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Longitudinal Associations Between Neighborhood Physical and Social Environments and Incident Type 2 Diabetes Mellitus: The Multi-Ethnic Study of Atherosclerosis (MESA)

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Abstract

IMPORTANCE—Neighborhood environments may influence the risk for developing type 2 diabetes mellitus (T2DM), but, to our knowledge, no longitudinal study has evaluated specific neighborhood exposures.

OBJECTIVE—To determine whether long-term exposures to neighborhood physical and social environments, including the availability of healthy food and physical activity resources and levels of social cohesion and safety, are associated with incident T2DM during a 10-year period.

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DESIGN, SETTING, AND PARTICIPANTS—We used data from the Multi-Ethnic Study of Atherosclerosis, a population-based cohort study of adults aged 45 to 84 years at baseline (July 17, 2000, through August 29, 2002). A total of 5124 participants free of T2DM at baseline underwent 5 clinical follow-up examinations from July 17, 2000, through February 4, 2012. Time-varying measurements of neighborhood healthy food and physical activity resources and social environments were linked to individual participant addresses. Neighborhood environments were measured using geographic information system (GIS)- and survey-based methods and combined into a summary score. We estimated hazard ratios (HRs) of incident T2DM associated with cumulative exposure to neighborhood resources using Cox proportional hazards regression models adjusted for age, sex, income, educational level, race/ethnicity, alcohol use, and cigarette smoking. Data were analyzed from December 15, 2013, through September 22, 2014.

MAIN OUTCOMES AND MEASURES—Incident T2DM defined as a fasting glucose level of at least 126 mg/dL or use of insulin or oral antihyperglycemics.

RESULTS—During a median follow-up of 8.9 years (37 394 person-years), 616 of 5124 participants (12.0%) developed T2DM (crude incidence rate, 16.47 [95% CI, 15.22-17.83] per 1000 person-years). In adjusted models, a lower risk for developing T2DM was associated with greater cumulative exposure to indicators of neighborhood healthy food (12%; HR per interquartile range [IQR] increase in summary score, 0.88 [95% CI, 0.79-0.98]) and physical activity resources (21%; HR per IQR increase in summary score, 0.79 [95% CI, 0.71-0.88]), with associations driven primarily by the survey exposure measures. Neighborhood social environment was not associated with incident T2DM (HR per IQR increase in summary score, 0.96 [95% CI, 0.88-1.07]).

CONCLUSIONS AND RELEVANCE—Long-term exposure to residential environments with greater resources to support physical activity and, to a lesser extent, healthy diets was associated with a lower incidence of T2DM, although results varied by measurement method. Modifying neighborhood environments may represent a complementary, population-based approach to prevention of T2DM, although further intervention studies are needed.

Type 2 diabetes mellitus (T2DM) is an important cause of death and disability worldwide.¹ Causes of the growing epidemic have been attributed to obesity, specific dietary patterns (eg, diets with a high glycemic load), physical inactivity, and to a lesser extent, smoking, alcohol use, and stress.²⁻⁶ Prevention of T2DM, therefore, has focused largely on behavioral modification.^{3,7-9} However, the extent to which individual behavioral modifications will succeed in unsupportive environments remains unknown.

A growing body of research linking health behaviors¹⁰ and risk factors for chronic disease¹¹⁻¹³ to environmental features has suggested that altering environments may foster behavioral changes.¹⁴ Neighborhood physical environments, including access to healthy food and physical activity (PA) resources, may influence individual diet and exercise levels.^{15,16} Similarly, local social norms and concerns about neighborhood safety might affect behaviors and stress.^{17,18} Modifying environmental resources to support healthy diets, PA, and lower stress levels may therefore aid in prevention of T2DM.

Most prior research linking environmental features to T2DM has been cross-sectional, which limits causal conclusions.^{14,19-21} The few longitudinal studies that exist have been

unable to evaluate long-term neighborhood exposures as they relate to incident T2DM, further limiting causal inference.^{22,23} One randomized study (Moving to Opportunity) that relocated low-income families from high-poverty to low-poverty neighborhoods²⁴ showed that changing neighborhood environments led to a reduced prevalence of obesity and T2DM. However, the study neither answer the equally policy relevant question regarding how the environment where people continually live, rather than residential relocation, influences their risk for developing T2DM, nor did it indicate which neighborhood features may be most important.²⁴ Longitudinal studies that seek to identify the specific components of neighborhoods that influence development of T2DM are thus warranted.

No study, to our knowledge, has evaluated prospectively whether cumulative exposures to specific neighborhood features are related to incident T2DM in a large, multiethnic, geographically distributed sample. To that end, we investigated whether long-term exposures to neighborhood physical and social environments, including the availability of healthy food and PA resources and levels of social cohesion and safety, are associated with the development of T2DM during a 10-year period.

Methods

Study Population and Analytic Sample

Beginning in 2000, the Multi-Ethnic Study of Atherosclerosis (MESA) recruited noninstitutionalized adults (aged 45-84 years) who self-identified as white, black, Hispanic, or Chinese from 6 locations (New York, New York; Baltimore, Maryland; Forsyth County, North Carolina; Chicago, Illinois; St Paul, Minnesota; and Los Angeles, California).²⁵ People with clinical cardiovascular disease were excluded. The first examination took place from July 17, 2000, through August 29, 2002, and 4 follow-up examinations occurred a mean of 1.6, 3.1, 4.8, and 9.5 years later. Retention rates were 92.4% (6239 of 6754 individuals), 89.2% (5946 of 6668 individuals), 86.8% (5704 of 6572 individuals), and 75.7% (4655 of 6149 individuals), respectively. Written informed consent was obtained from the participants, and the study was approved by institutional review boards at each site.

For this analysis of incident T2DM, we used data from the ancillary MESA Neighborhood Study.²⁶ Of the 6814 individuals enrolled at baseline, 6191 agreed to participate in the MESA Neighborhood Study. We excluded individuals with prevalent T2DM at baseline (n = 736) and those with missing exposure, outcome, or covariate data (n = 331), leaving 5124 individuals available for analyses.

Type 2 Diabetes Mellitus

Incident T2DM was determined at each examination according to the 2003 criteria of the American Diabetes Association,²⁷ which included a fasting plasma glucose level of at least 126 mg/dL (to convert to millimoles per liter, multiply by 0.0555) or the use of oral antihyperglycemics or insulin. Glucose levels were measured from blood samples taken after a 12-hour fast as previously described.²⁸ The use of oral antihyperglycemics and insulin was assessed by visual inspection of medications or by self-report on the study questionnaire.

Neighborhood Physical and Social Environments

Neighborhood healthy food and PA resources were assessed in 2 ways using methods consistent with prior studies.^{10,26,29-31} First, we constructed geographic information system (GIS)-based measures of access to food stores more likely to sell healthier foods (supermarkets and fruit and vegetable [FV] markets) and commercial recreational establishments (facilities for indoor conditioning, dance, bowling, golf, team and racquet sports, and water activities) using annual information from the National Establishment Time Series database for the years 2000 through 2012 (Table 1 and eAppendix 1 in the Supplement).³² For simplicity, these measures will be termed *GIS-based supermarkets and FV markets* and *GIS-based commercial recreational establishments*. Simple densities per square mile were created for 1-mile buffers around each participant's residence using software for GIS data (ArcGIS, version 9.3; Esri) (to convert miles to kilometers, multiply by 1.6). Densities were matched to participants annually such that changes over time occurred whenever neighborhood resources changed or a participant moved. One-mile densities were chosen as proxies for neighborhoods based on an area in which most individuals could reasonably walk and on federal government definitions of access to services.³³

As a complementary measure, we also used survey-based measures of neighborhood environments collected in 2003 through 2005 and 2010 through 2012 from MESA participants and an independent, but collocated, sample of non-MESA participants recruited from the same census tracts via random-digit dialing or list-based sampling.²⁶ Respondents were asked to rate the area within a 1-mile or a 20-minute walk of their home with respect to availability of healthy foods and walking environment. Social environment was also assessed using scales for safety and social cohesion (Table 1 and eAppendix 1 in the Supplement). We calculated the mean of survey responses within 1 mile of each participant's residential address, excluding their own responses, to create neighborhood measures and assigned them based on the closest survey time. A median of 78 responses were available within a 1-mile buffer (eTable 1 in the Supplement). All survey scales had good internal consistency (Cronbach α , 0.64-0.83) and ecometric properties (neighborhood reliabilities, 0.38-0.53).²⁶

Because different measures (eg, GIS- and survey-based data for healthy food and PA environments and safety and social cohesion scales for social environment) may reflect different aspects of the same environmental construct, we also calculated summary measures by summing the standardized component measures for healthy food, PA, and social environments (eAppendix 1 in the Supplement). The summary measures had good internal consistency for PA ($\alpha = 0.68$) and social environments ($\alpha = 0.78$) but internal consistency for the healthy food environment was lower ($\alpha = 0.39$). Pearson product moment correlations between the GIS- and survey-based measures were 0.30 for food environment and 0.57 for PA environment.

Covariates

Covariates measured at baseline included age, sex, race/ethnicity, educational level, family history of T2DM, and the presence of chronic stress (>6 months of serious financial, health,

job, or relationship problems). Time-varying information included household income per capita, alcohol consumption (none, moderate, or heavy according to established guidelines),³⁴ and smoking status (current, former, or never). Potential mediators of the association of neighborhood resources and T2DM, including body mass index (BMI) (calculated as weight in kilograms divided by height in meters squared), diet quality, and PA, were assessed via clinical examinations (BMI) and questionnaires (eAppendix 2 in the Supplement). At the neighborhood level, a time-varying socioeconomic index (neighborhood socioeconomic status [SES]) was developed using principal component analysis of census tract data from the US Census³⁵ and American Community Surveys^{36,37} and linked to each participant's address at their closest examination date (eAppendix 3 in the Supplement).

Statistical Analysis

We performed descriptive analyses of individual-level variables by T2DM status and tertiles of the summary neighborhood exposures from December 15, 2013, through September 22, 2014. Crude incidence rates across tertiles of each neighborhood exposure were calculated using Poisson regression. Cox proportional hazards regression models were used to estimate the hazard ratio (HR) of T2DM for each neighborhood exposure separately. Individuals were considered at risk until the diagnosis of T2DM, last follow-up visit, or administrative censoring at examination 5, whichever occurred first. Incident cases of T2DM were assigned to the midpoint between their previous T2DM-free and current examination dates. Because longterm neighborhood exposures are most relevant for slowly developing diseases like T2DM, we parameterized our exposures as time-varying cumulative means, defined as the mean across all months from the baseline to each follow-up examination. Although our outcome is censored by intervals, we elected to use Cox proportional hazards regression models because of our interest in time-varying exposures, which are not easily included in interval-censored models.³⁸ Clustering within census tracts was accounted for by computing robust SEs.

Potential confounders were defined a priori and entered into models in stages. Our primary models adjusted for age, sex, family history of T2DM, per capita household income, educational level, race/ethnicity, smoking status, and alcohol consumption. Additional models were adjusted for neighborhood SES although it is debatable whether SES is a cause or a consequence of some neighborhood exposures (eg, safety).^{39,40} To examine whether BMI, diet, and/or PA mediate the association between neighborhood resources and T2DM, we compared HRs before and after adjustment for these measures.^{41,42}

We evaluated the proportional hazard assumption by plotting Schoenfeld residuals³⁸ against time and found no violations. We found limited evidence of nonlinearity for neighborhood exposures in adjusted Cox proportional hazards regression models, permitting their inclusion as continuous variables. To facilitate comparisons across exposures with different scales, we estimated HRs for an interquartile range (IQR) increase in the neighborhood exposure. These IQRs corresponded to increases of 2.2 supermarkets and/or FV markets and 3.2 commercial recreational establishments for GIS-based exposures, and from 0.3- to 0.7-unit increases for survey-based exposures. To aid replication and comparison with other studies,

we also ran models that parameterized all exposures for 1-unit increases (eTable 2 in the Supplement).

Based on prior findings in the literature, we evaluated effect modification of the summary measures by age at baseline, sex, and household income per capita using interaction terms.^{13,14,23} Because residential environments are hypothesized to be especially salient for individuals with highly stressful lives,⁴³ we also evaluated effect modification by the presence of chronic stress.

We performed several sensitivity analyses. First, we ran interval-censored parametric survival models with a Weibull distribution³⁸ to assess sensitivity to our modeling approach. We also explored alternative exposure specifications using different geographic (3-mile buffer for GIS measures and census tracts for survey measures) and time (1-year lagged exposures for GIS measures; survey measures unavailable annually) scales. Because population density and regional norms may affect health behaviors independently of neighborhood resources,^{29,44} we ran additional models controlling for population density and study site. To help control for unmeasured confounding at the neighborhood level, we ran shared frailty models with random intercepts for each census tract (eTable 3 in the Supplement).^{45,46} Finally, although long-term neighborhood exposures are likely most relevant for T2DM risk, we examined baseline and change since baseline exposure measures to evaluate how these parameterizations were related to T2DM risk (eAppendix 4 in the Supplement provides details).

Results

During a median of 8.9 years (37 394 person-years), 616 of 5124 participants (12.0%) developed T2DM (crude incidence rate, 16.47 [95% CI, 15.22-17.83] per 1000 person-years). Compared with participants who did not develop T2DM, incident cases were more likely to be black or Hispanic and to have lower baseline household income, fewer years of education, less healthy diets, lower levels of moderate and vigorous PA, a higher BMI, and a family history of T2DM (Table 2). Participants developing T2DM also lived in poorer census tracts.

Neighborhood physical and social resources were highly patterned by race, diet, PA levels, BMI, and neighborhood SES, such that racial or ethnic minorities and those with greater risk factor profiles were generally more likely to reside in neighborhoods with fewer resources (Table 3). Temporal changes in neighborhood exposures varied by exposure type, ranging from mean 10-year changes of 2.01 for GIS-based commercial recreational establishments to -0.20 for GIS-based supermarkets and FV markets (eTable 4 in the Supplement). At baseline, the median duration of neighborhood residence was 15 years, and 1642 individuals (32.0%) moved during follow-up.

Higher baseline summary measures of neighborhood PA, social environment, and to a lesser extent, healthy food resources were associated with lower crude rates of T2DM (Table 4). For instance, participants residing in neighborhoods in the bottom tertile of summary PA environment developed T2DM at nearly double the rate of those living in neighborhoods in

the top tertile (incidence rates, 20.5 and 11.8 per 1000 person-years, respectively). The GIS-based supermarkets and FV markets and social cohesion exposures were not related to T2DM incidence rates.

After adjustment for baseline age, sex, income, educational level, race/ethnicity, alcohol use, and smoking status (model 1), an IQR increase in cumulative exposure to survey-based healthy food resources was associated with a 16% lower risk for T2DM (HR, 0.84 [95% CI, 0.76-0.93]), but no association was found using the GIS-based measure (HR, 0.99 [95% CI, 0.94-1.04]) (Figure). An IQR increase in the summary healthy food environment measure was associated with a 12% lower risk for developing T2DM (HR, 0.88 [95% CI, 0.79-0.98]). Further adjustment for neighborhood SES (model 2) attenuated the associations (Figure). For PA environments, greater cumulative exposure to neighborhoods with resources supporting PA was inversely associated with T2DM incidence; IQR increases in GIS-based, survey-based, and summary environmental measures were associated with 4% (HR, 0.96 [95% CI, 0.92-0.99]), 21% (HR, 0.79 [95% CI, 0.71-0.88]), and 21% (HR, 0.79 [95% CI, 0.69-0.90]) lower risk for T2DM, respectively. Adjusting for neighborhood SES attenuated the GIS-based association but left the other associations virtually unchanged. Social cohesion, safety, and the summary measure for social environment were largely unassociated with the risk for T2DM (HR per IQR increase [95% CI]: 0.99 [0.88-1.10]; 0.92 [0.80-1.05]; and 0.96 [0.86-1.07], respectively). Further adjustment of models for BMI, diet, and PA as potential mediators demonstrated minimal attenuation of most associations (25%; eTable 5 in the Supplement).

Neighborhood healthy food resources had a stronger inverse association with T2DM among participants who were younger, had higher incomes, and reported a chronic stress burden ($P = .06$ for multiplicative and additive interaction; eFigure in the Supplement). Similarly, the inverse association between neighborhood PA resources and T2DM was stronger in participants with higher incomes ($P = .07$ and $P = .04$ for multiplicative and additive interaction, respectively). Neighborhood social environment was inversely associated with T2DM in women but not men and in low-income but not high-income participants ($P = .07$ for multiplicative and additive interaction).

Sensitivity analyses demonstrated qualitatively similar findings when using interval-censored survival methods, different exposure specifications, controls for population density and study site, shared frailty models, and adjustment for baseline risk factors for T2DM (eTables 3, 6, and 7 in the Supplement). Alternative modeling strategies showed that baseline and change in neighborhood exposure levels were associated with incident T2DM in the expected (inverse) direction for survey-based measures although results were imprecise (eTable 8 in the Supplement). Baseline levels, but not change, were associated with T2DM for GIS-based commercial recreational establishments.

Discussion

In this large multiethnic cohort, long-term exposure to residential environments with greater resources to support PA and to a lesser extent healthy diets was associated with a lower incidence of T2DM during the 10-year study. The associations were generally robust to

adjustment for other risk factors and model specifications although associations were primarily found with survey-based, but not GIS-based, exposures. Inclusion of BMI, diet, and PA as hypothesized mediators only modestly attenuated the relationships. Neighborhood safety and social cohesion were largely unassociated with the development of T2DM.

Unlike previous studies of residential environments and T2DM,^{19,24} we measured specific, time-varying features of participants' neighborhoods using complementary measures. Geographic proximity to commercial recreational establishments and greater survey-based assessments of the walking environment were inversely associated with T2DM incidence. Previous work using the MESA cohort has demonstrated that an increase in commercial PA resources is associated with less age-related decline in PA.⁴⁸ Other studies have found that residential relocation to neighborhoods more supportive of PA is associated with increased levels of PA, independently of reasons for relocation.^{49,50} Our study suggests that such neighborhood associations with PA behavior may translate to reduced risk for T2DM.

We found that geographic proximity to supermarkets and FV markets had no association with T2DM incidence. This finding is consistent with recent observational and quasi-experimental evidence demonstrating that simply improving retail food infrastructure may not translate into healthier diets and decreased risk for chronic diseases.⁵¹⁻⁵³ On the other hand, survey-based measures of the local food environment were associated with T2DM, suggesting that such measures may take into account other factors like the affordability and quality of food that are known to influence diet and T2DM risk.⁵⁴⁻⁵⁶

Finally, although social features of residential environments have been hypothesized to be related to obesity and T2DM through their association with health behaviors and stress,^{17,18} we find limited support for these relationships. Additional research with alternative exposure measures is needed to further clarify the role of the social environment.

Although the use of multiple modalities for measuring neighborhood environments is a strength in our study, the difference in the associations for GIS- and survey-based measures of the food and PA environments are noteworthy. The most likely explanation for the discrepancies is that the GIS counts and survey responses measure different aspects of the same construct.¹⁰ For instance, our survey-based PA exposure assesses noncommercial neighborhood features related to walkability and aesthetics not captured in the GIS-based measures. Neighborhood residents also likely consider unmeasured attributes such as cost or quality that are not captured with simple counts from tax parcel data.⁵⁷ Differences between the GIS- and survey-based associations also could be the result of reverse causation if individuals with less interest in healthy food or PA resources are less likely to perceive that such resources are available. We think this reverse causation is unlikely for the following 2 reasons: the neighborhood survey assesses community ratings of the local environment (with a median of 78 residents in an area of 1 square mile from whom mean responses were calculated), and we excluded an individual's survey response from their own exposure measure. Nonetheless, future research would benefit from including multiple measures of the same neighborhood environmental constructs to further understand the most relevant features for T2DM risk.

We observed differences in the associations between neighborhood features and T2DM according to individual characteristics, although given the multiple comparisons assessed, caution should be exercised in interpreting the results. Household income appeared to be a consistent effect modifier such that increased healthy food and PA resources were more beneficial to high-income households than to low-income households. For low-income households, growing evidence suggests factors like cost may trump geographic proximity to healthy food and PA resources.^{58,59} The social environment demonstrated the opposite pattern, whereby increasing safety and social cohesion was associated with lower T2DM risk in low-income but not high-income households. Community safety and social relationships have been associated with BMI and PA in several studies,⁶⁰⁻⁶³ but further work is needed to understand if and why such associations may differ by income. The presence of chronic stressors also modified the association for healthy food environments such that increasing healthy food resources were associated with lower T2DM risk for those with chronic stressors. We are unaware of other studies evaluating this question although our findings are consistent with those in the literature that suggest environmental resources may be especially salient for individuals experiencing chronic stress.⁴³

Models adjusting for BMI as a mediator modestly attenuated the associations between residential healthy food and PA environments and T2DM incidence. Such modest attenuation is not surprising given the long-term nature of T2DM development⁶⁴ and the difficulty in separating direct and indirect effects in standard regression analyses.^{65,66} Diet and PA are also notoriously difficult to measure precisely, and measurement error can distort the magnitude of mediation observed.⁶⁷ Further work focusing specifically on mediation is warranted to quantify the behavioral and biological pathways through which features of the neighborhood environment may influence the risk for T2DM.

The primary strength of our study is the longitudinal measurement of specific features of neighborhood environments and T2DM status over time in a multiethnic sample. Given that T2DM develops during a protracted period, such long-term exposure measures are more relevant than simple cross-sectional exposures. Furthermore, using multiple measures for specific environmental features has several advantages. First, such measures can be used to evaluate which features may be most critical for mitigating T2DM risk, rather than focusing solely on neighborhood SES, which may be a proxy for many interrelated neighborhood features.⁶⁸ Second, specific measures of neighborhood environments may be less susceptible to problems of endogeneity or reverse causation, wherein the characteristics of a neighborhood environment are simply the result of the individual attributes and preferences of residents.⁶⁸ Finally, prospective collection of covariate information allowed for updating of confounding variables.

The study also has several limitations. As with all observational studies of neighborhood exposures, residential self-selection, wherein individuals with certain risk profiles select to live in certain neighborhoods, may bias the associations reported.⁶⁹ Although we attempted to minimize such bias by including individual-level variables related to neighborhood selection,⁷⁰ unobserved or mismeasured characteristics may influence neighborhood exposure and the risk for T2DM. Further use of experimental, quasi-experimental, and observational data with different methods may help to increase our confidence in the

associations observed. Other exposures, such as neighborhood traffic safety and availability of green spaces or those encountered near work or during a commute (eg, food stores), may also be relevant to T2DM risk.^{14,71,72} Finally, 1494 of 6149 eligible MESA participants (24.3%) were lost to or unavailable for follow up by examination 5, raising the possibility of bias owing to informative censoring. Dropout was not highly patterned by neighborhood exposures, however, making this bias less likely.

Conclusions

The prevalence of T2DM continues to increase in the United States despite its preventability through behavioral modifications.^{7,9} Although individualized prevention and treatment approaches are necessary to decrease the burden of T2DM, environmental modifications that promote healthy behaviors represent a complementary, perhaps prerequisite, population health approach. Our results suggest that modifying specific features of neighborhood environments, including increasing the availability of healthy foods and PA resources, may help to mitigate the risk for T2DM although additional intervention studies with measures of multiple neighborhood features are needed. Such approaches may be especially important for addressing disparities in T2DM given the concentration of low-income and minority populations in neighborhoods with fewer health-promoting resources.⁷³⁻⁷⁵

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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REFERENCES

1. Danaei G, Finucane MM, Lu Y, et al. Global Burden of Metabolic Risk Factors of Chronic Diseases Collaborating Group (Blood Glucose). National, regional, and global trends in fasting plasma glucose and diabetes prevalence since 1980: systematic analysis of health examination surveys and epidemiological studies with 370 country-years and 2.7 million participants. *Lancet*. 2011; 378(9785):31–40. [PubMed: 21705069]
2. Hu FB, Manson JE, Stampfer MJ, et al. Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. *N Engl J Med*. 2001; 345(11):790–797. [PubMed: 11556298]
3. Mozaffarian D, Kamineni A, Carnethon M, Djoussé L, Mukamal KJ, Siscovick D. Lifestyle risk factors and new-onset diabetes mellitus in older adults: the Cardiovascular Health Study. *Arch Intern Med*. 2009; 169(8):798–807. [PubMed: 19398692]
4. Hu FB. Globalization of diabetes: the role of diet, lifestyle, and genes. *Diabetes Care*. 2011; 34(6): 1249–1257. [PubMed: 21617109]
5. Mokdad AH, Bowman BA, Ford ES, Vinicor F, Marks JS, Koplan JP. The continuing epidemics of obesity and diabetes in the United States. *JAMA*. 2001; 286(10):1195–1200. [PubMed: 11559264]

6. Schulze MB, Hu FB. Primary prevention of diabetes: what can be done and how much can be prevented? *Annu Rev Public Health*. 2005; 26:445–467. [PubMed: 15760297]
7. 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(6):393–403. [PubMed: 11832527]
8. 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(18):1343–1350. [PubMed: 11333990]
9. Danaei G, Pan A, Hu FB, Hernán MA. Hypothetical midlife interventions in women and risk of type 2 diabetes. *Epidemiology*. 2013; 24(1):122–128. [PubMed: 23222556]
10. Moore LV, Diez Roux AV, Nettleton JA, Jacobs DR Jr. Associations of the local food environment with diet quality: a comparison of assessments based on surveys and geographic information systems: the Multi-Ethnic Study of Atherosclerosis. *Am J Epidemiol*. 2008; 167(8): 917–924. [PubMed: 18304960]
11. Auchincloss AH, Mujahid MS, Shen M, Michos ED, Whitt-Glover MC, Diez Roux AV. Neighborhood health-promoting resources and obesity risk (the Multi-Ethnic Study of Atherosclerosis). *Obesity (Silver Spring)*. 2013; 21(3):621–628. [PubMed: 23592671]
12. Mujahid MS, Diez Roux AV, Morenoff JD, et al. Neighborhood characteristics and hypertension. *Epidemiology*. 2008; 19(4):590–598. [PubMed: 18480733]
13. Auchincloss AH, Diez Roux AV, Brown DG, Erdmann CA, Bertoni AG. Neighborhood resources for physical activity and healthy foods and their association with insulin resistance. *Epidemiology*. 2008; 19(1):146–157. [PubMed: 18091002]
14. Sallis JF, Floyd MF, Rodríguez DA, Saelens BE. Role of built environments in physical activity, obesity, and cardiovascular disease. *Circulation*. 2012; 125(5):729–737. [PubMed: 22311885]
15. Papas MA, Alberg AJ, Ewing R, Helzlouer KJ, Gary TL, Klassen AC. The built environment and obesity. *Epidemiol Rev*. 2007; 29:129–143. [PubMed: 17533172]
16. Pearson TA, Palaniappan LP, Artinian NT, et al. American Heart Association Council on Epidemiology and Prevention. American Heart Association Guide for Improving Cardiovascular Health at the Community Level, 2013 update: a scientific statement for public health practitioners, healthcare providers, and health policy makers. *Circulation*. 2013; 127(16):1730–1753. [PubMed: 23519758]
17. Fowler-Brown AG, Bennett GG, Goodman MS, Wee CC, Corbie-Smith GM, James SA. Psychosocial stress and 13-year BMI change among blacks: the Pitt County Study. *Obesity (Silver Spring)*. 2009; 17(11):2106–2109. [PubMed: 19407807]
18. Morenoff, JD.; Diez Roux, AV.; Hansen, BB.; Osypuk, TL. Residential environments and obesity: what can we learn about policy interventions from observational studies?. In: Schoeni, RF.; House, JS.; Kaplan, GA.; Pollack, H., editors. *Making Americans Healthier: Social and Economic Policy as Health Policy*. Russell Sage Foundation; New York, NY: 2008. p. 309-343.
19. Müller G, Kluttig A, Greiser KH, et al. Regional and neighborhood disparities in the odds of type 2 diabetes: results from 5 population-based studies in Germany (DIAB-CORE consortium). *Am J Epidemiol*. 2013; 178(2):221–230. [PubMed: 23648804]
20. Andersen AF, Carson C, Watt HC, Lawlor DA, Avlund K, Ebrahim S. Life-course socio-economic position, area deprivation and type 2 diabetes: findings from the British Women's Heart and Health Study. *Diabet Med*. 2008; 25(12):1462–1468. [PubMed: 19046246]
21. Leal C, Chaix B. The influence of geographic life environments on cardiometabolic risk factors: a systematic review, a methodological assessment and a research agenda. *Obes Rev*. 2011; 12(3): 217–230. [PubMed: 20202135]
22. Schootman M, Andresen EM, Wolinsky FD, et al. The effect of adverse housing and neighborhood conditions on the development of diabetes mellitus among middle-aged African Americans. *Am J Epidemiol*. 2007; 166(4):379–387. [PubMed: 17625220]
23. Auchincloss AH, Diez Roux AV, Mujahid MS, Shen M, Bertoni AG, Carnethon MR. Neighborhood resources for physical activity and healthy foods and incidence of type 2 diabetes mellitus: the Multi-Ethnic Study of Atherosclerosis. *Arch Intern Med*. 2009; 169(18):1698–1704. [PubMed: 19822827]

24. Ludwig J, Sanbonmatsu L, Gennetian L, et al. Neighborhoods, obesity, and diabetes: a randomized social experiment. *N Engl J Med*. 2011; 365(16):1509–1519. [PubMed: 22010917]
25. Bild DE, Bluemke DA, Burke GL, et al. Multi-Ethnic Study of Atherosclerosis: objectives and design. *Am J Epidemiol*. 2002; 156(9):871–881. [PubMed: 12397006]
26. Mujahid MS, Diez Roux AV, Morenoff JD, Raghunathan T. Assessing the measurement properties of neighborhood scales: from psychometrics to ecometrics. *Am J Epidemiol*. 2007; 165(8):858–867. [PubMed: 17329713]
27. Genuth S, Alberti KG, Bennett P, et al. Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Follow-up report on the diagnosis of diabetes mellitus. *Diabetes Care*. 2003; 26(11):3160–3167. [PubMed: 14578255]
28. Bertoni AG, Burke GL, Owusu JA, et al. Inflammation and the incidence of type 2 diabetes: the Multi-Ethnic Study of Atherosclerosis (MESA). *Diabetes Care*. 2010; 33(4):804–810. [PubMed: 20097779]
29. Boone-Heinonen J, Diez-Roux AV, Goff DC, et al. The neighborhood energy balance equation: does neighborhood food retail environment + physical activity environment = obesity? the CARDIA Study. *PLoS One*. 2013; 8(12):e85141. [PubMed: 24386458]
30. Auchincloss AH, Moore KA, Moore LV, Diez Roux AV. Improving retrospective characterization of the food environment for a large region in the United States during a historic time period. *Health Place*. 2012; 18(6):1341–1347. [PubMed: 22883050]
31. Moore LV, Diez Roux AV. Associations of neighborhood characteristics with the location and type of food stores. *Am J Public Health*. 2006; 96(2):325–331. [PubMed: 16380567]
32. Walls & Associates. [Accessed July 23, 2013] National Establishment Time-Series (NETS) Database: Database Description. 2012. <http://exceptionalgrowth.org/downloads/NETSDatabaseDescription2013.pdf>
33. Ver PLoeg, M.; Bereneman, V.; Farrigan, T., et al. Access to Affordable and Nutritious Food: Measuring and Understanding Food Deserts and Their Consequences: Report to Congress. United States Department of Agriculture; Washington, DC: 2009.
34. Allen, JP.; Wilson, V., editors. Assessing Alcohol Problems: A Guide for Clinicians and Researchers. 2nd ed.. National Institute on Alcohol Abuse and Alcoholism; Bethesda, MD: 2003.
35. US Census Bureau. [Accessed July 10, 2012] Census 2000 Summary File 1 & Summary File 3 - United States. 2001. <https://www.census.gov/main/www/cen2000.html>
36. US Census Bureau. [Accessed July 10, 2012] 2005-2009 American Community Survey—United States. 2011. http://www.census.gov/acs/www/data_documentation/2009_release/
37. US Census Bureau. [Accessed July 10, 2012] 2007-2011 American Community Survey - United States. 2013. https://www.census.gov/newsroom/releases/archives/news_conferences/20121203_acs5yr.html
38. Allison, PD. Survival Analysis Using SAS: A Practical Guide. SAS Institute; Cary, NC: 2010.
39. Greenbaum RT, Tita GE. The impact of violence surges on neighbourhood business activity. *Urban Stud*. 2004; 41(13):2495–2514.
40. Chaix B, Leal C, Evans D. Neighborhood-level confounding in epidemiologic studies: unavoidable challenges, uncertain solutions. *Epidemiology*. 2010; 21(1):124–127. [PubMed: 19907336]
41. Hafeman DM, Schwartz S. Opening the black box: a motivation for the assessment of mediation. *Int J Epidemiol*. 2009; 38(3):838–845. [PubMed: 19261660]
42. Naimi AI, Kaufman JS, Howe CJ, Robinson WR. Mediation considerations: serum potassium and the racial disparity in diabetes risk. *Am J Clin Nutr*. 2011; 94(2):614–616. [PubMed: 21775571]
43. Mezuk B, Abdou CM, Hudson D, et al. “White box” epidemiology and the social neuroscience of health behaviors: the environmental affordances model. *Soc Ment Health*. 2013; 3(2) doi: 10.1177/2156869313480892.
44. Boone-Heinonen J, Evenson KR, Song Y, Gordon-Larsen P. Built and socioeconomic environments: patterning and associations with physical activity in U.S. adolescents. *Int J Behav Nutr Phys Act*. 2010; 7:45. doi:10.1186/1479-5868-7-45. [PubMed: 20487564]
45. Hougaard, P. Shared Frailty Models: Analysis of Multivariate Survival Data. Springer; New York, NY: 2000. p. 215-262.

46. Wienke, A. *Frailty Models in Survival Analysis*. Taylor & Francis; New York, NY: 2010.
47. Chiuve SE, Fung TT, Rimm EB, et al. Alternative dietary indices both strongly predict risk of chronic disease. *J Nutr*. 2012; 142(6):1009–1018. [PubMed: 22513989]
48. Ranchod YK, Diez Roux AV, Evenson KR, Sánchez BN, Moore K. Longitudinal associations between neighborhood recreational facilities and change in recreational physical activity in the Multi-Ethnic Study of Atherosclerosis, 2000-2007. *Am J Epidemiol*. 2014; 179(3):335–343. [PubMed: 24227016]
49. Giles-Corti B, Bull F, Knuiaman M, et al. The influence of urban design on neighbourhood walking following residential relocation: longitudinal results from the RESIDE Study. *Soc Sci Med*. 2013; 77:20–30. [PubMed: 23206559]
50. Calise TV, Heeren T, DeJong W, Dumith SC, Kohl HW III. Do neighborhoods make people active, or do people make active neighborhoods? evidence from a planned community in Austin, Texas. *Prev Chronic Dis*. 2013; 10:E102. [PubMed: 23786909]
51. Boone-Heinonen J, Gordon-Larsen P, Kiefe CI, Shikany JM, Lewis CE, Popkin BM. Fast food restaurants and food stores: longitudinal associations with diet in young to middle-aged adults: the CARDIA Study. *Arch Intern Med*. 2011; 171(13):1162–1170. [PubMed: 21747011]
52. Cummins S, Flint E, Matthews SA. New neighborhood grocery store increased awareness of food access but did not alter dietary habits or obesity. *Health Aff (Millwood)*. 2014; 33(2):283–291. [PubMed: 24493772]
53. Lucan SC, Hillier A, Schechter CB, Glanz K. Objective and self-reported factors associated with food-environment perceptions and fruit-and-vegetable consumption: a multilevel analysis. *Prev Chronic Dis*. 2014; 11:E47. [PubMed: 24674635]
54. Caspi CE, Kawachi I, Subramanian SV, Adamkiewicz G, Sorensen G. The relationship between diet and perceived and objective access to supermarkets among low-income housing residents. *Soc Sci Med*. 2012; 75(7):1254–1262. [PubMed: 22727742]
55. Anekwe TD, Rahkovsky I. The association between food prices and the blood glucose level of US adults with type 2 diabetes. *Am J Public Health*. 2014; 104(4):678–685. [PubMed: 24524504]
56. Beydoun MA, Powell LM, Wang Y. The association of fast food, fruit and vegetable prices with dietary intakes among US adults: is there modification by family income? *Soc Sci Med*. 2008; 66(11):2218–2229. [PubMed: 18313824]
57. Brownson RC, Hoehner CM, Day K, Forsyth A, Sallis JF. Measuring the built environment for physical activity: state of the science. *Am J Prev Med*. 2009; 36(4)(suppl):S99–123.e12. 123.e112. doi:10.1016/j.amepre.2009.01.005. [PubMed: 19285216]
58. Drewnowski A, Aggarwal A, Hurvitz PM, Monsivais P, Moudon AV. Obesity and supermarket access: proximity or price? *Am J Public Health*. 2012; 102(8):e74–e80. doi:10.2105/AJPH.2012.300660. [PubMed: 22698052]
59. Breyer B, Voss-Andreae A. Food mirages: geographic and economic barriers to healthful food access in Portland, Oregon. *Health Place*. 2013; 24:131–139. [PubMed: 24100236]
60. Burdette HL, Wadden TA, Whitaker RC. Neighborhood safety, collective efficacy, and obesity in women with young children. *Obesity (Silver Spring)*. 2006; 14(3):518–525. [PubMed: 16648624]
61. Mason P, Kearns A, Livingston M. “Safe going”: the influence of crime rates and perceived crime and safety on walking in deprived neighbourhoods. *Soc Sci Med*. 2013; 91:15–24. [PubMed: 23849234]
62. Bennett GG, McNeill LH, Wolin KY, Duncan DT, Puleo E, Emmons KM. Safe to walk? neighborhood safety and physical activity among public housing residents. *PLoS Med*. 2007; 4(10):1599–1606. [PubMed: 17958465]
63. Fish JS, Ettner S, Ang A, Brown AF. Association of perceived neighborhood safety with [corrected] body mass index. *Am J Public Health*. 2010; 100(11):2296–2303. [PubMed: 20864717]
64. Reis JP, Hankinson AL, Loria CM, et al. Duration of abdominal obesity beginning in young adulthood and incident diabetes through middle age: the CARDIA Study. *Diabetes Care*. 2013; 36(5):1241–1247. [PubMed: 23248193]
65. Cole SR, Hernán MA. Fallibility in estimating direct effects. *Int J Epidemiol*. 2002; 31(1):163–165. [PubMed: 11914314]

66. Blakely T. Commentary: estimating direct and indirect effects—fallible in theory, but in the real world? *Int J Epidemiol.* 2002; 31(1):166–167. [PubMed: 11914315]
67. le Cessie S, Debeij J, Rosendaal FR, Cannegieter SC, Vandenbroucke JP. Quantification of bias in direct effects estimates due to different types of measurement error in the mediator. *Epidemiology.* 2012; 23(4):551–560. [PubMed: 22526092]
68. Diez Roux AV. Estimating neighborhood health effects: the challenges of causal inference in a complex world. *Soc Sci Med.* 2004; 58(10):1953–1960. [PubMed: 15020010]
69. Oakes JM. The (mis)estimation of neighborhood effects: causal inference for a practicable social epidemiology. *Soc Sci Med.* 2004; 58(10):1929–1952. [PubMed: 15020009]
70. Sampson RJ, Sharkey P. Neighborhood selection and the social reproduction of concentrated racial inequality. *Demography.* 2008; 45(1):1–29. [PubMed: 18390289]
71. Cummins S. Commentary: investigating neighbourhood effects on health—avoiding the “local trap.”. *Int J Epidemiol.* 2007; 36(2):355–357. [PubMed: 17376797]
72. Macintyre S. Deprivation amplification revisited: or, is it always true that poorer places have poorer access to resources for healthy diets and physical activity? *Int J Behav Nutr Phys Act.* 2007; 4:32. doi:10.1186/1479-5868-4-32. [PubMed: 17683624]
73. Franco M, Diez Roux AV, Glass TA, Caballero B, Brancati FL. Neighborhood characteristics and availability of healthy foods in Baltimore. *Am J Prev Med.* 2008; 35(6):561–567. [PubMed: 18842389]
74. Duncan DT, Kawachi I, White K, Williams DR. The geography of recreational open space: influence of neighborhood racial composition and neighborhood poverty. *J Urban Health.* 2013; 90(4):618–631. [PubMed: 23099625]
75. Estabrooks PA, Lee RE, Gyurcsik NC. Resources for physical activity participation: does availability and accessibility differ by neighborhood socioeconomic status? *Ann Behav Med.* 2003; 25(2):100–104. [PubMed: 12704011]

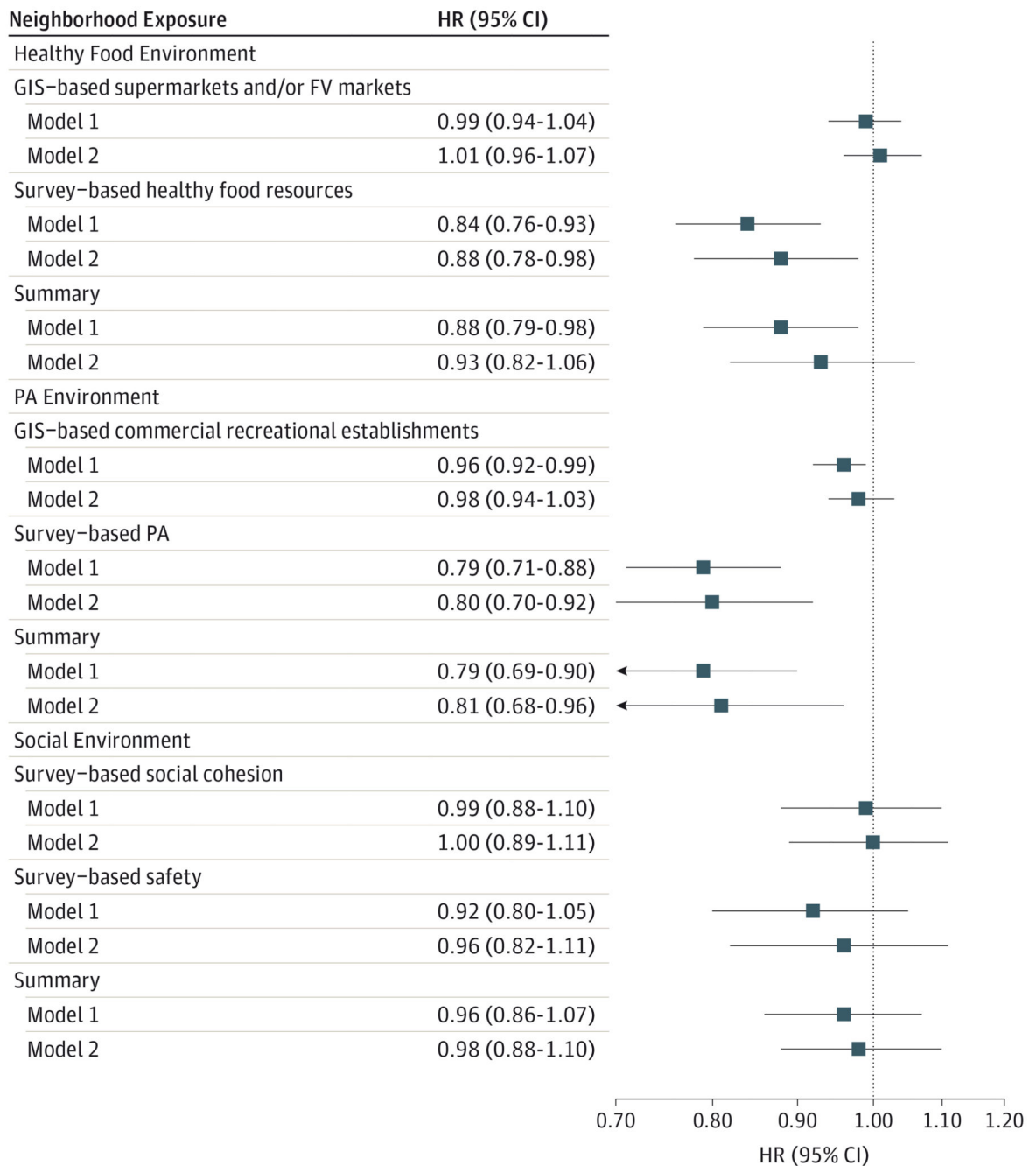


Figure. Adjusted Hazard Ratios (HRs) for Type 2 Diabetes Mellitus (T2DM) Incidence Corresponding to an Interquartile Range (IQR) Increase in Exposure to Neighborhood Resources, 2000 to 2012

Model 1 adjusts for baseline age, sex, family history of T2DM, household per capita income, educational level, smoking status, and alcohol use. Model 2 adjusts for all covariates in model 1 and adds neighborhood socioeconomic status. All exposures correspond to cumulative mean exposures over time. An IQR increase in exposure corresponds to the following changes for each exposure: 2.2 for geographic information system (GIS)-based supermarkets and/or fruit and vegetable (FV) markets; 0.6 for survey-

based healthy food resources; 2.1 for combined healthy food resources; 3.2 for GIS-based commercial recreational establishments; 0.4 for survey-based physical activity (PA); 1.2 for combined PA; 0.3 for social cohesion; 0.7 for safety; and 2.0 for combined social environment.

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Table 1
Neighborhood Measures for Healthy Food and PA Resources and Social Environments^a

Neighborhood Summary and Component Measures	Scale^b
Healthy food environment summary score	Sum of standardized component measures
GIS-based density of supermarkets and/or FV markets	No. of food stores likely to sell healthier foods (supermarkets, FV markets) per square mile
Survey-based healthy food availability	Likert scale, 1-5 (eg, "A large selection of fresh fruits and vegetables is available in my neighborhood.")
PA environment summary score	Sum of standardized component measures
GIS-based density of commercial recreational establishments	No. of commercial recreational establishments (gyms, pools, etc) per square mile
Survey-based walking environment	Likert scale, 1-5 (eg, "My neighborhood offers many opportunities to be physically active.")
Social environment summary score	Sum of standardized component measures
Survey-based social cohesion	Likert scale, 1-5 (eg, "People in my neighborhood can be trusted.")
Survey-based safety	Likert scale, 1-5 (eg, "I feel safe walking in my neighborhood, day or night.")

Abbreviations: FV, fruit and vegetable; GIS, geographic information system; PA, physical activity; T2DM, type 2 diabetes mellitus.

^aData are from the Neighborhood Multi-Ethnic Study of Atherosclerosis, 2000 to 2012.²⁵

^bAll measures are constructed such that higher values indicate more favorable environments.

Table 2
Baseline Sociodemographic, Behavioral, and T2DM Risk Factor Characteristics^a

Characteristic	Total Sample (N = 5124)	T2DM Developed During Follow-up	
		Yes (n = 616)	No (n = 4508)
Sociodemographic			
Age, mean (SD), y	60.7 (9.9)	60.9 (9.6)	60.7 (9.9)
Female, No. (%)	2747 (53.6)	325 (52.8)	2422 (53.7)
Race/ethnicity, No. (%) ^b			
White	2168 (42.3)	190 (30.8)	1978 (43.9)
Black	1311 (25.6)	190 (30.8)	1121 (24.9)
Hispanic	1041 (20.3)	161 (26.1)	880 (19.5)
Chinese American	604 (11.8)	75 (12.2)	529 (11.7)
Household per capita income per \$10 000, mean (SD)	51.8 (34.4)	48.0 (32.5)	52.4 (34.6)
Educational level, mean (SD), y	13.4 (3.8)	13.0 (4.0)	13.5 (3.8)
Behavioral and risk factors for T2DM			
Smoking status, No. (%)			
Former	1892 (36.9)	234 (38.0)	1658 (36.8)
Current	650 (12.7)	70 (11.4)	580 (12.9)
Alcohol use, No. (%) ^c			
Moderate	1582 (30.9)	152 (24.7)	1430 (31.7)
Heavy	419 (8.2)	29 (4.7)	390 (8.7)
Alternative Healthy Eating Index 2010, mean (SD) ^d	52.1 (11.7)	50.8 (11.4)	52.2 (11.8)
Intentional PA, No. (%) ^e			
Low	1821 (35.5)	248 (40.3)	1573 (34.9)
Middle	1599 (31.2)	186 (30.2)	1413 (31.3)
High	1704 (33.3)	182 (29.5)	1522 (33.8)
BMI, No. (%)			
18.0-24.9 (normal)	1568 (30.6)	77 (12.5)	1491 (33.1)
25.0-29.9 (overweight)	2044 (39.9)	221 (35.9)	1823 (40.4)
30.0 (obese)	1512 (29.5)	318 (51.6)	1194 (26.5)
Family history of T2DM, No. (%)	1791 (35.0)	298 (48.4)	1493 (33.1)
Neighborhood ^f			
Socioeconomic index, mean (SD)	0.5 (1.3)	0.2 (1.2)	0.6 (1.3)
Healthy food environment, median (IQR)			
GIS-based supermarkets and/or FV markets	1.0 (2.2)	1.0 (1.9)	1.0 (2.2)
Survey-based measure	3.5 (0.7)	3.4 (0.6)	3.5 (0.7)
Summary measure	-0.2 (2.1)	-0.4 (2.0)	-0.3 (2.2)

Characteristic	Total Sample (N = 5124)	T2DM Developed During Follow-up	
		Yes (n = 616)	No (n = 4508)
PA environment, median (IQR)			
GIS-based commercial recreational establishments	1.9 (2.9)	1.9 (2.7)	2.1 (2.9)
Survey-based measure	3.9 (0.4)	3.8 (0.3)	3.9 (0.4)
Summary measure	-0.1 (1.2)	-0.5 (1.0)	-0.4 (1.2)
Social environment, median (IQR)			
Survey-based social cohesion	3.5 (0.3)	3.5 (0.4)	3.5 (0.3)
Survey-based safety	3.7 (0.7)	3.6 (0.7)	3.7 (0.7)
Summary measure	-0.0 (2.1)	-0.1 (2.5)	-0.0 (2.0)

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in square meters); FV, fruit and vegetable; GIS, geographic information system; PA, physical activity; T2DM, type 2 diabetes mellitus.

^aData are from the Neighborhood Multi-Ethnic Study of Atherosclerosis, 2000 to 2012.²⁵

^bPercentages have been rounded and may not total 100.

^cDefined according to National Institute on Alcohol Abuse and Alcoholism definitions for men and women. Moderate drinking is defined as no more than 4 drinks on any single day and no more than 14 drinks per week for men, and no more than 3 drinks on any single day and no more than 7 drinks per week for women. Heavy drinking is defined as consumption in excess of moderate.

^dThe Alternative Healthy Eating Index 2010 is designed to capture a healthy diet and was compiled based on a food frequency questionnaire.⁴⁷ The index ranges from 2.5 to 87.5, with higher scores indicating a better-quality diet (high intake of fruits, vegetables, soy, protein, white meat, cereal fiber, polyunsaturated fat, and multivitamins and lower intake of alcohol, saturated fat, and red meat). Data were missing for 595 participants.

^eRefers to moderate and vigorous intentional PA, including walking for exercise, dance, team sports (eg, basketball, softball), dual sports (eg, tennis), individual activities (eg, golf, yoga), and conditioning activities (eg, running, swimming, cycling). Physical activity is measured in metabolic equivalent of task minutes per week and is categorized into tertiles for descriptive purposes.

^fNeighborhood characteristics are explained in the Neighborhood Physical and Social Environments subsection of the Methods section and in eAppendix 1 in the Supplement.

Table 3
Baseline Sociodemographic, Behavioral, and T2DM Risk Factor Characteristics by Tertiles of Baseline Neighborhood Summary Measures^a

Characteristic	Tertiles									
	All Participants (N = 5124)	Summary of Healthy Food Environment			Summary of PA Environment			Summary of Social Environment		
	Low (n = 1704)	Middle (n = 1739)	High (n = 1681)	Low (n = 1785)	Middle (n = 1705)	High (n = 1634)	Low (n = 1793)	Middle (n = 1680)	High (n = 1651)	
Sociodemographic										
Age, mean (SD), y	60.7 (9.9)	61.9 (10.2)	61.8 (10.3)	61.0 (10.0)	61.3 (10.3)	62.3 (10.2)	60.9 (10.3)	61.9 (10.4)	61.7 (9.8)	
Female sex, No. (%)	2747 (53.6)	899 (51.7)	935 (55.6)	931 (52.2)	957 (56.1)	859 (52.6)	991 (55.3)	904 (53.8)	852 (51.6)	
Race/ethnicity, No. (%)										
White	2168 (42.3)	877 (51.5)	655 (39.0)	537 (30.1)	740 (43.4)	891 (54.5)	381 (21.2)	734 (43.7)	1053 (63.8)	
Black	1311 (25.6)	549 (32.2)	376 (22.4)	615 (34.5)	326 (19.1)	370 (22.6)	689 (38.4)	297 (17.7)	325 (19.7)	
Hispanic	1041 (20.3)	233 (13.7)	492 (29.3)	398 (22.3)	393 (23.0)	250 (15.3)	583 (32.5)	321 (19.1)	137 (8.3)	
Chinese American	604 (11.8)	45 (2.6)	158 (9.4)	235 (13.2)	346 (20.3)	123 (7.5)	140 (7.8)	328 (19.5)	136 (8.2)	
Household per capita income per \$10 000, mean(SD)	51.8 (34.4)	51.6 (32.2)	49.3 (34.0)	43.5 (30.2)	48.2 (32.4)	64.4 (37.0)	39.6 (29.1)	49.5 (32.9)	67.4 (35.2)	
Educational level, mean (SD), y	13.4 (3.8)	13.6 (3.4)	13.2 (3.9)	12.6 (4.0)	13.2 (3.8)	14.6 (3.4)	12.2 (4.2)	13.5 (3.8)	14.7 (2.9)	
Risk factors for T2DM										
Smoking status, No. (%)										
Former	1892 (36.9)	673 (39.5)	600 (34.5)	619 (36.8)	582 (34.1)	674 (41.2)	619 (34.5)	608 (36.2)	665 (40.3)	
Current	650 (12.7)	247 (14.5)	201 (11.6)	262 (14.7)	212 (12.4)	176 (10.8)	300 (16.7)	192 (11.4)	158 (9.6)	
Alcohol use, No. (%)^b										
Moderate	1582 (30.9)	499 (29.3)	536 (30.8)	434 (24.3)	505 (29.6)	643 (39.4)	440 (24.5)	538 (32.0)	604 (36.6)	
Heavy	419 (8.2)	127 (7.5)	120 (6.9)	97 (5.4)	124 (7.3)	198 (12.1)	116 (6.5)	152 (9.0)	151 (9.1)	

Characteristic	Tertiles											
	All Participants (N = 5124)	Summary of Healthy Food Environment			Summary of PA Environment			Summary of Social Environment				
		Low (n = 1704)	Middle (n = 1739)	High (n = 1681)	Low (n = 1785)	Middle (n = 1705)	High (n = 1634)	Low (n = 1793)	Middle (n = 1680)	High (n = 1651)		
Alternative Healthy Eating Index 2010, mean(SD) ^c	52.1 (11.7)	52.5 (11.2)	53.8 (11.9)	51.0 (11.5)	51.5 (11.7)	53.7 (12.0)	50.6 (11.6)	52.5 (11.6)	53.1 (12.0)			
Intentional PA fertile, % ^b												
Low	1821 (35.5)	642 (37.7)	535 (31.8)	775 (43.4)	598 (35.1)	448 (27.4)	727 (40.5)	611 (36.4)	483 (29.3)			
Middle	1599 (31.2)	526 (30.9)	563 (32.4)	537 (30.1)	542 (31.8)	520 (31.8)	511 (28.5)	562 (33.5)	526 (31.9)			
High	1704 (33.3)	536 (31.5)	532 (30.6)	473 (26.5)	565 (33.1)	666 (40.8)	555 (31.0)	507 (30.2)	642 (38.9)			
BMI, No. (%)												
25.0-29.9 (overweight)	2044 (39.9)	686 (40.3)	706 (40.6)	652 (38.8)	716 (40.1)	674 (39.5)	703 (39.2)	655 (39.0)	686 (41.6)			
30.0 (obese)	1512 (29.5)	612 (35.9)	458 (26.3)	442 (26.3)	584 (32.7)	412 (25.2)	623 (34.7)	472 (28.1)	417 (25.3)			
Family history of T2DM, No. (%)	1791 (35.0)	665 (39.0)	615 (35.4)	511 (30.4)	685 (38.4)	524 (32.1)	640 (35.7)	595 (35.4)	556 (33.7)			
Neighborhood												
Socioeconomic index, mean (SD) ^b	0.5 (1.3)	-0.0 (0.8)	0.4 (0.9)	1.3 (1.6)	-0.2 (0.8)	0.3 (0.9)	1.6 (1.4)	-0.4 (0.9)	0.4 (1.4)	1.2 (1.4)		

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in square meters); PA, physical activity; T2DM, type 2 diabetes mellitus.

^aData are from the Neighborhood Multi-Ethnic Study of Atherosclerosis, 2000 to 2012.²⁵

^bDescribed in Table 2.

^cDescribed in Table 2. Data were missing for 595 participants.

Table 4
Crude Incidence Rates of T2DM by Tertiles of Neighborhood Measures at Baseline^a

Neighborhood Measure	Tertile, Incidence per 1000 Person-years (95% CI)		
	Low	Middle	High
Healthy food environment			
GIS-based supermarkets or FV markets	17.9 (15.7-20.4)	15.8 (13.7-18.1)	15.8 (13.7-18.1)
Survey-based	17.5 (15.3-20.0)	19.8 (17.5-22.5)	12.1 (10.3-14.1)
Summary	16.9 (14.8-19.3)	18.2 (16.0-20.8)	14.3 (12.3-16.6)
PA environment			
GIS-based commercial recreational establishments	20.3 (17.8-23.3)	14.6 (12.8-16.7)	15.4 (13.4-17.8)
Survey-based	20.8 (18.4-23.5)	17.8 (15.6-20.3)	10.6 (9.0-12.7)
Summary	20.5 (18.2-23.2)	17.1 (15.0-19.6)	11.8 (10.0-13.8)
Social environment			
Survey-based social cohesion	18.5 (16.3-21.1)	14.6 (12.7-16.9)	16.3 (14.2-18.8)
Survey-based safety	18.7 (16.5-21.3)	17.3 (15.2-19.8)	13.4 (11.5-15.6)
Summary	19.7 (17.4-22.3)	15.7 (13.6-18.1)	14.0 (12.1-16.2)

Abbreviations: FV, fruit and vegetable; GIS, geographic information system; PA, physical activity; T2DM, type 2 diabetes mellitus.

^aData are from the Neighborhood Multi-Ethnic Study of Atherosclerosis, 2000 to 2012.²⁵ Incidence rates were calculated using Poisson regression according to tertiles of the neighborhood exposures at baseline. Low tertile indicates worst; high, best. Overall incidence rate in the full sample was 16.5 (95% CI, 15.2-17.8) per 1000 person-years. Measures are described in Table 1.