Scientific Statement: Socioecological Determinants of Prediabetes and Type 2 Diabetes

James O. Hill, PhD
James M. Galloway, MD
April Goley, RN, MSN, FNP-C
David G. Marrero, PhD
Regan Minners, BA
Brenda Montgomery, RN, MS, CDE
Gregory E. Peterson, DO
Robert E. Ratner, MD
Eduardo Sanchez, MD, MPH
Vanita R. Aroda, MD

In this article, we examine the socioecological determinants—the biological, geographic, and built environment factors—that influence risk for prediabetes and type 2 diabetes. A socioecological perspective looks beyond the individual to evaluate a multitude of influences, from the surrounding home, work, school, and community environments to social determinants and the influence of public policy on individual behavior (1). Figure 1, adapted from the Institute of Medicine socioecological model of childhood obesity, provides a good framework for understanding potential socioecological determinants of risk for type 2 diabetes.

In November 2012, the American Diabetes Association Prevention Committee convened a writing group to review the evidence on socioecological factors contributing to recent increases in prediabetes and type 2 diabetes. Drawing from the work of the committee, in this article we review the overarching evidence-based contributions of socioecological factors to risk for type 2 diabetes. Rather than incorporate the entire universe of relational observations, this scientific statement is intended to evaluate the extent to which data indicate a contributing role of social and environmental factors to the current epidemic of type 2 diabetes.

Epidemiological trends in obesity and diabetes—The world is in the midst of parallel and rapidly advancing epidemics—obesity and type 2 diabetes—that began in the latter half of the 20th century and continue to grow, unchecked. Current prevalence rates are staggering and are expected to continue to climb over the ensuing decades.

In the U.S., one-third of adults and 16–18% of youth are obese (2), up from 5 to 6% three decades ago (Fig. 2). Increases in rates of type 2 diabetes have closely followed the increases in obesity. In the U.S., diabetes affects 8.3% of the population, including 18.8 million with diagnosed diabetes and another 7 million who remain undiagnosed (3,4). An additional 35% of U.S. adults, or 79 million Americans aged ≥20 years, have prediabetes and are therefore at increased risk for developing type 2 diabetes. Moreover, it is estimated that one in three American adults will have diabetes by the year 2050 if current trends continue (5).

These epidemics have become global. An estimated 500 million people worldwide are obese, and another 1.5 billion are overweight (Table 1). Further, 2.8 million people die each year (7) due to overweight and obesity.

In 2011, 366 million people worldwide had diabetes. In that same year, diabetes caused 4.6 million deaths (8).

The International Diabetes Federation estimates that by 2030, the number of individuals with diabetes will rise by almost 43% to 552 million. In 2011, about 280 million people had prediabetes (8); by 2030 this number is expected to rise to nearly 400 million.

Globalization and westernization of the developing world continue to contribute to the rapid worldwide growth of type 2 diabetes and obesity (9).

Current consequences of the obesity and diabetes epidemics—These parallel epidemics present serious global crises with significant public health and economic consequences. In the U.S., diabetes is the seventh leading cause of death, with a doubling of the risk of death in people with diabetes compared with those without diabetes (10). And these data may underrepresent the problem as death records may not accurately portray the extent to which diabetes contributes to mortality (11).

The rising prevalence of type 2 diabetes has contributed substantially to the increasing prevalence of complications related to the disease. In the U.S., the number of people aged ≥35 years with diabetes and self-reported heart disease or stroke increased from 4.2 million to 7.6 million from 1997 to 2011. Similarly, the number of persons who initiated treatment for end-stage renal disease attributable to diabetes increased from over 2,600 in 1980 to over 48,000 in 2008 (12).

Diabetes also affects the workforce and work productivity. Insurance costs average 2.3 times higher for people with diabetes than for those without diabetes; while annual insurance costs for people without diabetes average $5,615, for people with diabetes those costs rise to $12,915. Diabetes is also a significant predictor of lost work productivity and health-related work limitations (13).

There are also potential ramifications of overweight, obesity, and diabetes.
on the military. Currently, one out of every four military recruits is overweight or obese and unable to join the armed forces (14). The increasing prevalence of obesity and type 2 diabetes in younger populations may limit the potential pool of military recruits and military readiness.

Costs for diagnosed and undiagnosed diabetes are even more staggering (Table 2). In the U.S., the total cost of diagnosed diabetes in 2012 was $245 billion (15), which includes $176 billion in direct costs and $69 billion in reduced productivity. This represents a 41% increase from the last estimate of $174 billion in 2007. Gestational diabetes mellitus, prediabetes, and undiagnosed diabetes cost an additional $217 billion a year. Currently one in five health care dollars is spent caring for someone with diagnosed diabetes. Nearly one out of three Medicare recipients has diagnosed diabetes (16), and diabetes represents a disproportionate amount—nearly one-third—of the Medicare budget (17).

These new data attribute the higher costs to changing demographics in the U.S.; an increasing prevalence of diabetes, with a 27% growth in the disease prevalence in the last 5 years; decreasing mortality; and refinement of the data calculations.

**Socioecological perspective of prediabetes and diabetes risk**—While much research on risk factor assessment and obesity and diabetes...
Table 1—Definitions of overweight and obesity*

| Category            | Definitions                                                                 |
|---------------------|-----------------------------------------------------------------------------|
| Adults              | Overweight is defined as a BMI (a ratio of weight in kilograms to the square of height in meters) of 25.0–29.9 kg/m². Obesity is defined as a BMI of ≥30 kg/m². |
| Children and adolescents | Overweight and obesity in children are defined by categories based on the CDC age- and sex-specific growth curves. Overweight, including obesity, is defined as a BMI at or above the 85th percentile. Obesity is defined as a BMI at or above the 95th percentile. |

*BMI thresholds vary by race/ethnicity, and some ethnic groups are at elevated risk at lower BMIs.

Socioecological influences in obesity and diabetes risk—As illustrated in Fig. 1, there are various levels and sectors of influence on obesity and diabetes risk. These social and environmental influences are ultimately mediated through increases in energy intake relative to energy expenditure. Here we describe changes in patterns in food consumption and physical activity in relation to diabetes risk, followed by concrete examples of factors beyond the individual and involving broader societal and environmental factors that contribute to increased diabetes risk through either or both of these mechanisms. We have limited our review to areas that are data-rich relative to their influence on diabetes risk. We have thus not addressed certain areas such as the effect of public policy and social media, as the data in these areas focus primarily on intermediary behaviors rather than diabetes risk.

Patterns in food consumption and obesity and diabetes risk
Socioecological influences in obesity and diabetes risk are mediated through increased food and beverage intake and changes in physical activity. Patterns of food consumption over the last few decades suggest factors beyond the individual, which contribute to increased calorie intake. In the U.S., data compiled by the Centers for Disease Control and Prevention (CDC) and the National Center for Health Statistics show that total calorie intake increased from 2,450 kcal/day in the 1970s to 2,656 kcal/day in the 2000s in males aged ≥20 years and increased from 1,542 kcal/day to 1,811 kcal/day in females aged ≥20 years during the same time period (21). Moreover, daily total energy intake increased in parallel in children aged 2–18 years from 1,842 kcal/day to 2,022 kcal/day from the periods of 1977–1978 to 2003–2006 (22).

Portion size increases may have contributed in part to the excess calorie intake seen in the last few decades. Indeed, portion sizes of soft drinks, fruit drinks, and fast food have all increased for both children and adults since the 1970s (23). Retail food promotions, excess availability of calorie-dense food, and increase in food consumption away from the home are all possible contributing factors (24).

The U.S. food supply has been high in fat for many years, and the additional energy intake seems to have come from carbohydrates and possibly from increased added sugar. A food supply high in sugar and fat is also high in energy density. Controlled research has clearly shown that all of these factors—high sugar, high fat, and high energy density—increase voluntary energy intake (25).

Observational research also relates weight gain to qualitative components of the diet. One prospective evaluation involving three separate cohorts included over 120,000 men and women free of...
chronic disease at baseline and found that increased consumption of potato chips, potatoes, sugar-sweetened beverages (SSBs), unprocessed red meat, and processed meats was positively associated with weight gain, while intake of fruits, vegetables, whole grains, nuts, and yogurt was inversely associated with weight gain (26). A 13-year prospective analysis of the Coronary Artery Risk Development in Young Adults (CARDIA) study evaluating cardiovascular risk factor development in young adults showed that increased fast food consumption was associated with an increase in both weight gain and insulin resistance as compared with infrequent fast food consumption (27). In the Nurses’ Health Studies, consumption of whole grains, cereal fiber, and polyunsaturated fatty acids was associated with reduced risk of diabetes, while consumption of rapidly absorbed carbohydrates and trans fat was associated with increased diabetes risk (28–30).

Several large studies with long duration of follow-up show a strong relationship between SSB consumption and type 2 diabetes. In a study following over 50,000 women for 8 years, consumption of ≥1 SSB per day was associated with an 83% greater risk of developing type 2 diabetes compared with consumption of <1 SSB per day, with BMI being a significant mediator of that risk (31). Similar findings have been shown in several other large prospective studies. An analysis by Malik et al. (32), which pooled together 11 prospective studies evaluating risk of metabolic syndrome and type 2 diabetes with SSB intake, included over 300,000 participants and over 15,000 cases of type 2 diabetes and demonstrated a 26% greater risk of developing type 2 diabetes in individuals in the highest category of SSB intake compared with those in smallest category of intake.

There is agreement that the U.S. food supply is high in sugar, fat, and energy density and that such a food supply is associated with increased energy intake. We have far less agreement and data on the effect of modifying individual components of food on food intake and diabetes risk. Such research is needed and could help develop better interventions to prevent obesity and diabetes.

Changes in physical activity and obesity and diabetes risk
Concomitant with the changes in food availability and consumption, studies reveal that recommended levels of physical activity are not being met. Despite an increase from the previous decade, only 19% of adults are meeting current physical activity guidelines. According to the recent Institute of Medicine report “Accelerating Progress in Obesity Prevention: Solving the Weight of the Nation” (4), while there is an overall increase in leisure-time physical activity, there has been an overall decline in active transportation (e.g., walking, biking). In children and adolescents, there have been decreases in physical education classes, reported availability of leisure time, and in active transport (walking/bicycling to school). Increased use of digital media and decreased access to and incorporation of physical activity in normal home, work and school routines have all contributed to not meeting recommended physical activity goals.

Just as with dietary patterns, both epidemiological and interventional studies suggest an increased risk of obesity and diabetes with decreased physical activity. In some countries, such as Great Britain and the Netherlands, reduced energy expenditure is thought to be a greater contributor to current obesity trends than increased food intake (33). Sedentary behavior such as prolonged television watching in particular confers increased risk of obesity and diabetes. In the Nurses’ Health Study, for example, every 2-h/day increment of time watching television was associated with a 23% increase in obesity and a 14% increased risk of diabetes, while a 2-h/day increment of standing or walking was associated with a 12% reduction in risk and each 1-h/day increment of brisk walking was associated with a 34% reduction in risk (34).

Relationship of the environment with changes in food consumption and physical activity
The influence of changes in food consumption and physical activity trends on obesity and diabetes risk has been exacerbated by accompanying environmental changes. Here we review several examples and the evidence of their contribution to obesity and diabetes risk.

1. Effects of global urbanization and acculturation. Urbanization and economic growth may contribute to increased diabetes risk through a number of factors: from increasing access to high-fat, calorie-dense foods and beverages to promoting consumption of larger portions, and increased processed foods prepared outside of the home (35). While increasing efficiencies in everyday home and work life, advances in technology and transportation may also contribute to decreased energy expenditure and increased time engaged in sedentary lifestyle behaviors.

Several studies in the developing world have evaluated impact of urbanization on diabetes prevalence. An evaluation in China, which saw a greater than twofold increase in diabetes prevalence from 3% in 1994 to 7–10% in 2008, found that diabetes was approximately twice as prevalent in high versus low urbanized areas, even after accounting for factors such as community, province, age, sex, and household income. Modern markets, as described by the number of grocery stores, cafes, internet cafes, restaurants, mobile eateries, fast food restaurants, and ice cream parlors in the community, were positively associated with diabetes prevalence, as were community-level factors such as transportation infrastructure (presence and higher number of paved vs. gravel or dirt roads and bus and/or train stations in the community) and communications (percentage of households with a television, computer, or cell phone and presence of a cinema, newspaper, and telephone service in the community) (36). Similar associations between urbanization and cardiovascular risk factors and diabetes have been documented in other countries (37–40).

Of note, while global urbanization is resulting in increases in the prevalence of diabetes worldwide, in the U.S., there remains a disproportionately high prevalence of diabetes in rural communities. Indeed, some studies report higher rates of diabetes and obesity in rural areas compared with urban centers in the U.S. (41–45). This is likely related to a multitude of other social and environmental influences, such as poverty, low socioeconomic status, and reduced access to health care, as well as attributes of the built and neighborhood environments, such as access to safe walkable communities and healthy food establishments, as discussed below. In a study of behaviors and weight status in rural U.S. communities, eating out frequently at buffets, caterers, and fast food restaurants was associated with higher rates of obesity, and perceiving the community as unpleasant for physical activity was also associated with obesity (46).
Migration studies also suggest an influence of acculturation on obesity and diabetes risk. Lifestyle changes—increased calorie consumption and decreased physical activity—in the new environments are thought to be key drivers of the acculturation effect. In the Multi-Ethnic Study of Atherosclerosis (MESA), greater acculturation, assessed by nativity, number of years in the U.S., and language spoken at home, was associated with higher diabetes prevalence in non–Mexican-origin Hispanics. This was partly mediated by BMI and diet (47). Studies in Japanese and Chinese Americans have also demonstrated higher diabetes prevalence associated with greater acculturation to a “Western” lifestyle (48,49). Similar examples are seen on the international level. Studies of Indian immigrants living in Western countries suggest influences of both dietary and activity changes, with increases in consumption of meat products and soft drinks, and lower levels of physical activity compared with those living in India (50).

2. Social determinants and the community environment. Social determinants of mortality and diabetes risk are also recognized, with factors such as poverty and education level contributing to mortality and health risk (51,52). Here, too, social determinants may influence diabetes risk by affecting access to and practice of healthy behaviors in the surrounding environment. Zip code, reflective of the socioeconomic environment, is increasingly recognized as a determinant of diabetes and complications. People living in low-income communities are more likely to be hospitalized for diabetes or related complications compared with those living in affluent areas (53). A randomized social experiment by Ludwig et al. (54) illustrates the impact of the neighborhood environment on health outcomes. In this study of over 4,000 women with children living in high-poverty urban public housing, moving from a high-poverty area to a low-poverty census tract was associated with modest reductions in the prevalence of extreme obesity and diabetes at 10–15 years of follow-up.

a. The community and the built environment. Physical and built environments significantly influence the likelihood of population-level engagement in physical activity and contribute to health risk and disparities. The built environment refers to “environments that are modified by humans, including homes, schools, workplaces, highways, urban sprawl, and accessibility to amenities, leisure, and pollution” and can contribute to diabetes risk through access to physical activity and other factors such as stress (55). Features of the neighborhood and built environment that influence physical activity include walkability/bikeability, community design, accessible destinations, safe intersections, green spaces, public transit availability, and availability of recreational facilities, promotion of neighborhood and social interactions, as well as personal safety within the environment. “Urban sprawl” is characterized by homes being far from community amenities, which require transit on busy, high-speed roads that are not conducive to walking or biking and increase vehicular transit time. The direct impact of the community and built environment on diabetes risk per se is a relatively new area of study, but studies on its effects on weight, BMI, and activity levels, primary risk factors for diabetes, illustrate its potential influence.

Such features of the built environment decrease physical activity, affect body weight, and thus have the potential to increase risk of diabetes. Higher BMIs have been noted in communities typified by this “urban sprawl” (56,57), likely related to decreased physical activity. Increased vehicular transit time has been noted in these urban environments and is associated with obesity. In a merged analysis from the California Health Interview Survey 2001, U.S. 2000 Census, and the California Department of Transportation, obesity and physical inactivity were significantly associated with vehicle miles of travel, and the highest mean rank obesity was associated with the highest rank of vehicle miles of travel (58). Another analysis reported a 6% increase in likelihood of obesity with each additional hour spent in a car per day and a 4.8% decrease in likelihood of obesity with each additional kilometer walked per day. Safe, walkable neighborhoods with a mixed-use community design are more conducive to active transportation and are associated with less automobile use and lower BMIs (59). Furthermore, safe neighborhoods facilitate more outdoor play and recreation for children and families, and the lack of these promotes more sedentary indoor activities, which in turn is a risk factor for overweight and obesity.

Convenient access to recreational areas and facilities also factors into health risk. In the National Longitudinal Study of Adolescent Health, lower socioeconomic status and high-minority residential blocks had reduced access to physical activity/recreational facilities. This was associated with decreased physical activity and increased overweight (60). Conversely, an increase in the number of recreational facilities is associated with both increased moderate-vigorous physical activity and decreased prevalence of overweight. Sallis et al. (61) also reported a 20–45% increased risk of overweight and obesity in children lacking access to sidewalks, paths, parks, playgrounds, or recreational centers.

b. The community and the food environment. The community environment also influences access to and consumption of healthy foods and may contribute to or reinforce health disparities. The term “food desert” is now used to refer to areas with limited access to affordable nutritious foods (62). These food desert communities have limited proximity to supermarkets and grocery stores and may have no food access or are served by less nutritious convenience stores or fast food restaurants (63). Food deserts disproportionately affect lower-income, minority, and rural neighborhoods, while access to fast food restaurants and energy-dense foods is higher in lower-income and minority neighborhoods (64–66). Most of the studies evaluating the effect of food deserts are cross-sectional and thus cannot be interpreted for causality, but highlight the potential effect on diet and health and the need for additional studies and intervention strategies.

Access to neighborhood grocery stores and farmers’ markets appears to be associated with healthier food intake and lower levels of overweight and obesity (65,67–69), while greater availability of fast food restaurants and lower prices of fast food restaurant items appear to be associated with poorer diet. In one study evaluating the effect of proximity to fast food restaurants on obesity among ninth graders, the rate of obesity in the ninth graders increased by 5.2% for schools located within 0.10 mile of a fast food restaurant compared with schools located within 0.25 miles. The same study evaluated the effect of distance to fast food restaurant on weight gain during pregnancy and found that living within half a mile of a fast food restaurant increased the likelihood of gaining more than 20 kg during pregnancy by 2.5% (70).
Another related phenomenon, termed “food insecurity,” refers to limited or uncertain access to food resulting from inadequate financial resources. Rates of food insecurity have been rising in the U.S. since 1999, with a reported 12% of the population living in households experiencing food insecurity in 2004. A relationship between food insecurity and diabetes prevalence has been documented in cross-sectional analysis of the National Health and Nutrition Examination Survey (NHANES) 1999–2002, with participants with severe food insecurity having an approximately twofold risk of diabetes compared with those without food insecurity, which persisted even after accounting for BMI (71). In the circumstances of food insecurity, it is possible that individuals rely more heavily on less nutritious, inexpensive, calorie-dense food alternatives, again highlighting the importance of affordable access to good-quality nutritious food.

c. Work and home environments. While enhancing efficiencies of work and communication, technological advances have significantly redefined work environments. Several studies have documented a high level of sedentary behavior in the work environment, which is often prolonged, defined as ≥20 min, as well as less engagement in light-intensity activity during working hours (72–75). Increased sedentary behavior appears to disproportionately affect professional, managerial, white collar jobs compared with technical or blue collar jobs. Time spent in sedentary behaviors—whether it is television viewing at home or sitting or screen time at work—is now considered an independent risk factor for several health outcomes (34,72,74,76–97). There is a large pool of evidence associating sedentary behavior with increased obesity, diabetes, and cardiovascular risk, as well as premature mortality, but only a suggestion that interventions that decrease or interrupt sedentary behavior in the work environment may improve metabolic risk (98–100). Church et al. (101) estimated that physical activity in the workplace has decreased by 120–140 kcal/day over the past five decades.

Home environment mediators may also contribute to health disparities. In the Neighborhood Impact on Kids Project, for example, children from lower socioeconomic status homes, associated with increased overweight/obesity, had greater media access in their bedrooms, higher daily screen time and lower access to portable play equipment, such as bikes and jump ropes, compared with children from higher socioeconomic status homes (102). Intervention trials are necessary to validate this hypothesis.

Home environments have also evolved over the last decades, with increased and more prominent television and digital media exposure contributing to sedentary behavior, increased availability and use of labor-saving devices in the home environment, and cultural shifts in eating patterns at home (4). Archer et al. (103) estimated that energy expended in household work has declined by 25% since 1965. Families are eating fewer meals together (104), with more calories consumed outside of the home, and fast food and less nutritious foods and beverages are being consumed more frequently at home than previously (4). Sociological factors such as single-parent homes or two working parents have been invoked as contributors.

d. School and the surrounding environment. There is increasing recognition and focus on the importance of the school environment in students’ eating and physical activity behaviors. The school environment directly influences quantity and quality of food availability, with increasing portion sizes and more energy-dense foods available to children and adolescents in the school environment. The school environment is an important determinant of access to physical education and cultural norms surrounding eating and physical activity behaviors. Academic priorities and limited availability of qualified staff and resources saw a decrease in emphasis on physical education activity in the 1990s. In high-school students, the percent attending physical education classes daily declined from 41.6% in 1991 to 33.3% in 2009 (4). As with adults, active transport (i.e., walking or biking to school) has also decreased from 20.2% of school trips in 1977 to 12.5% of school trips in 2001 (4). School food environments have also changed over the last few decades, with more availability and promotion of high-calorie foods and snacks (105).

The environment surrounding schools is also associated with food choices and eating behaviors and may also contribute to socioeconomic health disparities. Babej et al. (106) report that adolescents who live and attend school in areas with more fast food restaurants and convenience stores are more likely to consume soda and fast food compared with those who live and attend school in healthier food environments.

The importance of the school environment in modifying risk for overweight, obesity, and type 2 diabetes is highlighted in the HEALTHY study (107) and in other school-based intervention studies evaluating glucose and diabetes risk (108,109). In the HEALTHY study, 4,603 students across 42 schools were assigned to a multicomponent school-based intervention addressing nutrition, physical activity, behavioral knowledge and skills, and communications and social marketing, compared with a control arm followed for assessment only. Students were evaluated at the beginning of the sixth grade and at the end of the eighth grade. Although there was a decrease in the combined prevalence of overweight and obesity in both the intervention and the control schools, the intervention schools had greater reductions in BMI z-score, percentage of students with waist circumference at or above the 90th percentile, fasting insulin levels, and a near-significant reduction in the prevalence of obesity (P = 0.05). Among the ~50% of students who were overweight or obese at the beginning of the sixth grade, there was a significant 21% decrease in risk of being obese at the end of the eighth grade in the intervention schools. Although mean plasma glucose levels did not significantly differ, it was notable that 30% of those who were in the 95th percentile or higher of BMI in the eighth grade had glucose levels of 100 mg/dL or higher compared with 19% of those under the 85th percentile.

**Addressing obesity and lifestyle changes is critical in preventing diabetes and prediabetes**—Nearly 26 million American adults have diabetes (3). Moreover, almost 80 million have prediabetes, defined by impaired glucose tolerance (IGT) or impaired fasting glucose, placing them at substantially increased risk for developing type 2 diabetes and cardiovascular disease (110–113). The prevalence of diabetes in the U.S. is expected to double over the next 30 years, making it a major public health priority (114,115). The development of sustainable strategies to prevent the development of diabetes remains a mounting challenge for the health of the U.S. population.

Increasing evidence that type 2 diabetes may be prevented or delayed by lifestyle modification interventions...
designed to achieve modest weight loss and increase physical activity serves as the premise for large-scale interventions (116–118). Indeed, the relationship between obesity and diabetes is of such interdependence that the term “diabesity” has been coined (10). The first study to document this link was conducted in Da Qing, China (117). In this study, 577 adults with IGT were randomized by clinic site to one of three conditions or a no treatment control: a diet-only intervention, an exercise only intervention, or a combined diet and exercise intervention. The cohort was followed initially for 6 years with the end point being the development of type 2 diabetes. After 6 years, the diet-only condition reduced risk of converting to type 2 diabetes by 31%, the exercise only condition by 46%, and the combined diet and exercise condition by 41%. The association between lifestyle and diabetes among individuals with increased risk was further substantiated by the Finnish Diabetes Prevention Study (FDPS) and the Diabetes Prevention Program (DPP) (118,119). In the FDPS, 522 individuals at five centers in Finland who had IGT were randomized to a diet intervention or control group. Intervention subjects were given detailed, individualized instructions of dietary changes to achieve a weight loss of at least 5%. In addition, supervised exercise sessions were offered. The results showed that subjects in the intervention group had a 58% reduction in risk. This finding was replicated in the DPP, which studied 3,234 adult participants with IGT from 27 centers across the U.S. (119).

The DPP was unique in comparison with the previous studies in that it had a very heterogeneous population with 45% minority representation. Moreover, it was the first study to compare a lifestyle intervention with medication. Subjects were randomized to one of four conditions: lifestyle with a goal of 7% weight loss and at least 150 min/week of moderate-intensity physical activity, therapeutic intervention with metformin, using troglitazone that was later stopped due to potential liver toxicity, or a placebo control. The results showed that metformin reduced risk by 31%. The lifestyle condition, on the other hand, reduced risk by 58% overall and nearly 70% in individuals aged ≥ 65 years. Thus, lifestyle was nearly twice as effective as the medication option.

Collectively, these studies illuminate the vital role that lifestyle modification plays in preventing type 2 diabetes in high-risk individuals. Aside from family history, obesity is the most significant risk factor for type 2 diabetes. Research has also demonstrated that primary prevention programs can be effectively implemented in community settings in a cost-effective manner (120–122). This illustrates that diabetes prevention interventions could potentially be scalable on a national level given sufficient support.

**Call to action**—To date, intervention for the prevention of diabetes has followed a medical model of identification of individuals at risk and enrollment of at-risk volunteers into one-on-one or small group intervention trials. It is essential to better understand how social and environmental variables influence behaviors that lead to obesity, prediabetes, and diabetes and to learn how to modify these variables to prevent and manage them. Research in this area remains associational in nature, but the amount of data linking these factors is substantial. Efforts aimed toward individual behavior change are difficult, with extensive data on the failure of weight loss programs and the compensatory biologic responses that promote weight regain. The extent to which the environment may limit the effectiveness of prior efforts to achieve weight loss and maintenance is unclear, but the associations noted above clearly point to a potential role for environmental interventions in the initial prevention of overweight and obesity.

The American Diabetes Association recognizes the association between social and environmental factors and the development of obesity and type 2 diabetes. To date, the medical model focused primarily on the individual has provided limited benefit in curbing the epidemics of obesity and type 2 diabetes. A public health approach to alter the environments in which we live, learn, and work in order to enable healthy behavior and healthy lifestyles and, therefore, to promote health and prevent disease may hold promise for making further progress against these epidemics.

This review provides a number of potential targets for intervention to prospectively evaluate the role of social and environmental factors in the development of obesity and diabetes. Only well-designed interventions that focus both on the individual and on the social and physical environment in which the individual lives will answer the question. Our failure to adequately address the growing epidemics of obesity and type 2 diabetes will soon overwhelm our health care system, and investment in targeted research toward these identified social and environmental factors appears to be a productive avenue to improve our nation’s health.

**Acknowledgments**—No potential conflicts of interest relevant to this article were reported. J.O.H., D.G.M., R.M., G.E.P., and V.R.A. researched data and wrote the manuscript. J.M.G., A.G., B.M., R.E.R., and E.S. contributed to the discussion and reviewed and edited the manuscript.

**References**

1. Committee on Progress in Preventing Childhood Obesity. Progress in Preventing Childhood Obesity: How Do We Measure Up? Washington, DC, the National Academies Press, 2007

2. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of obesity in the United States, 2009–2010. NCHS data brief, 82. Hyattsville, MD, National Center for Health Statistics, 2012

3. Centers for Disease Control and Prevention. National Diabetes Fact Sheet: National Estimates and General Information on Diabetes and Prediabetes in the United States, 2011. Atlanta, GA, U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, 2011

4. Institute of Medicine. Accelerating Progress in Obesity Prevention: Solving the Weight of the Nation. Washington, DC, the National Academies Press, 2012

5. Boyle JP, Thompson TJ, Gregg EW, Barker LE, Williamson DF. Projection of the year 2050 burden of diabetes in the US adult population: dynamic modeling of incidence, mortality, and prediabetes prevalence. Popul Health Metr 2010;8:29

6. Behavioral Risk Factor Surveillance System, CDC 2000. Obesity trends among U.S. adults, BRFSS, 1990, 2000, 2010

7. World Health Organization. World Health Statistics 2012. Geneva, World Health Org, 2012

8. International Diabetes Federation. IDF Diabetes Atlas. 5th ed. International Diabetes Federation, 2012

9. Hu FB. Globalization of diabetes: the role of diet, lifestyle, and genes. Diabetes Care 2011;34:1249–1257

10. Centers for Disease Control and Prevention National Center for Chronic Disease Prevention and Health Promotion. Successes and opportunities for
population-based prevention and control at a glance 2011. Available from http://www.cdc.gov/chronicdisease/resources/publications/AAG/dt.htm. Accessed 22 April 2013
11. McEwen LN, Kim C, Haan M, et al.; TRIAD Study Group. Diabetes reporting as a cause of death: results from the Translating Research Into Action for Diabetes (TRIAD) study. Diabetes Care 2006;29:247–253
12. Centers for Disease Control and Prevention. Diabetes complications. Available from http://www.cdc.gov/diabetes/statistics/complications_national.htm. Accessed 19 April 2013
13. Tunceli K, Bradley CJ, Nerenz D, Williams I, Pladevall M, Elston Lafata J. The impact of diabetes on employment and work productivity. Diabetes Care 2005;28:2662–2667
14. Christeson W, Taggart AD, Messmer-Zidell S. Too Fat to Fight: Retired Military Leaders Want Junk Food out of America’s Schools. Washington, DC, Mission: Readiness, 2010
15. American Diabetes Association. Economic costs of diabetes in the U.S. in 2012. Diabetes Care 2013;36:1033–1046
16. Centers for Medicare and Medicaid Services. Chronic Conditions Among Medicare Beneficiaries, Chart Book. Baltimore, MD, 2012
17. Gold M, Thornton C, Hedley, et al. Federal Medical and Disability Program Costs Associated With Diabetes, 2005: Summary of Methods and Key Findings. Mathematica Policy Research, Inc., Washington DC, 2007
18. National Research Council and Institute of Medicine. U.S. Health in International Perspective: Shorter Lives, Poorer Health. Panel on Understanding Cross-National Health Differences Among High-Income Countries. Woolf SH, Aron L, Eds. Committee on Population, Division of Behavioral and Social Sciences and Education, and Board on Population Health and Public Health Practice, Institute of Medicine. Washington, DC, the National Academies Press, 2013
19. Hurowitz JC. Toward a social policy for health. N Engl J Med 1993;329:130–133
20. Centers for Disease Control and Prevention. The social-ecological model: a framework for prevention. Available from http://www.cdc.gov/violenceprevention/overview/social-ecologicalmodel.html. Accessed 14 May 2013
21. National Center for Health Statistics. Health, United States, 2011: With Special Feature on Socioeconomic Status and Health. Hyattsville, MD, U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, 2012
22. Poti JM, Popkin BM. Trends in energy intake among US children by eating location and food source, 1977–2006. J Am Diet Assoc 2011;111:1156–1164
23. Nielsen SJ, Popkin BM. Patterns and trends in food portion sizes, 1977–1998. JAMA 2003;289:450–453
24. United States Department of Agriculture. Food consumption data system. Available from http://www.ers.usda.gov/topics/food-choices/health-food-consumption-demand/food-away-from-home.aspx#.UUKx6UHTFI1. Accessed 19 April 2013
25. Hall KD, Guo J, Dore M, Chow CC. The progressive increase of food waste in America and its environmental impact. PLoS ONE 2009;4:e7940
26. Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. Changes in diet and lifestyle and long-term weight gain in women and men. N Engl J Med 2011;364:2392–2404
27. Pereira MA, Kartashov AI, Ebbeling CB, et al. Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. Lancet 2005;365:36–42
28. de Munter JS, Hu FB, Spiegelman D, Franz M, van Dam RM. Whole grains, bran, and germ intake and risk of type 2 diabetes: a prospective cohort study and systematic review. PLoS Med 2007;4:e261
29. Schulze MB, Liu S, Rimm EB, Manson JE, Willett WC, Hu FB. Glycemic index, glycemic load, and dietary fiber intake and incidence of type 2 diabetes in younger and middle-aged women. Am J Clin Nutr 2004;80:348–356
30. Salmeron J, Hu FB, Manson JE, et al. Dietary fat intake and risk of type 2 diabetes in women. Am J Clin Nutr 2001;73:1019–1026
31. Fagherazzi G, Vilier A, Sae Sartorelli D, Lajous M, Balkau B, Clavel-Chapelon F. Consumption of artificially and sugar-sweetened beverages and incident type 2 diabetes in the Etude Epidemiologique auprès des femmes de la Mutuelle Générale de l’Education Nationale–European Prospective Investigation into Cancer and Nutrition cohort. Am J Clin Nutr 2013;97:517–523
32. Malik VS, Popkin BM, Bray GA, Despres JP, Willett WC, Hu FB. Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. Diabetes Care 2010;33:2477–2483
33. Prentice AM, Jebb SA. Obesity in Britain: gluttony or sloth? BMJ 1995;311:437–439
34. Hu FB, Li TY, Colditz GA, Willett WC, Manson JE. Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. JAMA 2003;289:1785–1791
35. Krämer U, Herder C, Sugiri D, et al. Traffic-related air pollution and incident type 2 diabetes: results from the SALIA cohort study. Environ Health Perspect 2010;118:1273–1279
36. Attard SM, Herring AH, Mayer-Davis EJ, Popkin BM, Meigs JB, Gordon-Larsen P. Multilevel examination of diabetes in modernising China: what elements of urbanisation are most associated with diabetes? Diabetologia 2012;55:3182–3192
37. Delisle H, Ntandou-Bouzitou G, Agueh V, Sodjinou R, Fayomi B. Urbanisation, nutrition transition and cardiometabolic risk: the Benin study. Br J Nutr 2012;107:1534–1544
38. Allender SJ, Wickramasinghe K, Goldacre M, Matthews D, Katulanda P. Quantifying urbanization as a risk factor for noncommunicable disease. J Urban Health 2011;88:906–918
39. Assah FK, Ekelund U, Brage S, Mhanya JC, Wareham NJ. Urbanization, physical activity, and metabolic health in sub-Saharan Africa. Diabetes Care 2011;34:491–496
40. Mathenge W, Foster A, Kuper H. Urbanization, ethnicity and cardiovascular risk in a population in transition in Nakuru, Kenya: a population-based survey. BMC Public Health 2010;10:569
41. Elberzards MS, Pamuk ER. The importance of place of residence: examining health in rural and nonrural areas. Am J Public Health 2004;94:1682–1686
42. Krishna S, Gillespie KN, McBride TM. Diabetes burden and access to preventive care in the rural United States. J Rural Health 2010;26:3–11
43. Tai-Seale T. Nutrition and Overweight Concerns in Rural Areas. Rural Healthy People 2010: A Companion Document to Healthy People 2010, Vol. 1. College Station, TX, The Texas A&M University System Health Science Center, School of Rural Public Health, Southwest Rural Health Research Center, 2003, p. 187–198
44. Jackson JE, Doescher MP, Jerant AF, Hart LG. A national study of obesity prevalence and trends by type of rural county. J Rural Health 2005;21:140–148
45. Stewart JE, Battersby SE, Lopez-De Fede A, Remington KC, Hardin JW, Mayfield-Smith K. Diabetes and the socioeconomic and built environment: geovisualization of disease prevalence and potential contextual associations using ring maps. Int J Health Geogr 2011;10:18
46. Casey AA, Elliott M, Glanz K, et al. Impact of the food environment and physical activity environment on behaviors and weight status in rural U.S. communities. Prev Med 2008;47:600–604
47. Kandula NR, Diez-Roux AV, Chan C, et al. Association of acculturation
Scientific Statement

86. Krishnan S, Rosenberg L, Palmer JR. Physical activity and television watching in relation to risk of type 2 diabetes: the Black Women’s Health Study. Am J Epidemiol 2009;169:428–434
87. Katzmarzyk PT, Church TS, Craig CL, Bouchard C. Sitting time and mortality from all causes, cardiovascular disease, and cancer. Med Sci Sports Exerc 2009; 41:998–1005
88. van der Ploeg HP, Chey T, Korda RJ, Banks E, Bauman A. Sitting time and all-cause mortality risk in 222 497 Australian adults. Arch Intern Med 2012;172: 494–500
89. Patel AV, Bernstein L, Deka A, et al. Leisure time spent sitting in relation to total mortality in a prospective cohort of US adults. Am J Epidemiol 2010;172: 419–429
90. Stamatakis E, Hamer M, Dunstan DW. A worksite diabetes prevention program: two-year impact on employee health. AAOHN J 2006;54:389–395
91. Church TS, Thomas DM, Tudor-Locke C, et al. Trends over 5 decades in U.S. occupation-related physical activity and their associations with obesity. PLoS ONE 2011;6:e19657
92. Ford ES. Combined television viewing and computer use and mortality from all-causes and diseases of the circulatory system among adults in the United States. BMC Public Health 2012;12:70
93. Healy GN, Dunstan DW, Salmon J, et al. Breaks in sedentary time: beneficial associations with metabolic risk. Diabetes Care 2008;31:661–666
94. Cooper AR, Sibire S, Montgomery AA, et al. Sedentary time, breaks in sedentary time and metabolic variables in people with newly diagnosed type 2 diabetes. Diabetologia 2012;55:580–590
95. Garber CE, Blissmer B, Deschenes MR, et al.; American College of Sports Medicine. American College of Sports Medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. Med Sci Sports Exerc 2011;43:1334–1359
96. Wijndaele K, Healy GN, Dunstan DW, et al. Increased cardiometabolic risk is associated with increased TV viewing time. Med Sci Sports Exerc 2010;42: 1511–1518
97. Miller R, Brown W. Steps and sitting in a working population. Int J Behav Med 2004;11:219–224
98. Dunstan DW, Howard B, Healy GN, Owen N. Too much sitting—a health hazard. Diabetes Res Clin Pract 2012;97: 368–376
99. Finkel-Poli R, Wolfe R, Backholer K, de Courten M, Peeters A. Impact of a pedometer-based workplace health program on cardiovascular and diabetes risk profile. Prev Med 2011;53:162–171
100. Aldana S, Barlow M, Smith R, et al. Sedentary time, breaks in sedentary time and metabolic variables in people et al. Sedentary time, breaks in sedentary time. Med Sci Sports Exerc 2010;42:1511–1518
101. Patel AV, Bernstein L, Deka A, et al. Leisure time spent sitting in relation to total mortality in a prospective cohort of US adults. Am J Epidemiol 2010;172: 419–429
102. Tandon PS, Zhou C, Sallis JF, Cain KL, Frank LD, Saelens BE. Home environment relationships with children’s physical activity, sedentary time, and screen time by socioeconomic status. Int J Behav Nutr Phys Act 2012;9:88
103. Archer E, Shook RP, Thomas DM, et al. 45-Year trends in women’s use of time and household management energy expenditure. PLoS One 2013;8:e56620
104. Neumark-Sztainer D, Hannan PJ, Story M, Croll J, Perry C. Family meal patterns: associations with sociodemographic characteristics and improved dietary intake among adolescents. J Am Diet Assoc 2009;109(Suppl. 2):S57–S66
105. Bajtc-Bonic J, Tont S, Kacem A, et al. Availability and consumption of competitive foods in US public schools. J Am Diet Assoc 2003;103:317–322
106. Babey SH, Wolstein J, Diamant AL. Food environment nears and school related to consumption of soda and fast food. Policy Brief UCLA Cen Health Policy Res 2011.(PB2011-6):1–8
107. Foster GD, Linder B, Banaworski T, et al.; HEALTHY Study Group. A school-based intervention for diabetes risk reduction. N Engl J Med 2010;363:43–453
108. Trevisol FP, Yin Z, Hernandez A, Hale DE, Garcia OA, Mobley C. Impact of a Bienestar school-based diabetes mellitus prevention program on fasting capillary glucose levels: a randomized controlled trial. Arch Pediatr Adolesc Med 2004; 158:911–917
109. Rosenbaum M, Nomas C, Weir R, et al.; Camino Diabetes Prevention Group. School-based intervention acutely improves insulin sensitivity and decreases inflammatory markers and body fatness in junior high school students. J Clin Endocrinol Metab 2007;92:504–508
110. Lu W, Resnick HE, Jain AK, et al. Effects of isolated post-challenge hyperglycemia on mortality in American Indians: the Strong Heart Study. Ann Epidemiol 2003;13:182–188
111. Meigs JB, Wilson PW, Nathan DM, D’Agostino RB Sr, Williams K, Haffner SM. Prevalence and characteristics of the metabolic syndrome in the San Antonio Heart and Framingham Offspring Studies. Diabetes 2003;52:2160–2167
112. Park S, Barrett-Connor E, Wingard DL, Shan J, Edelstein S. GHb is a better predictor of cardiovascular disease than fasting or postchallenge plasma glucose in women without diabetes. The Rancho Bernardo Study. Diabetes Care 1996;19: 450–456
113. Smith NL, Barzilay JJ, Shaffer D, et al. Fasting and 2-hour postchallenge serum glucose measures and risk of incident cardiovascular events in the elderly: the Cardiovascular Health Study. Arch Intern Med 2002;162:209–216
114. Narayan KM, Boyle JP, Thompson TJ, Sorensen SW, Williamson DF. Lifetime risk for diabetes mellitus in the United States. JAMA 2003;290:1884–1890
115. King H, Aubert RE, Herman WH. Global burden of diabetes, 1995-2025: prevalence, numerical estimates, and projections. Diabetes Care 1998;21:1414–1431
116. Golay A, Ybarra J. Link between obesity and type 2 diabetes. Best Pract Res Clin Endocrinol Metab 2005;19:649–663
117. 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 and Diabetes Study. Diabetes Care 1997;20:537–544
118. Tuomilehto J, Lindstrom J, Eriksson JG, et al.; Finnish Diabetes Prevention Study Group. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. N Engl J Med 2001;344:1343–1350
119. Knowler WC, Barrett-Connor E, Fowler SE, et al.; Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med 2002;346:393–403
120. Ackermann RT, Marrero DG, Hicks KA, et al. An evaluation of cost sharing to finance a diet and physical activity intervention to prevent diabetes. Diabetes Care 2006;29:1237–1241
121. Ackermann RT, Marrero DG. Adapting the Diabetes Prevention Program lifestyle intervention for delivery in the community: the YMCA model. Diabetes Educ 2007;33:69–78
122. Ackermann RT, Anderson E, Brizendine E, Zhou H, Marrero DG. Translating the Diabetes Prevention Study into the Community: The DEPLOY pilot study. Am J Prev Med 2008;35:357–363