

# Scientific Statement: Socioecological Determinants of Prediabetes and Type 2 Diabetes

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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  $\geq 20$  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  $\geq 35$  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).

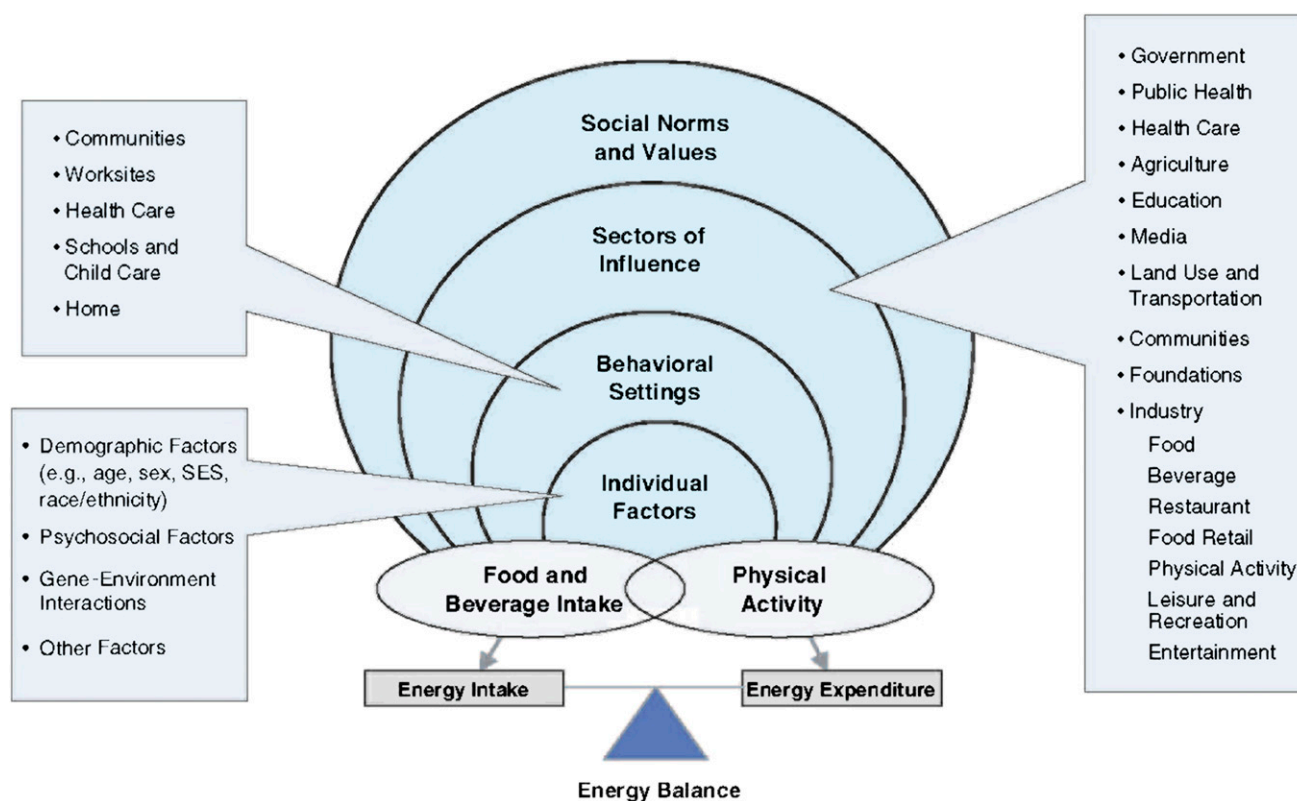
There are also potential ramifications of overweight, obesity, and diabetes

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**Figure 1**—Levels and sectors of influence on obesity and diabetes risk (progress in preventing childhood obesity) (© 2007 the National Academies Press). SES, socioeconomic status.

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.

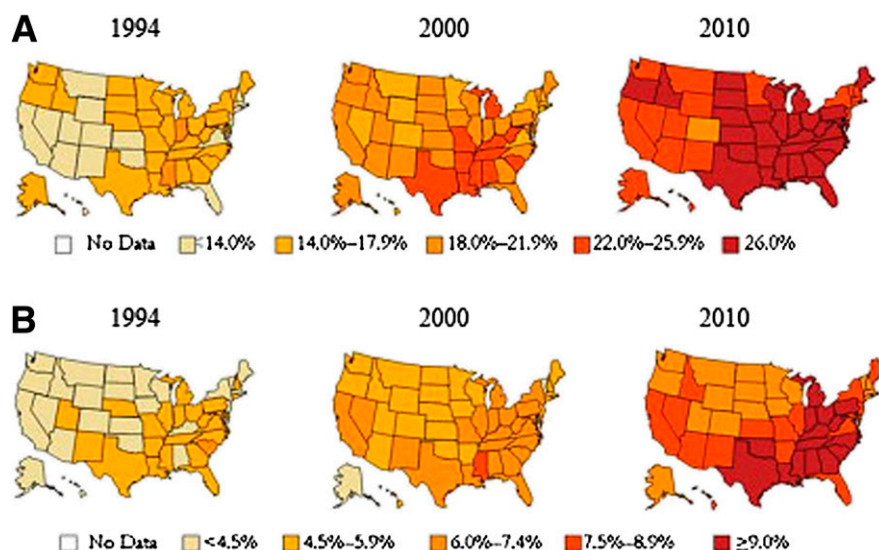
Costs for diagnosed and undiagnosed diabetes are even more staggering (Table 2). In the U.S., the total cost of diagnosed diabetes in 2012 was \$245 billion (15), which includes \$176 billion in direct costs and \$69 billion in reduced productivity. This represents a 41% increase from the last estimate of \$174 billion in 2007. Gestational diabetes mellitus, pre-diabetes, 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

### Age-Adjusted Prevalence of Obesity and Diagnosed Diabetes Among U.S. Adults Aged 18 Years or Older



**Figure 2**—Obesity (A) and diabetes (B) trends among U.S. adults, 1994–2010 (Behavioral Risk Factor Surveillance System [BRFSS]) (ref. 6).

**Table 1—Definitions of overweight and obesity\*****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/m<sup>2</sup>.  
Obesity is defined as a BMI of  $\geq 30$  kg/m<sup>2</sup>.

**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.

risk reduction has focused on the individual, a socioecological perspective recognizes the influence of social and environmental factors on those risks. As noted in a recently released report, U.S. adults have among the highest rates of diabetes and death from ischemic heart disease compared with rates from other high-income countries (18). Factors such as unhealthy behaviors, economic and social conditions, policy and social norms, environmental influences, and inadequate health care are thought to contribute to this risk. This is not a new concept. In 1993, Hurowitz (19) discussed the relative contribution of “socioeconomic factors” to illness and death. Socioecological models have been used to frame prevention strategies in other areas, including injury prevention (20) and obesity (4).

Figure 1, adapted from the Institute of Medicine socioecological model of childhood obesity, provides a good framework to also understand potential

socioecological determinants of diabetes risk.

Due to the broad scope of social and environmental influences on diabetes, we did not encompass all observations in this article but aimed to include overarching research-backed attributes that tied social and environmental factors to diabetes risk. This scientific statement is not intended to recreate the wealth of evidence or rigorously outlined strategies (4) surrounding obesity prevention but to evaluate the extent to which data indicate a contributing role of social and environmental factors to the current diabetes and obesity epidemics.

### **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 other 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 caloric intake increased from 2,450 kcal/day in the 1970s to 2,656 kcal/day in the 2000s in males aged  $\geq 20$  years and increased from 1,542 kcal/day to 1,811 kcal/day in females aged  $\geq 20$  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

**Table 2—Annual per capita health care expenditures in the U.S. by diabetes status, 2012 (in actual dollars)**

Cost component	Unadjusted			Adjusted for age and sex		
	With diabetes (\$)	Without diabetes (\$)	Ratio of cost with diabetes to without diabetes	Without diabetes (\$)	Ratio of cost with diabetes to without diabetes	Attributed to diabetes (\$)
Institutional care	6,907	1,436	4.8	2,839	2.4	4,067
Outpatient care	3,100	1,281	2.4	1,674	1.9	1,428
Outpatient medications and supplies	3,734	778	4.8	1,340	2.8	2,394
Total*	13,741	3,495	3.9	5,853	2.3	7,888

Adapted from ref. 15. Data sources: Nationwide Inpatient Sample (NIS) (2010), National Nursing Home Survey (NNHS) (2004), National Ambulatory Medical Care Survey (NAMCS) (2008–2010), National Hospital Ambulatory Medical Care Survey (NHAMCS) (2007–2009), Medical Expenditure Panel Survey (MEPS) (2006–2010), National Home and Hospice Care Survey (NHHCSS) (2007), National Health Interview Survey (NHIS) (2009–2011), and the U.S. Census Bureau (2012).

\*Numbers do not necessarily sum to totals due to rounding of supporting data.

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 15-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  $\geq 1$  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, cafeterias, 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 increase 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 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 sources. 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  $\geq 20$  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. Babey 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  $\sim 50\%$  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, medication 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  $\geq 65$  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.

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## 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/ddt.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](http://www.cdc.gov/diabetes/statistics/complications_national.htm). Accessed 19 April 2013
  13. Tunceli K, Bradley CJ, Nerenz D, Williams LK, 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, Messner-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#UUKXx6UTHFI>. 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 grain, 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. Salmerón 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, Saes Sartorelli D, Lajous M, Balkau B, Clavel-Chapelon F. Consumption of artificially and sugar-sweetened beverages and incident type 2 diabetes in the Etude Epidémiologique auprès des femmes de la Mutuelle Générale de l'Éducation Nationale–European Prospective Investigation into Cancer and Nutrition cohort. *Am J Clin Nutr* 2013;97:517–523
  32. Malik VS, Popkin BM, Bray GA, Després 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, Mbanya 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. Eberhardt 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



- levels and prevalence of diabetes in the Multi-Ethnic Study of Atherosclerosis (MESA). *Diabetes Care* 2008;31:1621–1628
48. Huang B, Rodriguez BL, Burchfiel CM, Chyou PH, Curb JD, Yano K. Acculturation and prevalence of diabetes among Japanese-American men in Hawaii. *Am J Epidemiol* 1996;144:674–681
  49. Hosler AS, Melnik TA. Prevalence of diagnosed diabetes and related risk factors: Japanese adults in Westchester County, New York. *Am J Public Health* 2003;93:1279–1281
  50. Varghese S, Moore-Orr R. Dietary acculturation and health-related issues of Indian immigrant families in Newfoundland. *Can J Diet Pract Res* 2002;63:72–79
  51. Galea S, Tracy M, Hoggatt KJ, Dimaggio C, Karpati A. Estimated deaths attributable to social factors in the United States. *Am J Public Health* 2011;101:1456–1465
  52. Whiting D. *Diabetes: Equity and Social Determinants. Equity, Social Determinants and Public Health Programmes*. World Health Organization, 2010, p. 77–94
  53. Agency for Healthcare Research and Quality. Statistical Brief #72. Healthcare Cost and Utilization Project (HCUP). February 2011. Agency for Healthcare Research and Quality, Rockville, MD. Available from [www.hcup-us.ahrq.gov/reports/statbriefs/sb72.jsp](http://www.hcup-us.ahrq.gov/reports/statbriefs/sb72.jsp). Accessed 23 April 2013
  54. Ludwig J, Sanbonmatsu L, Gennetian L, et al. Neighborhoods, obesity, and diabetes—a randomized social experiment. *N Engl J Med* 2011;365:1509–1519
  55. Pasala SK, Rao AA, Sridhar GR. Built environment and diabetes. *Int J Diabetes Dev Ctries* 2010;30:63–68
  56. Ewing R, Schmid T, Killingsworth R, Zlot A, Raudenbush S. Relationship between urban sprawl and physical activity, obesity, and morbidity. *Am J Health Promot* 2003;18:47–57
  57. Lopez R. Urban sprawl and risk for being overweight or obese. *Am J Public Health* 2004;94:1574–1579
  58. Lopez-Zetina J, Lee H, Friis R. The link between obesity and the built environment. Evidence from an ecological analysis of obesity and vehicle miles of travel in California. *Health Place* 2006;12:656–664
  59. Frank LD. Multiple pathways from lane use to health: walkability associations with active transportation, body mass index, and air quality. *J Am Plann Assoc* 2006;72:75–87
  60. Gordon-Larsen P, Nelson MC, Page P, Popkin BM. Inequality in the built environment underlies key health disparities in physical activity and obesity. *Pediatrics* 2006;117:417–424
  61. Sallis JF, Glanz K. The role of built environments in physical activity, eating, and obesity in childhood. *Future Child* 2006;16:89–108
  62. Karpyn A, Young C, Weiss S. Reestablishing healthy food retail: changing the landscape of food deserts. *Child Obes* 2012;8:28–30
  63. United States Department of Agriculture. Food deserts. Available from <http://apps.ams.usda.gov/fooddeserts/foodDeserts.aspx>. Accessed 23 April 2013
  64. Gordon C, Purciel-Hill M, Ghai NR, Kaufman L, Graham R, Van Wye G. Measuring food deserts in New York City's low-income neighborhoods. *Health Place* 2011;17:696–700
  65. Larson NI, Story MT, Nelson MC. Neighborhood environments: disparities in access to healthy foods in the U.S. *Am J Prev Med* 2009;36:74–81
  66. Yousefian A, Leighton A, Fox K, Hartley D. Understanding the rural food environment—perspectives of low-income parents. *Rural Remote Health* 2011;11:1631
  67. Rahman T, Cushing RA, Jackson RJ. Contributions of built environment to childhood obesity. *Mt Sinai J Med* 2011;78:49–57
  68. United States Department of Agriculture. *Access to Affordable and Nutritious Food: Measuring and Understanding Food Deserts and Their Consequences*. Report to Congress. USDA Economic Research Service, June 2009
  69. Morton LW, Blanchard TC. *Starved for Access: Life in Rural America's Food Deserts*. Rural Realities Vol. 1, Issue 4. Rural Sociological Society, 2007
  70. Currie J. The effect of fast food restaurants on obesity and weight gain. National Bureau of Economic Research working paper 14721, 2009. Available from <http://www.nber.org/papers/w14721>. Accessed 23 April 2013
  71. Seligman HK, Bindman AB, Vittinghoff E, Kanaya AM, Kushel MB. Food insecurity is associated with diabetes mellitus: results from the National Health Examination and Nutrition Examination Survey (NHANES) 1999–2002. *J Gen Intern Med* 2007;22:1018–1023
  72. Healy GN, Dunstan DW, Salmon J, Shaw JE, Zimmet PZ, Owen N. Television time and continuous metabolic risk in physically active adults. *Med Sci Sports Exerc* 2008;40:639–645
  73. Thorp AA, Healy GN, Winkler E, et al. Prolonged sedentary time and physical activity in workplace and non-work contexts: a cross-sectional study of office, customer service and call centre employees. *Int J Behav Nutr Phys Act* 2012;9:128
  74. Thorp AA, Healy GN, Owen N, et al. Deleterious associations of sitting time and television viewing time with cardiometabolic risk biomarkers: Australian Diabetes, Obesity and Lifestyle (AusDiab) study 2004–2005. *Diabetes Care* 2010;33:327–334
  75. McCrady SK, Levine JA. Sedentariness at work: how much do we really sit? *Obesity (Silver Spring)* 2009;17:2103–2105
  76. Jakes RW, Day NE, Khaw KT, et al. Television viewing and low participation in vigorous recreation are independently associated with obesity and markers of cardiovascular disease risk: EPIC-Norfolk population-based study. *Eur J Clin Nutr* 2003;57:1089–1096
  77. Stamatakis E, Hamer M, Mishra GD. Early adulthood television viewing and cardiometabolic risk profiles in early middle age: results from a population, prospective cohort study. *Diabetologia* 2012;55:311–320
  78. Yates T, Khunti K, Wilmot EG, et al. Self-reported sitting time and markers of inflammation, insulin resistance, and adiposity. *Am J Prev Med* 2012;42:1–7
  79. Pinto Pereira SM, Ki M, Power C. Sedentary behaviour and biomarkers for cardiovascular disease and diabetes in mid-life: the role of television-viewing and sitting at work. *PLoS ONE* 2012;7:e31132
  80. Cameron AJ, Welborn TA, Zimmet PZ, et al. Overweight and obesity in Australia: the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab). *Med J Aust* 2003;178:427–432
  81. Salmon J, Bauman A, Crawford D, Timperio A, Owen N. The association between television viewing and overweight among Australian adults participating in varying levels of leisure-time physical activity. *Int J Obes Relat Metab Disord* 2000;24:600–606
  82. Dunstan DW, Salmon J, Healy GN, et al.; AusDiab Steering Committee. Association of television viewing with fasting and 2-h postchallenge plasma glucose levels in adults without diagnosed diabetes. *Diabetes Care* 2007;30:516–522
  83. Hu FB, Leitzmann MF, Stampfer MJ, Colditz GA, Willett WC, Rimm EB. Physical activity and television watching in relation to risk for type 2 diabetes mellitus in men. *Arch Intern Med* 2001;161:1542–1548
  84. Dunstan DW, Salmon J, Owen N, et al.; AusDiab Steering Committee. Physical activity and television viewing in relation to risk of undiagnosed abnormal glucose metabolism in adults. *Diabetes Care* 2004;27:2603–2609
  85. Ford ES, Schulze MB, Kröger J, Pischon T, Bergmann MM, Boeing H. Television watching and incident diabetes: findings from the European Prospective Investigation into Cancer and Nutrition-Potsdam Study. *J Diabetes* 2010;2:23–27

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. Screen-based entertainment time, all-cause mortality, and cardiovascular events: population-based study with ongoing mortality and hospital events follow-up. *J Am Coll Cardiol* 2011;57:292–299
91. Dunstan DW, Barr EL, Healy GN, et al. Television viewing time and mortality: the Australian Diabetes, Obesity and Lifestyle Study (AusDiab). *Circulation* 2010;121:384–391
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, Sebire S, Montgomery AA, et al. Sedentary time, breaks in sedentary time and metabolic variables in people with newly diagnosed type 2 diabetes. *Diabetologia* 2012;55:589–599
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. Freak-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. A worksite diabetes prevention program: two-year impact on employee health. *AAOHN J* 2006;54:389–395
101. 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
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* 2003;103:317–322
105. Fox MK, Gordon A, Nogales R, Wilson A. Availability and consumption of competitive foods in US public schools. *J Am Diet Assoc* 2009;109(Suppl. 2):S57–S66
106. Babey SH, Wolstein J, Diamant AL. Food environments near home and school related to consumption of soda and fast food. *Policy Brief UCLA Cent Health Policy Res* 2011;(PB2011-6):1–8
107. Foster GD, Linder B, Baranowski T, et al.; HEALTHY Study Group. A school-based intervention for diabetes risk reduction. *N Engl J Med* 2010;363:443–453
108. Treviño RP, Yin Z, Hernandez A, Hale DE, Garcia OA, Mobley C. Impact of the 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, Nonas C, Weil 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 JI, 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, Lindström 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