–150.
141. Dewey KG, Peerson JM, Brown KH et al. Growth of breast-fed infants deviates from current reference data: a pooled analysis of US, Canadian, and European data sets. World Health Organization Working Group on Infant Growth. Pediatrics 1995: 96: 495–503.
142. Elliott KG, Kjolhede CL, Gournis E, Rasmussen KM. Duration of breastfeeding associated with obesity during adolescence. Obes Res 1997: 5: 538–541.
143. Gillman MW, Rifas-Shiman SL, Camargo CA Jr. et al. Risk of overweight among adolescents who were breastfed as infants. JAMA 2001: 285: 2461–2467.
144. Birch LL, Fisher JO. Development of eating behaviors among children and adolescents. Pediatrics 1998: 101: 539–549.
145. Coulson NS, Eiser C, Eiser JR. Diet, smoking and exercise: interrelationships between adolescent health behaviours. Child Care Health Dev 1997: 23: 207–216.
146. Yerushalmy J. The relationship of parents' cigarette smoking to outcome of pregnancy – implications as to the problem of inferring causation from observed associations. Am J Epidemiol 1971: 93: 443–456.
147. Butler NR, Goldstein H, Ross EM. Cigarette smoking in pregnancy: its influence on birth weight and perinatal mortality. Br Med J 1972: 2: 127–130.
148. Levin ED, Wilkerson A, Jones JP, Christopher NC, Briggs SJ. Prenatal nicotine effects on memory in rats: pharmacological and behavioral challenges. Brain Res Dev Brain Res 1996: 97: 207–215.
149. Hamosh M. Does infant nutrition affect adiposity and cholesterol levels in the adult? J Pediatr Gastroenterol Nutr 1988: 7: 10–16.
150. Hauner H, Rohrig K, Petruschke T. Effects of epidermal growth factor (EGF), platelet-derived growth factor (PDGF) and fibroblast growth factor (FGF) on human adipocyte development and function. Eur J Clin Invest 1995: 25: 90–96.
151. Birch LL, McPhee L, Steinberg L, Sullivan S. Conditioned flavor preferences in young children. Physiol Behav 1990: 47: 501–505.
152. Steptoe A. Psychobiological processes in the etiology of disease. In: Martin PR, ed. Handbook of Behavior Therapy and Psychological Science: An Integrative Approach. Pergamon Press, New York, 1991, pp 325–347.
153. Cook VV, Hurley JS. Prevention of type 2 diabetes in childhood. Clin Pediatr 1998: 37: 123–129.
154. Holcomb JD, Lira J, Kingery PM et al. Evaluation of Jump Into Action: a program to reduce the risk of non- insulin dependent diabetes mellitus in school children on the Texas- Mexico border. J Sch Health 1998: 68: 282–288.
155. Holcomb JD, Kingery PM, Sherman LD. Evaluation of a diabetes education program for fifth-grade students. J Health Educ 1999: 30: 73–82.
156. McAuley KA, Williams SM, Mann JI et al. Diagnosing insulin resistance in the general population. Diabetes Care 2001: 24: 460–464.
157. Marlow E, Melkus GD, Bosma AM. STOP diabetes! An educational model for Native American adolescents in the prevention of diabetes. Diabetes Educ 1998: 24: 446–450.

158. McKenzie SB, O'Connell J, Smith LA, Ottinger WE. A primary intervention program (pilot study) for Mexican American children at risk for type 2 diabetes. Diabetes Educ 1998: 24: 180–187.
159. Trevino RP, Pugh JA, Hernandez AE et al. Bienestar: a diabetes risk-factor prevention program. J Sch Health 1998: 68: 62–67.
160. Teufel NI, Ritenbaugh CK. Development of a primary prevention program: insight gained in the Zuni Diabetes Prevention Program. Clin Pediatr (Phila) 1998: 37: 131–141.
161. Macaulay AC, Paradis G, Potvin L et al. The Kahnawake Schools Diabetes Prevention Project: intervention, evaluation, and baseline results of a diabetes primary prevention program with a native community in Canada. Prev Med 1997: 26: 779–790.
162. Mackenzie SB, O'Connell J, Smith LA, Ottinger WE. A primary intervention program (pilot study) for Mexican American children at risk for type 2 diabetes. Diabetes Educ 1998: 24: 180–187.
163. Bandura A. Social Functions of Thought and Action: a Social Cognitive Theory. Prentice Hall, Englewood Cliffs, NJ, 1986.
164. Stone EJ, Baranowski T, Sallis JF. Review of behavioral research for cardiopulmonary health. Emphasis on youth, gender, and ethnicity. J Health Educ 1995: 26: S9–S17.
165. Perry CL, Luepker RV, Murray DM et al. Parent involvement with children's health promotion: a one-year follow-up of the Minnesota home team. Health Educ Q 1989: 16: 171–180.
166. Parcel GS, Simons-Morton B, O'Hara NM, Baranowski T, Wilson B. School promotion of healthful diet and physical activity: impact on learning outcomes and self-reported behavior. Health Educ Q 1989: 16: 181–199.
167. Walter HJ, Hofman A, Vaughan RD, Wynder EL. Modification of risk factors for coronary heart disease. Five-year results of a school-based intervention trial. N Engl J Med 1988: 318: 1093–1100.
168. Nader PR, Sallis JF, Patterson TL et al. A family approach to cardiovascular risk reduction: results from the San Diego Family Health Project. Health Educ Q 1989: 16: 229–244.
169. Luepker RV, Perry CL, McKinlay SM et al. Outcomes of a field trial to improve children's dietary patterns and physical activity. The Child and Adolescent Trial for Cardiovascular Health. CATCH collaborative group [see comments]. JAMA 1996: 275: 768–776.
170. Davis SM, Lambert LC, Gomez Y. Southwest cardiovascular curriculum project: study findings for American Indian elementary students. J Health Educ 1995: 275: 768–776.
171. Green LW, Kreuter MW. Health Promotion Planning: An Educational and Environmental Approach. Mayfield, Mountain View, CA, 1991.
172. Knowler WC, Williams RC, Pettitt DJ, Steinberg AG. Genetics and type 2 diabetes mellitus: an association in American Indians with genetic admixture. Am J Human Genet 1988: 43: 520–526.
173. O'Sullivan JB, Mahan CM. Blood sugar levels, glycosuria, and body weight related to development of diabetes mellitus. The Oxford epidemiologic study 17 years later. JAMA 1965: 194: 587–592.
174. Baird JD. Diabetes mellitus and obesity. Proc Nutr Soc 1973: 32: 199–203.
175. Everhard J, Knowler WC, Bennett PH. Incidence and risk factors for noninsulin-dependent diabetes. In: Harris MI, Hamman RF, eds. National Diabetes Data Group: Diabetes in America. National Institute of Diabetes and Digestive and Kidney Diseases, Bethesda, MD, 1985.
176. Haffner SM, Kennedy E, Gonzalez C, Stern MP, Miettinen H. A prospective analysis of the HOMA model. The Mexico City Diabetes Study. Diabetes Care 1996: 19: 1138–1141.
177. Lillioja S, Mott DM, Spraul M et al. Insulin resistance and insulin secretory dysfunction as precursors of non-insulin-dependent diabetes mellitus. Prospective studies of Pima Indians. N Engl J Med 1993: 329: 1988–1992.
178. Warram JH, Martin BC, Krolewski AS, Soeldner JS, Kahn CR. Slow glucose removal rate and hyperinsulinemia precede the development of type II diabetes in the offspring of diabetic parents. Ann Intern Med 1990: 113: 909–915.
179. Saad MF, Knowler WC, Pettitt DJ et al.The natural history of impaired glucose tolerance in the Pima Indians. N Engl J Med 1988: 319: 1500–1506.
180. Sicree RA, Zimmet PZ, King HO, Coventry JS. Plasma insulin response among Nauruans. Prediction of deterioration in glucose tolerance over 6 yr. Diabetes 1987: 36: 179–186.
181. Haffner SM, Miettinen H, Gaskill SP, Stern MP. Decreased insulin secretion and increased insulin resistance are independently related to the 7-year risk of NIDDM in Mexican-Americans. Diabetes 1995: 44: 1386–1391.
182. Bergstrom RW, Newell-Morris LL, Leonetti DL et al. Association of elevated fasting C-peptide level and increased intra-abdominal fat distribution with development of NIDDM in Japanese-American men. Diabetes 1990: 39: 104–111.
183. Huang TT, Johnson MS, Goran MI et al. Development of a prediction equation for insulin sensitivity from anthropometry and fasting insulin in prepubertal and early pubertal children. Diabetes Care 2002: 25: 1203–1210.
184. Stone EJ, Perry CL, Luepker RV. Synthesis of cardiovascular behavioral research for youth health promotion. Health Educ Q 1989: 16: 155–169.
185. Dean H. NIDDM-Y in First Nation children in Canada. Clin Pediatr 1998: 37: 89–96.
186. Auslander WF, Thompson S, Dreitzer D, White NH, Santiago JV. Disparity in glycemic control and adherence between African-American and Caucasian youths with diabetes. Family and community contexts. Diabetes Care 1997: 20: 1569–1575.
187. Rogers RW. A protection motivation theory of fear appeals and attitude change. J Psychol 1975: 91: 93–114.
188. Gochman DS. Personal and social determinants of health behavior. In: Gochman DS, ed. Handbook of Health Behavior Research I. Social and Personal Determinants. Plenum Press, New York, 1997.
189. Fabrega H. Historical and cultural foundations of health behavior. In: Gochman DS, ed. Handbook of Health Behavior Research I. Personal and Social Determinants. Plenum Press, New York, 1997.
190. Dressler WW, Oths KS. Cultural determinants of health behavior. In: Gochman, DS, ed. Handbook of Health Behavior Research I. Personal and Social Determinants. New York: Plenum Press 1997.
191. Rappaport J. Terms of empowerment/exemplars of prevention: toward a theory for community psychology. Am J Community Psychol 1987: 15: 121–148.
192. Hovland CI, Janis IL, Kelley HH. Communication and Persuasion: Psychological Studies of Opinion Change. Greenwood Press, Westport, CT, 1953.
193. Teufel NI, Ritenbaugh CK. Development of a primary prevention program: insight gained in the Zuni

Diabetes Prevention Program. Clin Pediatr 1998: 37: 131–141.
194. Pihoker C, Scott CR, Lensing SY, Cradock MM, Smith J. Non-insulin dependent diabetes mellitus in African-American youths of Arkansas. Clin Pediatrics 1998: 37: 97–102.
195. Bandura A. Self-efficacy: toward a unifying theory of behavioral change. Psychol Rev 1977: 84: 191–215.
196. Glasgow RE, Toobert DJ, riddle M et al. Diabetes-specific social learning variables and self-care behaviors among persons with type II diabetes. Health Psychol 1989: 8: 285–303.
197. Dishman RK, Ickes W, Morgan WP. Self-motivation and adherence to habitual physical activity. J Appl Social Psychol 1980: 10: 115–132.
198. Glasgow RE, Hampson SE, Strycker LA, Ruggiero L. Personal-model beliefs and social–environmental barriers related to diabetes self-management. Diabetes Care 1997: 20: 556–561.
199. Wallston B, Alagna S, Devellis B, Devellis R. Social support and physical health. Health Psychol 1983: 2: 367–391.
200. Vugia HD. Support groups in oncology: building hope through the human bond. J Psychosoc Oncol 1991: 9: 89–107.
201. Bentler PM. Etiologies and consequences of adolescent drug use: implications for prevention. J Addict Dis 1992: 11: 47–61.
202. Palardy N, Greening L, Ott J, Holderby A, Atchison J. Adolescents' health attitudes and adherence to treatment for insulin-dependent diabetes mellitus. J Dev Behav Pediatr 1998: 19: 31–37.
203. Spruijt-Metz D. Adolescent, Affect, and Health. Psychology Press, London, 1999.
204. Spruijt-Metz D. Personal incentives as determinants of adolescent health behavior. The meaning of behavior. Health Educ Res 1995: 10: 355–364.
205. Brown LK, Diclemente RJ, Reynolds LA. HIV prevention for adolescents: utility of the Health Belief Model. AIDS Educ Prev 1991: 3: 50–59.
206. Brown LK, Diclemente RJ, Park T. Predictors of condom use in sexually active adolescents. J Adolesc Health 1992: 13: 651–657.
207. Sturges JW, Rogers RW. Preventive health psychology from a developmental perspective: an extension of protection motivation theory. Health Psychol 1996: 15: 158–166.
208. Tappe MK, Duda JL, Menges-Ehrnwald P. Personal investment predictors of adolescent motivational orientation toward exercise. Can J Sport Sci 1990: 15: 185–192.
209. Marrero DG, Fremion AS, Golden MP. Improving compliance with exercise in adolescents with insulin-dependent diabetes mellitus: results of a self-motivated home exercise program. Pediatrics 1988: 81: 519–525.
210. Piaget J. Langage et pensee. Revue Praticien 1965: 15: 2253–2254.
211. Joe J. Perceptions of diabetes by Indian adolescents. In: Joe JR Young RS, eds. Diabetes of Culture and Civilization: the Impact of Culture Change on Indigenous Peoples. Mouton de Gruter, Berlin, 1994, pp 329–356.
212. Jones KL. Non-insulin dependent diabetes in children and adolescents: the therapeutic challenge. Clin Pediatr 1998: 37: 103–110.
213. Braithwaite RL, Lythcott N. Community empowerment as a strategy for health promotion for black and other minority populations. JAMA 1989: 261: 282–283.
214. Parker RG. Empowerment, community mobilization and social change in the face of HIV/AIDS. AIDS 1996: 10(Suppl. 3): S27–31.