**The Relative Contributions of Socioeconomic, Local Environmental, Psychosocial, Lifestyle/Behavioral, Biophysiologic, and Ancestral Factors to Racial/Ethnic Disparities in Type 2 Diabetes**

**Rebecca S. Piccolo, ScM** (corresponding author)

London School of Hygiene and Tropical Medicine

Keppel Street

London, U.K. WC1E 7HT

T: +1 (315) 430 3911

[rebecca.piccolo@lshtm.ac.uk](mailto:rpiccolo@neriscience.com)

**S.V. Subramanian, PhD**

Professor of Population Health and Geography

Harvard University

677 Huntington Avenue

Kresge Building 7th Floor, 716

Boston, Massachusetts 02115-6096

T: +1 (617) 432 6299

svsubram@hsph.harvard.edu

**Neil Pearce, PhD DSc**

Professor of Epidemiology and Biostatistics

London School of Hygiene and Tropical Medicine

Keppel Street

London, U.K. WC1E 7HT

T: +44 (0)20 9588151

**Jose C. Florez, MD, PhD**

Assistant Physician in Endocrinology

Center for Human Genetic Research/Diabetes Unit

Massachusetts General Hospital

Associate Professor

Harvard Medical School

Tel. (617) 643-3308

JCFLOREZ@mgh.harvard.edu

**John B. McKinlay**

Senior Vice President, and

Head, Department of Epidemiology,

New England Research Institutes

9 Galen St.

Watertown, MA 02472

T: +1 (617) 972-3012

F: +1 (617) 926-4282

**Word Count:** 2991/3000

**ACKNOWLEDGEMENTS**

**Author Contributions:**

Rebecca S. Piccolo had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

*Study concept and design:* McKinlay, Florez

*Acquisition of data:* Piccolo, McKinlay

*Analysis and Interpretation of data:* Piccolo, Subramanian, Florez

*Drafting of the manuscript:* Piccolo

*Critical revision of the manuscript for important intellectual content:* Piccolo, Subramanian, Pearce, Florez, McKinlay

*Statistical analysis:* Piccolo

*Obtained funding:* McKinlay

*Administrative, technical, or material support:* Piccolo, Pearce, McKinlay

*Study supervision:* Piccolo, McKinlay

**Conflicts of Interest Disclosures:** All authors attest that they have no potential conflicts of interest, including relevant financial interests, activities, relationships, and affiliations. This study was supported by grants from the National Institutes of Health (DK056842 and DK080786).

**Role of the sponsor:** The National Institute of Diabetes and Digestive and Kidney Diseases had no role in the design and conduct of the study, in the collection, analysis, and interpretation of the data, or in the preparation, review, or approval of the manuscript.

**Online-Only Material:** eAppendix A provides additional detail on the operationalization of the measures included in the Boston Area Community Health Survey. eTable 1 provides the results that were the basis for the development of the risk scores. eTable 2 provides the full standardized and unstandardized results.

**ABSTRACT**

**Background:** Racial/ethnic minorities in the US have a higher prevalence of type 2 diabetes (T2D). While many independent risk factors for T2D have been identified, they are often viewed in isolation without considering the joint contributions of competing risk factors.

**Objective:** To assess the relative contributions of six domains of influence to racial/ethnic disparities in T2D: (1) socioeconomic, (2), local environmental, (3) psychosocial, (4) lifestyle/behavioral, (5) biophysiologic, and (6) biogeographic ancestry.

**Research Design/Subjects:** We analyzed cross-sectional data from the Boston Area Community Health (BACH) III Survey (2010-12), the third wave of a random, population-based sample of men and women from three racial/ethnic groups (black, Hispanic, white) living in Boston, Massachusetts (N=2,764). We constructed structural equation models to evaluate the direct effects of each conceptual domain on T2D prevalence, as well as the indirect effect of each conceptual domain on the magnitude of the racial/ethnic disparities in T2D.

**Measures:** Surveys and measurements were conducted in-home after an 8 hour fast. Prevalent diabetes was defined by self-report of T2D, fasting glucose >125 mg/dL, or HbA1c ≥6.5%.

**Results:** In the final model 38.9% and 21.8% of total effect of self-identified black race and Hispanic ethnicity, respectively, on T2D prevalence was mediated by the socioeconomic, environmental, psychosocial, and lifestyle/behavioral risk scores. The largest mediator was the socioeconomic risk score, which explained 21.8% and 26.2% of the total effect of black race and Hispanic ethnicity, respectively.

**Conclusions:** In BACH, socioeconomic factors explain the greatest contribution to the excess prevalence of T2D among racial/ethnic minorities.

Word Count: 249/250

**BACKGROUND**

Disparities in type 2 diabetes (T2D) by race/ethnicity are a serious public health problem in the United States and worldwide. Compared to white adults, the prevalence of diabetes is 77% higher among black and 66% higher among Hispanic adults in the US.1 Racial/ethnic disparities in T2D are associated with disparities in glycemic control, 2 elevated rates of diabetes-related complications, and greater health care costs.[3](#_ENREF_9)

Many potential contributors to these disparities have been identified,4 including variations in lifestyles and behaviors, biophysiological, psychosocial, socio-demographic, and environmental factors, and biogeographic ancestry (BGA).5, 6 However, research to date has largely focus on individual risk factors in isolation and the relative contributions of these influences have not been identified. 4

Since racial/ethnic differences in T2D appear to result from a broad range of influences, a more complete understanding requires a multidimensional approach. A multilevel risk model, reflecting the many factors that contribute to T2D risk, may advance understanding, and better inform the design of interventions to target racial and ethnic disparities.

The aim of this research is to develop and test a conceptual risk model that takes a multilevel approach to T2D disparities (**Figure 1**). Statistical methods are available that allow us to test this conceptual model which includes both direct and indirect (mediating) effects. Our aim is to assess the relative contributions of six domains of influence to racial/ethnic disparities in T2D: (1) socioeconomic, (2), local environmental, (3) psychosocial, (4) lifestyle/behavioral, (5) biophysiologic, and (6) BGA.

**Methods**

***Study Sample***

The Boston Area Community Health (BACH) Survey is a longitudinal, random, population-based, cohort of 5,502 residents (2,301 men, 3,201 women) aged 30-79 from three racial/ethnic groups in Boston, MA. [7](#_ENREF_29) BACH has conducted total of three surveys to date (BACH I: 2002-2006; BACH II: 2008-2010; BACH III: 2010-2012). The current analysis uses cross-sectional data from the third survey (BACH III, N=3,155). Analyses were restricted to 2,764 participants still residing in Boston, Massachusetts at BACH III due to the availability of environmental parameters. Survey participants were interviewed in the morning after being instructed to fast overnight (≥8 hours) and after providing written informed consent. The interviews were conducted by trained, certified phlebotomists fluent in English and/or Spanish. The response rate, conditional on previous participation, was 81.4%.[8](#_ENREF_30) The New England Research Institutes’ Institutional Review Board approved the study.

***Measures***

The primary determinant of interest was self-identified race/ethnicity (non-Hispanic black [black], non-Hispanic white [white], and Hispanic). The primary outcome was prevalent T2D. Fasting glucose (FG) was measured with a HemoCue 201 point-of-care analyzer. Glycated hemoglobin (HbA1c) was measured by Quest Laboratories in Cambridge, MA. Participants who (a) self-reported T2D (“have you ever been told by a doctor or other health professional that you have type 2 diabetes?”), or (b) had FG >125 mg/dL or HbA1c ≥6.5% were classified as having T2D.[9](#_ENREF_12) Medication inventory and age of diagnosis was used to further separate type 1 versus type 2 diabetes. Eight individuals younger than 35 years at diagnosis and on continuous insulin therapy were considered to have type 1 diabetes and were excluded. The medication inventory also confirmed over 80% of the self-reported cases.

Our multilevel approach builds upon an earlier theoretical model[10](#_ENREF_31) and includes six domains of influence (**Figure 1**) which are hypothesized to directly and/or indirectly. The constructs measured within each domain are briefly described below. Additional details can be found in **eAppendix A**.

***Socio-economic influences*** considered included household income, education, occupation, perceptions of everyday discrimination,[11](#_ENREF_32) immigration status, acculturation,[12](#_ENREF_33) health literacy, type of health insurance, and number of health care visits. ArcGIS 10.1 (ESRI, Redlands, California) was used to geocode participants’ residences and link participants with geographic features. ***Environmental influences*** included census-tract socioeconomic status,[13](#_ENREF_35) percent poverty, percent non-Hispanic black, percent non-Hispanic white, violent and property crime per 1,000 population, distance to the closest grocery store, convenience store, and fast food (miles), percentage of recreational open space, perceived social and physical disorder,[14](#_ENREF_37) and number of years at current address. ***Spatial access to health care*** was assessed by distance to the closest community health center, acute care hospital, or health care center (miles). ***Psychosocial influences***considered included hours of sleep each night, major life events,[15](#_ENREF_38) and sense of personal control.[16](#_ENREF_39) ***Lifestyle/behavioral*** factors assessed: dietary patterns (2005 Block food frequency questionnaire[17](#_ENREF_40) assessed average daily intake of sodium, vegetables, fruits, meats/beans, grains, fiber, and saturated fat comprising a “healthy eating score” which was adjusted for total kilocalories), physical activity,18 and smoking history. BMI, waist circumference, and body fat percentage were measured by trained field interviewers. ***Biophysiological influences***included:blood pressure (average of three readings taken during the in-home visit), total cholesterol, HDL cholesterol, triglycerides (Quest laboratories, Cambridge, MA), reported high blood pressure or cardiovascular disease, and for women only menopausal status and history of gestational diabetes.

To measure ***BGA*** we evaluated 63 ancestry informative markers, including 33 autosomal single nucleotide polymorphisms (SNPs) differentiating Native American versus European ancestry and 30 SNPs differentiating West African versus European ancestry. The 63 markers combined can provide an estimate of percent West African, Native American, and European ancestry for each participant.19, 20 Genotyping was conducted at the Broad Institute using the Sequenom iPLEX platform. ***Family history*** of diabetes was also considered as an independent risk factor for T2D.21Race/ethnicity, age, and gender, BGA, and family history of diabetes were considered exogenous factors.

***Structural Equation Modeling***

We applied two-level structural equation modeling (SEM) to assess associations between race/ethnicity, confounding and mediating characteristics, and T2D. Two-level SEM allows us to include both direct and indirect effects of each risk domain on T2D as hypothesized in the conceptual model (**Figure 1**) while accommodating clustering of participant observations (level 1) within their census tract of residence (level 2). Direct effects are depicted as arrows from independent to dependent variables. For example, socioeconomic risk may have a direct effect on T2D (depicted in **Figure 1** by a single arrow from socioeconomic risk to T2D, the final outcome variable). Indirect effects are depicted as a series of arrows operating through mediating construct(s). For example, socioeconomic risk may contribute to increased lifestyle/behavioral risk which in turn contributes to T2D and serves as a mediating influence. We relied on published literature and inherent temporality to determine the direction of effects. Correlations between measurement errors of two variables are represented by bi-directional curves. Standardized coefficients (sβ)[22](#_ENREF_46) and their *p*-values are reported. We performed mediation analysis to assess the percentage of the racial/ethnic effect explained by each the five mediating domains of influence. The mediated, or indirect, effect is calculated as the product of the direct effects (sβ) among the independent, mediating, and any subsequent dependent variables.[23](#_ENREF_47) The overall percent mediated was calculated as the indirect effect over the total effect. Descriptive statistics were estimated using SAS callable Sudaan Version 11 and SEMs were estimated using Mplus Version 7 (Muthen and Muthen, Los Angeles, CA).

***Development of the Risk Scores***

We used data based on the mediating domains of influence to create risk scores. Variables listed in **Figure 1** were reduced from those in the conceptual model using race/ethnicity-, age-, and gender-adjusted models (**eTable 1**). Variables that did not either (1) meet a minimal criterion for association with T2D (*p*<0.10) or (2) reduce the race/ethnic effect (OR) by 10% were not included in the domain risk score. For categorical variables, we created a weighted scoring system by rounding up all regression coefficients (ln(OR)) to the nearest integer, using methods similar to those utilized in Bang *et al.*[24](#_ENREF_49) which is the basis for the American Diabetes Association self-screening tool. For continuous variables, risk was based on clinically accepted “high risk” criteria (see **Table 1** for citations). If clinical criteria were not available, tertiles were used. After constructing the final model, all variables were added in the model singly to ensure their effects were captured by the risk scores.

To minimize reductions in precision, we used Multivariate Imputation by Chained Equations (MICE)[25](#_ENREF_50) in R (Vienna, Austria). We created 15 datasets and conducted imputations within each racial/ethnic by gender combination to preserve interaction effects, while taking into account the complex survey sample design. We obtained DNA samples from 73.1% of participants, 24.4% of participants were missing household income and 25.8% were missing dietary data. The proportions of missing data for other covariates were low (<10%). BACH’s sampling design requires weighting observations inversely proportional to their probability of selection for results to be generalizable to the base population. 26 Sampling weights were post-stratified in order to produce estimates representative of the black, Hispanic, and white Boston, MA.

**Results**

The prevalence of diabetes in the BACH III study was 23.4%. The demographic characteristics of 2,476 participants the analytic sample are presented in **Table 2.**  The sample was comprised of approximately 1/3 black (29.0%), Hispanic (32.8%), and white (34.6%) participants and the average age of the participants was 54. Compared with non-diabetic participants, participants with T2D were older, had greater West African genetic ancestry, were of lower SES, reported greater discrimination, lower health literacy, lived in lower SES/greater poverty census tracts and neighborhoods with more minority residents, reported greater neighborhood disorder, short (<6 hours) or long (>9 hours) sleep, reported a lower sense of control, less physical activity, greater BMI, waist circumference and body fat percentage, had higher blood pressure, total cholesterol, and triglycerides and lower HDL cholesterol.

Using results from the race/ethnic-, gender-, and age-adjusted models (**eTable 1**), we identified 24 variables within the 5 mediating domains that were associated with T2D prevalence and/or racial/ethnic disparities in T2D (**Table 1**). This produced risk scores with the following ranges and means: socioeconomic (0-10, 4.3), environmental (0-1, 0.4), psychosocial (0-5, 1.7), lifestyle/behavioral (0-8, 3.2), and biophysiological (0-11, 2.7).

The SEM specified in **Figure 1** fits the data well. Age and gender had direct effects on almost all factors with the exception of environmental risk. For simplicity, age and gender effects and non-significant pathways (*p*≥0.05) are not presented in **Figure 2** (full results are available in **eTable 2**). The lifestyle/behavioral domain was the largest direct predictor of T2D status (sβ =0.25, *p*<0.001) followed by biophysiologic factors (0.19, *p*<0.001), socioeconomic factors (0.13, *p*=0.003), and family history of diabetes (0.10, *p*=0.005). There was a marginal direct effect of self-identified race/ethnicity on T2D prevalence (Black, 0.18, *p*=0.054; Hispanic, 0.10, *p*=0.069). The standardized coefficients represented in **Figure 2** can be interpreted as a one standard deviation difference in the predictor (i.e. lifestyle/behavioral risk) is associated with a 0.25 standard deviation difference in the outcome (i.e. T2D). Unstandardized coefficients are available online (**eTable 2**). The unstandardized coefficients suggest that for every one unit increase in the lifestyle/behavioral risk score the odds of T2D increase 35% (OR=1.35) and for every one unit increase in the biophysiological risk score the odds increase 29% (OR=1.29).

Self-identified black race had a significant direct effect on socioeconomic risk (sβ=0.23, *p*=0.003) and environmental risk (0.14, *p*=0.001) only. There was no direct effect of self-identified black race on psychosocial, lifestyle/behavioral, biophysiological risk, or T2D. However, black race has an indirect effect on these outcomes through socioeconomic factors. However, black race has an indirect effect on these outcomes through socioeconomic factors. Socioeconomic risk is 43.3% mediated by lifestyle/behavioral risk. The mediation analysis (**Figure 2**) indicate that 38.9% of total effect of black race was mediated by the socioeconomic, environmental, psychosocial, lifestyle/behavioral risk scores with 21.8% of the total effect of black race being explained by socioeconomic risk.

Self-identified Hispanic ethnicity had a significant direct effect on socioeconomic risk (0.17, *p*<0.001), environmental risk (0.29, *p*<0.001), and psychosocial risk (0.17, *p*=0.04). There was no significant direct effect of Hispanic ethnicity on lifestyle/behavioral risk, biophysiological risk, or T2D. Mediation analyses indicate that 45.7% of total effect of Hispanic ethnicity was explained by the calculated risk scores. The largest mediator, 26.2%, was the socioeconomic risk score.

Despite the considerable differences in BGA among participants with and without T2D in the bivariate results (**Table 2**), neither West African ancestry (OR=1.02, *p*=0.658) nor Native American ancestry (OR=0.94, *p*=0.428*)* contributed to T2D once self-identified race/ethnicity was included in the model (**eTable 1**). The final SEM also indicated that there was no significant direct effect of West African (StdYX=-0.003, *p*=0.069*)* or Native American ancestry (StdYX=-0.016, *p*=0.725) on T2D once self-identified race/ethnicity was accounted for (**eTable 2**).

**Discussion**

To our knowledge, this study presents the first examination of a multilevel risk model aimed at explaining racial/ethnic disparities in T2D. While many authors have proposed similar conceptual frameworks with the aim of understanding and eliminating health disparities,10, 27 to our knowledge the BACH study is the first survey to amass this data and test this model of health disparities in T2D in a community-based population with adequate numbers of black, Hispanic, and white participants.

Under our conceptual framework, biophysiological and individual lifestyle/behavioral factors were considered more proximate to T2D. The data supported this temporality as individual lifestyle/behavioral risk had the largest direct effect on T2D and biophysiological risk the second largest direct effect. However, the mediation analyses indicate that only 5% and 11% of the total effect of black race can be explained by excess biophysiological and lifestyle/behavioral risk, respectively. Among Hispanic participants, the percentage mediated was even lower. Further, the mediation analyses indicate that the largest explainable proportion of the excess odds of T2D among black and Hispanic participants is attributable to socioeconomic risk. The socioeconomic risk score developed, which is a composite of household income, education, occupation, immigration status, acculturation, health literacy, insurance status and utilization of health care explains 22% of the excess odds of T2D among black and 26% of the excess odds among Hispanic participants. The statistical analyses indicate that while much of the excess odds of T2D among blacks and Hispanics remains unexplained (61% and 54%, respectively), adverse socioeconomic conditions explains the largest explainable proportion of racial/ethnic disparities in T2D.

Our data, supported by our previous findings,[28](#_ENREF_55) suggest that the effects of BGA on T2D are attenuated with further adjustment for self-identified race/ethnicity and nearly eliminated when socioeconomic and lifestyle/behavioral pathways are considered. This finding is supported by several studies.5, 6, 29 However, other studies have found the effect of BGA on T2D to be more robust to adjustment,*6, 30, 31*  including research from BACH which demonstrate that the effect of BGA on prediabetic illness may be robust to adjustment for social factors.[32](#_ENREF_59) Race and ethnicity are complex multidimensional constructs reflecting biogeographic origin, biological factors, as well as social, cultural, and economic factors. [33](#_ENREF_60) Our findings suggest that while BGA may be associated with T2D, it is likely that the social, cultural, and economic facets of race/ethnicity may better explain T2D disparities in the BACH study.

Family history of diabetes, which may have a genetic component, but may also be the result of similar socioeconomic, environmental, psychosocial, lifestyle, and biophysiological risk profiles between parent and offspring, had a modest direct effect of T2D prevalence (0.10, *p*=0.005) and was associated with race/ethnicity.

While race/ethnicity had no direct effect on lifestyle/behavioral risk, it is important to note that socioeconomic risk, which was highly associated with race/ethnicity, did have a significant direct effect on lifestyle/behavioral risk. Overall, lifestyle/behavioral risk explained 43.3% of the socioeconomic effect on T2D. Studies that aim to assess the role of lifestyle and behavioral factors on the socioeconomic gradient of health in T2D have found similar results. For example, the Whitehall II cohort study found that lifestyle/behavioral factors accounted for 33-45% of the socioeconomic gradient in T2D.[34](#_ENREF_61)

Each domain of the conceptual model presented here suggests a particular structural intervention. Increased socioeconomic risk suggests policy interventions affecting social conditions; environmental risk--community intervention; psychosocial risk--primary prevention aimed at reducing psychological strain and increasing coping mechanisms; lifestyle/behavioral risk--primary prevention directed at increasing healthy and decreasing unhealthy behaviors; and biophysiological risk--secondary prevention efforts aimed at stopping/slowing the progression of disease. The results of these analyses, as well as the results of several trials,[35](#_ENREF_62) suggest that interventions targeting lifestyle/behavioral and biophysiologic risk may reduce T2D risk overall. However, these results demonstrate that interventions aimed at reducing disparities may need to target socioeconomic risk factors in order to lessen the racial/ethnic divide.

***Strengths and limitations***

A substantial limitation to this analysis is the cross-sectional design. One-time measurement of health behaviors may underestimate their contribution. Life-course and repeated measures designs have shown to increase the proportion of social inequalities that can be explained by potential modifiable risk factors.

Second, although the sample is limited to Boston, Massachusetts, the BACH Survey sample has been compared to other large regional (Behavioral Risk Factor Surveillance System) and national (National Health and Nutrition Examination Survey) on a number of socio-demographic and health-related variables. Results suggest that BACH Survey estimates of key health conditions are comparable with national trends.[7](#_ENREF_29)

Third, although we measured BGA markers, which are thought to estimate the genetic contribution to increased diabetes prevalence in certain populations, the current status of genetic discovery in T2D across various ethnic groups does not allow us to generate comprehensive multiethnic genetic risk scores for T2D. We therefore cannot make well informed conclusions regarding genetic contributors to racial/ethnic disparities in T2D.

Fourth, our assessment of the metabolic/biophysiologic domain encompasses cardiovascular risk factors such as blood pressure and dyslipidemia, but deliberately excludes glycemic parameters, because these are included in the outcome definition and are direct components of T2D pathogenesis. A complementary study focusing on fasting glucose and HbA1c in this cohort has been published previously.32

Key strengths of this study stem from the community-based, stratified, random sample design of the BACH Survey, a large cohort of Black, Hispanic, and White men and women. Since this study was designed to test this specific conceptual model of disparities, validated scales with published metrics measuring the constructs of interest were used where available (**eAppendix A**). Finally, we did not rely solely on self-report for T2D status. Participants were contacted in the morning in their home, giving a more accurate prevalence of T2D.

***Conclusions***

Our study found that while lifestyle/behavioral and biophysiologic risk factors had the greatest direct effect on T2D risk, socioeconomic factors had the greatest impact on explaining racial/ethnic disparities in T2D.

**REFERENCES**

**1.** 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, GA2011.

**2.** Kirk JK, D'Agostino RB, Jr., Bell RA, et al. Disparities in HbA1c levels between African-American and non-Hispanic white adults with diabetes: a meta-analysis. *Diabetes Care.* Sep 2006;29(9):2130-2136.

**3.** Feng Y, Mao G, Ren X, et al. Ser1369Ala variant in sulfonylurea receptor gene *ABCC8* is associated with antidiabetic efficacy of gliclazide in Chinese type 2 diabetic patients. *Diabetes Care.* Oct 2008;31(10):1939-1944.

**4.** Golden SH, Brown A, Cauley JA, et al. Health disparities in endocrine disorders: biological, clinical, and nonclinical factors--an Endocrine Society scientific statement. *J Clin Endocrinol Metab.* Sep;97(9):E1579-1639.

**5.** Florez JC, Price AL, Campbell D, et al. Strong association of socioeconomic status with genetic ancestry in Latinos: implications for admixture studies of type 2 diabetes. *Diabetologia.* Aug 2009;52(8):1528-1536.

**6.** Qi L, Nassir R, Kosoy R, et al. Relationship between diabetes risk and admixture in postmenopausal African-American and Hispanic-American women. *Diabetologia.* May 2012;55(5):1329-1337.

**7.** McKinlay JB, Link CL. Measuring the urologic iceberg: design and implementation of the Boston Area Community Health (BACH) Survey. *European urology.* Aug 2007;52(2):389-396.

**8.** Piccolo RS, Araujo AB, Pearce N, McKinlay JB. Cohort Profile: The Boston Area Community Health (BACH) survey. *International Journal of Epidemiology.* 2014;43(1):42-51.

**9.** American Diabetes Association. Standards of medical care in diabetes--2012. *Diabetes Care.* Jan 2012;35 Suppl 1:S11-63.

**10.** McKinlay J, Marceau L. US public health and the 21st century: diabetes mellitus. *Lancet.* Aug 26 2000;356(9231):757-761.

**11.** Williams DR, Yu Y, Jackson J, Anderson N. Racial differences in physical and mental health: socioeconomic status, stress, and discrimination. *Journal of health psychology.* 1997;2(3):335-351.

**12.** Marin G, Gamba RJ. A New Measurement of Acculturation for Hispanics: The Bidemensional Acculturation Scale for Hispanics (BAS). *Hispanic Journal of Behavioral Sciences.* 1996;18(3):297-316.

**13.** Diez-Roux AV, Kiefe CI, Jacobs DR, Jr., et al. Area characteristics and individual-level socioeconomic position indicators in three population-based epidemiologic studies. *Annals of epidemiology.* Aug 2001;11(6):395-405.

**14.** Ross CE, Mirowsky J. Disorder and decay: The concept and measurement of perceived neighborhood disorder. *Urban Affairs Review.* 1999;34:412-432.

**15.** Rahe RH. Subjects' recent life changes and their near-future illness susceptibility. *Adv Psychosom Med.* 1972;8:2-19.

**16.** Mirowsky J, Ross CE. Eliminating defense and agreement bias from measures of the sense of control: A 2 x 2 index. *Social Psychology Quarterly.* 1991;54(2):127-145.

**17.** Block G, Hartman AM, Dresser CM, Carroll MD, Gannon J, Gardner L. A data-based approach to diet questionnaire design and testing. *American journal of epidemiology.* Sep 1986;124(3):453-469.

**18.** Washburn RA, Ficker JL. Physical Activity Scale for the Elderly (PASE): the relationship with activity measured by a portable accelerometer. *J Sports Med Phys Fitness.* Dec 1999;39(4):336-340.

**19.** Smith MW, Patterson N, Lautenberger JA, et al. A high-density admixture map for disease gene discovery in african americans. *American journal of human genetics.* May 2004;74(5):1001-1013.

**20.** Price AL, Patterson N, Yu F, et al. A Genomewide Admixture Map for Latino Populations. *American journal of human genetics.* 2007;80(6):1024-1036.

**21.** Meigs JB, Shrader P, Sullivan LM, et al. Genotype score in addition to common risk factors for prediction of type 2 diabetes. *N Engl J Med.* Nov 20 2008;359(21):2208-2219.

**22.** Muthén BO. Applications of causally defined direct and indirect effects in mediation analysis using SEM in Mplus. 2011.

**23.** Kline RB. *Priciples and practice of structural equation modeling*. 2nd ed. New York: Guilford Press; 2005.

**24.** Bang H, Edwards AM, Bomback AS, et al. Development and validation of a patient self-assessment score for diabetes risk. *Annals of internal medicine.* Dec 1 2009;151(11):775-783.

**25.** van Buuren S, Groothuis-Oudshoorn K. mice: Mutlivariate Imputation by Chained Equations in R. *Journal of Statistical Software.* 2011;45(3):1-67.

**26.** Cochran W. *Sampling Techniques, 3rd ed*. New York: John Wiley & Sons; 1977.

**27.** Warnecke RB, Oh A, Breen N, et al. Approaching health disparities from a population perspective: the National Institutes of Health Centers for Population Health and Health Disparities. *American journal of public health.* Sep 2008;98(9):1608-1615.

**28.** Piccolo RS, Pearce N, Araujo AB, McKinlay JB. The contribution of biogeographic ancestry and socioeconomic status to racial/ethnic disparities in type 2 diabetes: Results from the Boston Area Community Health (BACH) Survey. *Annals of epidemiology.* 2014;In Press.

**29.** Maruthur NM, Kao WH, Clark JM, et al. Does genetic ancestry explain higher values of glycated hemoglobin in African Americans? *Diabetes.* Sep 2011;60(9):2434-2438.

**30.** Cheng CY, Reich D, Haiman CA, et al. African ancestry and its correlation to type 2 diabetes in African Americans: a genetic admixture analysis in three U.S. population cohorts. *PloS one.* 2012;7(3):e32840.

**31.** Lai CQ, Tucker KL, Choudhry S, et al. Population admixture associated with disease prevalence in the Boston Puerto Rican health study. *Hum Genet.* Mar 2009;125(2):199-209.

**32.** Meigs JB, Grant RW, Piccolo R, et al. Association of African genetic ancestry with fasting glucose and HbA1c levels in non-diabetic individuals: the Boston Area Community Health (BACH) Prediabetes Study. *Diabetologia.* Sep 2014;57(9):1850-1858.

**33.** Williams DR. Race and health: basic questions, emerging directions. *Annals of epidemiology.* Jul 1997;7(5):322-333.

**34.** Stringhini S, Tabak AG, Akbaraly TN, et al. Contribution of modifiable risk factors to social inequalities in type 2 diabetes: prospective Whitehall II cohort study. *BMJ.* 2012;345:e5452.

**35.** Knowler WC, Fowler SE, Hamman RF, et al. 10-year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study. *Lancet.* Nov 14 2009;374(9702):1677-1686.

**36.** Alberti KG, Eckel RH, Grundy SM, et al. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation.* Oct 20 2009;120(16):1640-1645.

**37.** Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation.* Dec 17 2002;106(25):3143-3421.

**38.** OMB (Office of Management and Budget). Recommendations from the Interagency Committee for the Review of the Racial and Ethnic Standards to the Office of Management and Budget concerning changes to the standards for the classification of federal data on race and ethnicity, Revisions to the standards for the classification of federal data on race and ethnicity. In: Register F, ed1997a, 1997b:36873-36946.

**39.** Lynch J, Kaplan G. Socioeconomic position. In: Berkman LF, Kawachi I, eds. *Social Epidemiology*. New York Oxford University Press; 2000:13-35.

**40.** Baker D, Williams M, Parker R, Grazmararian J, Nurss J. Development of a brief test to measure functional health literacy. *Patient Educ Couns.* 1999;38(1):33-42.

**41.** Diez-Roux AV, Nieto FJ, Muntaner C, et al. Neighborhood environments and coronary heart disease: a multilevel analysis. *American journal of epidemiology.* Jul 1 1997;146(1):48-63.

**42.** Yang W, Spears K, Zhang F, Lee W, Himler HL. Evaluation of personal and built environment attributes to physical activity: a multilevel analysis on multiple population-based data sources. *Journal of obesity.* 2012;2012:548910.

**43.** Neckerman KM, Bader MD, Richards CA, et al. Disparities in the food environments of New York City public schools. *Am J Prev Med.* Sep 2010;39(3):195-202.

**44.** Moore LV, Diez Roux A. Associations of neighborhood characteristics with the location and type of food stores. *American journal of public health.* 2006;96(2):325-331.

**45.** Oreskovic NM, Kuhlthau KA, Romm D, Perrin JM. Built environment and weight disparities among children in high- and low- income towns. *Academic Pediatrics.* 2009;9(5):315-321.

**46.** Ross CE, Mirowsky J. Neighborhood Disorder, Subjective Alienation, and Distress. *Journal of health and social behavior.* 2009;50(March):49-64.

**47.** Ross CE, Mirowsky J, Pribesh S. Powerlessness and the Amplification of Threat: Neighborhood Disadvantage, Disorder, and Mistrust. *American Sociological Review.* 2001;66(4):568-591.

**48.** Commonwealth of Massachusetts. Office of Geographic Information (MassGIS). 2013; <http://www.mass.gov/anf/research-and-tech/it-serv-and-support/application-serv/office-of-geographic-information-massgis/>. Accessed November 16, 2013.

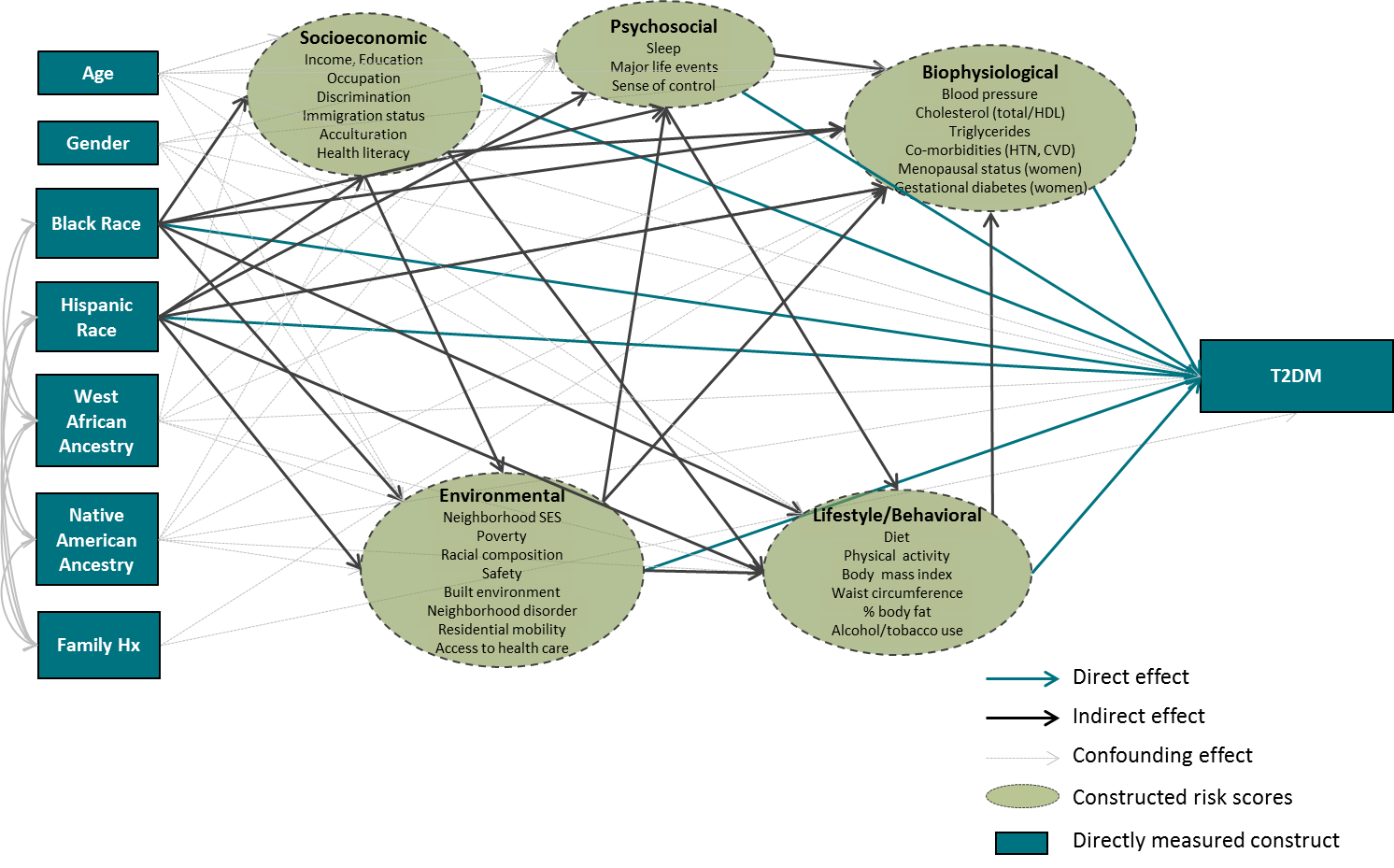
**49.** Wolinsky FD, Wyrwich KW, Metz SM, Babu AN, Tierney WM, Kroenke K. Test-retest reliability of the Mirowsky-Ross 2 x 2 Index of the Sense of Control. *Psychological reports.* Apr 2004;94(2):725-732.

**50.** USDA Center for Nutrition Policy and Promotion. Health Eating Index-2005. 2008.

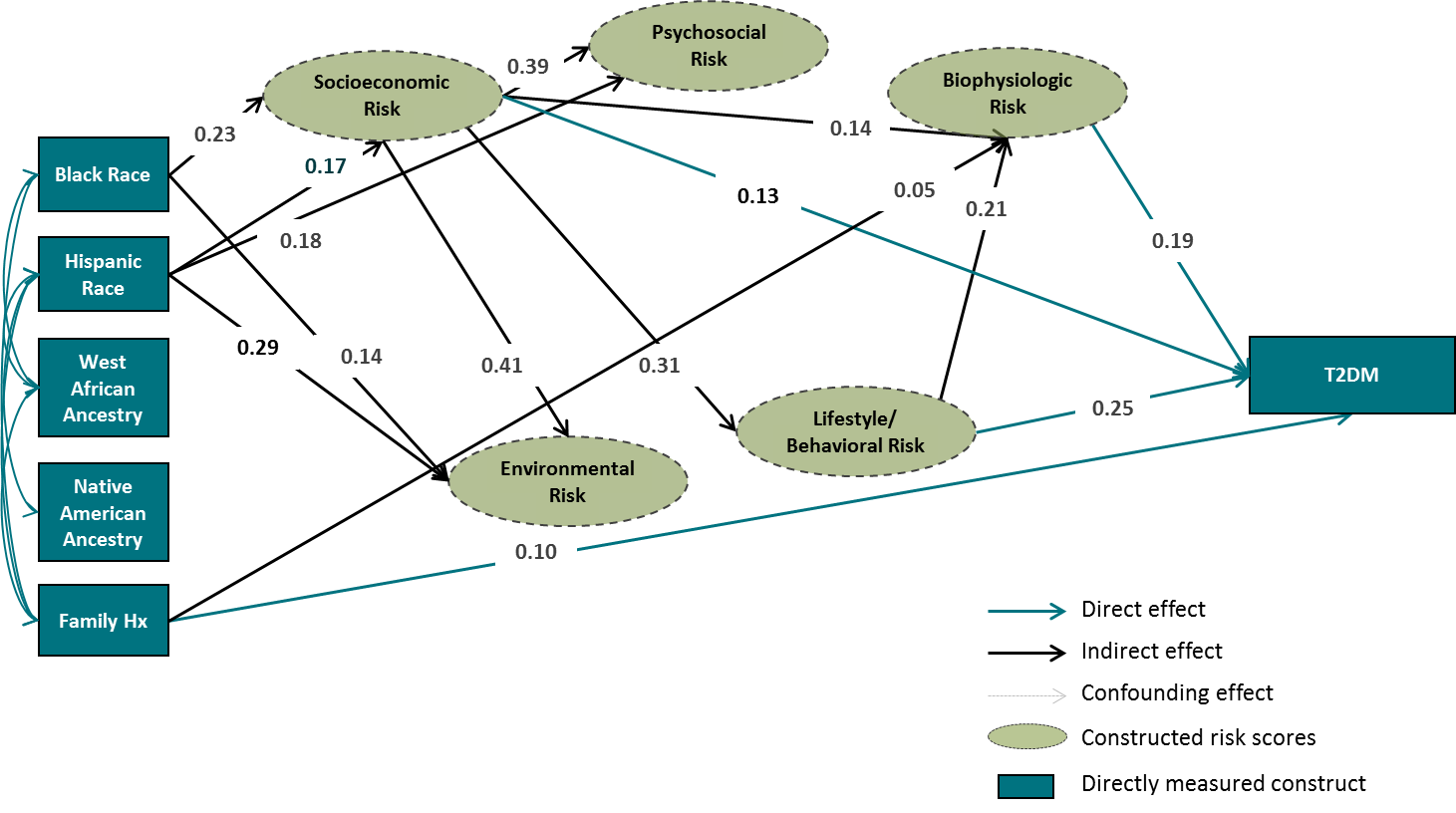
**FIGURES**

**Figure 1. Conceptual Model of Potential Factors Influencing Racial/Ethnic Disparities in T2D**

Potential factors influencing racial/ethnic disparities in T2D are grouped into five domains of influence: social structure, environmental, psychosocial, lifestyle/behavioral, Biophysiological. Race/ethnicity, age, gender, and genetic constructs are considered exogenous. Constructs operationalized in the BACH III Survey are listed in the ovals (conceptual domains).



**Figure 2. Final Structural Equation Model of Factors in the Pathway from Race/Ethnicity to T2D**

****

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
|  | Total Effect of Race/Ethnicity | Direct Effect of Race/Ethnicity | % Mediated by | | | | |
| Socioeconomic Factors | Environmental Factors | Psychosocial Factors | Lifestyle/ Behavioral Factors | Biophysiological Factors |
| Black | 0.29 | 0.18 | 21.8% | 1.3% | 0.0% | 11.21% | 4.6% |
| Hispanic | 0.18 | 0.10 | 26.2% | 4.3% | 5.0% | 5.77% | 4.6% |

**TABLES**

**Table 1.** Development of the “risk score”

|  |  |  |
| --- | --- | --- |
| **Domain/Variable** | **High Risk**  **(+1)** | **Very High Risk**  **(+2)** |
| **Socioeconomic** |  |  |
| Income | $20,000 - $49,999 | <$20,000 |
| Education | High school or equivalent/Some college | Less than High School |
| Occupation | Manual labor/Never worked |  |
| Born in the US | Yes |  |
| Acculturation | High/Bicultural (≥2.5 for English domain) |  |
| Health Literacy | Inadequate/Marginal |  |
| Insurance Status | Public |  |
| Visits to HCP in the past year | 7+ |  |
| **Neighborhood** |  |  |
| CT Poverty | > 20% |  |
| **Psychosocial** |  |  |
| Sleep Duration | < 6 hours | > 9 hours |
| Major Life Events | > 1 MLE |  |
| Sense of Control (tertiles) | < 1.0 | < 0.43 |
| **Lifestyle/Behavioral** |  |  |
| Physical activity | Low |  |
| Smoking history | Current |  |
| BMI[24](#_ENREF_49) | 25-29 | 30-39 (> 40 adds 3 risk points) |
| Waist circumference[36](#_ENREF_65) | ≥ 102 cm (men)  ≥ 88 cm (women) |  |
| Body fat percentage (tertiles) | > 25% (men)  > 35% (women) | > 33% (men)  > 42% (women) |
| **Biophysiological** |  |  |
| Blood pressure[36](#_ENREF_65) | SBP ≥ 130 or DBP ≥ 85 or self-report of hypertension diagnosis |  |
| Cholesterol (total)[37](#_ENREF_66) | ≥ 200 mg/dL | ≥ 240 mg/dL |
| HDL Cholesterol[36](#_ENREF_65) | < 40 mg/dL (men)  < 50 mg/dL (women) |  |
| Triglycerides[36](#_ENREF_65) | ≥ 150 |  |
| Cardiovascular disease |  | Yes |
| Menopausal status | Post | Surgical/Undetermined |
| Gestational diabetes |  | Yes |

**Table 2. Characteristics of the BACH III study population overall by diabetes status (N=2,476)**

|  | **Overall**  **N=2,764** | **Type 2 Diabetes**  **N=892** | **No Type 2 Diabetes**  **N=1872** | ***P*-value** |
| --- | --- | --- | --- | --- |
| Self-Identified Race/Ethnicity |  |  |  |  |
| Black | 929 (27.10%) | 351 (39.51%) | 578 (23.42%) | < 0.001 |
| Hispanic | 937 (12.20%) | 340 (12.55%) | 597 (12.09%) |  |
| White | 898 (60.71%) | 201 (47.94%) | 697 (64.48%) |  |
| Age1 |  |  |  |  |
| 34-44 | 405 (27.54%) | 61 (14.68%) | 344 (31.34%) | < 0.001 |
| 45-54 | 739 (26.97%) | 177 (23.07%) | 562 (28.13%) |  |
| 55-64 | 812 (19.99%) | 300 (24.12%) | 512 (18.77%) |  |
| 65-74 | 536 (13.76%) | 236 (21.46%) | 300 (11.49%) |  |
| 75-88 | 272 (11.74%) | 119 (16.67%) | 153 (10.28%) |  |
| Gender, % Male | 1018 (46.46%) | 344 (52.00%) | 674 (44.82%) | 0.056 |
| **Genetic Influences** |  |  |  |  |
| % West African, Mean (SE) | 29.84 (1.23) | 39.02 (2.27) | 27.12 (1.43) | < 0.001 |
| % Native American, Mean (SE) | 6.85 (0.29) | 6.53 (0.56) | 6.95 (0.36) | 0.545 |
| % European, Mean (SE) | 63.31 (1.27) | 54.45 (2.33) | 65.93 (1.49) | < 0.001 |
| Family History of Diabetes | 1483 (46.52%) | 602 (62.12%) | 882 (41.91%) | < 0.001 |
| **Socio-economic Influences** |  |  |  |  |
| Income |  |  |  |  |
| <$20,000 | 1234 (26.68%) | 524 (44.69%) | 710 (21.35%) | < 0.001 |
| $20,000 - $49,999 | 798 (25.10%) | 234 (25.63%) | 564 (24.94%) |  |
| ≥ $50,000 | 732 (48.22%) | 134 (29.67%) | 598 (53.70%) |  |
| Education |  |  |  |  |
| Less than High School | 560 (8.16%) | 278 (16.21%) | 282 (5.78%) | < 0.001 |
| High school or equivalent | 867 (24.44%) | 298 (32.72%) | 569 (21.99%) |  |
| Some college | 576 (21.17%) | 176 (23.79%) | 400 (20.39%) |  |
| College or advanced degree | 761 (46.23%) | 140 (27.28%) | 620 (51.84%) |  |
| Occupation |  |  |  |  |
| Professional, Managerial, Sales, and Office | 1324 (65.27%) | 345 (52.95%) | 979 (68.92%) | < 0.001 |
| Service | 715 (17.52%) | 224 (19.53%) | 492 (16.92%) |  |
| Manual labor | 495 (13.67%) | 209 (21.83%) | 286 (11.25%) |  |
| Never worked | 229 (3.54%) | 114 (5.70%) | 115 (2.90%) |  |
| Discrimination (0-45), Mean (SE) | 9.34 (0.25) | 10.31 (0.57) | 9.05 (0.29) | 0.057 |
| Born in US | 1645 (78.97%) | 488 (77.50%) | 1157 (79.41%) | 0.490 |
| Acculturation (English not First language) |  |  |  |  |
| Low | 669 (8.53%) | 253 (10.54%) | 416 (7.93%) | 0.178 |
| High/Bicultural | 2095 (91.47%) | 639 (89.46%) | 1456 (92.07%) |  |
| Health literacy |  |  |  |  |
| Inadequate | 708 (13.44%) | 328 (24.12%) | 380 (10.27%) | < 0.001 |
| Marginal | 298 (6.25%) | 120 (10.28%) | 178 (5.06%) |  |
| Adequate | 1759 (80.32%) | 445 (65.60%) | 1313 (84.67%) |  |
| **Environmental Influences** |  |  |  |  |
| CT SES |  |  |  |  |
| Low | 1269 (25.55%) | 447 (34.07%) | 822 (23.03%) | < 0.001 |
| Middle | 968 (39.87%) | 315 (40.65%) | 653 (39.64%) |  |
| High | 527 (34.58%) | 130 (25.29%) | 397 (37.33%) |  |
| CT Poverty |  |  |  |  |
| < 5% | 159 (10.45%) | 33 (6.84%) | 126 (11.52%) | 0.018 |
| 5-9.9% | 280 (14.37%) | 88 (12.22%) | 192 (15.01%) |  |
| 10-19.9% | 792 (35.78%) | 210 (32.94%) | 582 (36.62%) |  |
| ≥ 20% | 1533 (39.40%) | 561 (48.01%) | 972 (36.86%) |  |
| CT Racial Composition |  |  |  |  |
| % Black, Mean (SE) | 26.80 (1.07) | 32.61 (1.86) | 25.09 (1.18) | < 0.001 |
| % Hispanic, Mean (SE) | 16.62 (0.53) | 18.25 (0.88) | 16.14 (0.55) | 0.017 |
| % White, Mean (SE) | 53.75 (1.25) | 47.33 (2.06) | 55.65 (1.38) | < 0.001 |
| Property crime per 1,000, Mean (SE) | 74.05 (3.58) | 77.84 (4.28) | 72.93 (3.84) | 0.219 |
| Violent crime per 1,000, Mean (SE) | 6.35 (0.33) | 6.51 (0.37) | 6.30 (0.35) | 0.505 |
| Low Access to… (> 0.5 mi) |  |  |  |  |
| Supermarkets | 1316 (51.01%) | 415 (49.53%) | 901 (51.45%) | 0.529 |
| Grocery Stores | 251 (11.58%) | 69 (9.76%) | 182 (12.12%) | 0.370 |
| Convenience Stores | 164 (7.94%) | 52 (8.40%) | 112 (7.81%) | 0.616 |
| Fast Food | 882 (33.66%) | 269 (30.71%) | 613 (34.53%) | 0.578 |
| CT % Open Space, Mean (SE) | 0.08 (0.01) | 0.07 (0.01) | 0.08 (0.01) | 0.105 |
| Physical Disorder (6-30), Mean (SE) | 13.55 (0.14) | 13.82 (0.20) | 13.48 (0.18) | 0.191 |
| Social Disorder (6-30), Mean (SE) | 13.86 (0.16) | 14.58 (0.26) | 13.65 (0.18) | 0.003 |
| Years Lived at Current Address, Mean (SE) | 15.51 (0.48) | 17.17 (0.86) | 15.02 (0.56) | 0.031 |
| **Access to/Use of health care** |  |  |  |  |
| Distance to Community Health Center (Miles), Mean (SE) | 0.60 (0.03) | 0.56 (0.04) | 0.61 (0.03) | 0.200 |
| Distance to Acute Care Hospital (Miles), Mean (SE) | 1.24 (0.04) | 1.24 (0.06) | 1.24 (0.05) | 0.958 |
| Distance to Any Health Care Center (Miles) , Mean (SE) | 0.53 (0.03) | 0.49 (0.04) | 0.54 (0.03) | 0.326 |
| Usual Source of Care | 2714 (98.75%) | 880 (98.48%) | 1834 (98.82%) | 0.651 |
| Difficulty in Traveling to Health Care Provider |  |  |  |  |
| Very difficult | 54 (1.67%) | 22 (1.96%) | 32 (1.58%) | 0.171 |
| Somewhat difficult | 199 (6.62%) | 79 (9.26%) | 120 (5.84%) |  |
| Not too/Not at all Difficult | 477 (17.19%) | 181 (19.75%) | 296 (16.43%) |  |
| Not at all difficult | 2034 (74.52%) | 611 (69.02%) | 1423 (76.15%) |  |
| Insurance Status |  |  |  |  |
| Private | 1001 (51.41%) | 218 (33.84%) | 783 (56.61%) | < 0.001 |
| Public | 1671 (46.03%) | 654 (64.14%) | 1016 (40.67%) |  |
| None | 92 (2.56%) | 20 (2.02%) | 73 (2.72%) |  |
| Visits to Health Care Provider in the past year |  |  |  |  |
| 0-1 times | 395 (16.71%) | 74 (9.49%) | 321 (18.85%) | < 0.001 |
| 2-6 times | 1459 (51.96%) | 411 (49.12%) | 1048 (52.81%) |  |
| 7+ times | 910 (31.32%) | 407 (41.39%) | 503 (28.35%) |  |
| **Psychosocial** |  |  |  |  |
| Sleep Duration |  |  |  |  |
| < 6 hours | 622 (17.53%) | 259 (27.98%) | 363 (14.44%) | < 0.001 |
| 6-9 hours | 2097 (81.28%) | 617 (69.96%) | 1480 (84.62%) |  |
| > 9 hours | 45 (1.19%) | 17 (2.06%) | 28 (0.93%) |  |
| Major Life Events (0 to 10), Mean (SE) | 0.60 (0.03) | 0.68 (0.05) | 0.57 (0.03) | 0.080 |
| Sense of Control (-16 to 16), Mean (SE) | 0.72 (0.02) | 0.61 (0.03) | 0.76 (0.02) | < 0.001 |
| **Lifestyle/Behavioral Influences** |  |  |  |  |
| **Dietary Influences** |  |  |  |  |
| < 1500 mg Sodium | 615 (15.16%) | 216 (18.03%) | 399 (14.31%) | 0.144 |
| 3-4 Servings of Vegetables | 276 (12.47%) | 83 (9.84%) | 194 (13.25%) | 0.239 |
| 2-3 Servings of Fruit | 382 (18.09%) | 114 (15.60%) | 268 (18.83%) | 0.299 |
| 2-3 Servings of Meat/Beans | 588 (23.07%) | 187 (19.96%) | 401 (23.98%) | 0.204 |
| 6-11 Servings of Grain | 400 (18.99%) | 111 (14.22%) | 289 (20.39%) | 0.051 |
| 25-30 g of Fiber | 171 (7.17%) | 51 (5.28%) | 120 (7.73%) | 0.172 |
| < 14 g Saturated Fat | 1040 (29.19%) | 352 (31.29%) | 688 (28.58%) | 0.435 |
| FFQ Score (0-7), Mean (SE) | 1.24 (0.03) | 1.14 (0.07) | 1.27 (0.04) | 0.092 |
| Total Kcal, Mean (SE) | 1745.4 (32.02) | 1685.1 (79.47) | 1763.2 (34.47) | 0.370 |
| Physical activity |  |  |  |  |
| Low | 1132 (33.21%) | 480 (47.31%) | 652 (29.03%) | < 0.001 |
| Medium | 1286 (50.51%) | 337 (39.80%) | 949 (53.68%) |  |
| High | 346 (16.28%) | 76 (12.89%) | 270 (17.29%) |  |
| BMI, Mean (SE) | 29.42 (0.22) | 32.63 (0.42) | 28.47 (0.22) | < 0.001 |
| Waist Circumference (cm), Mean (SE) | 97.05 (0.54) | 106.58 (1.09) | 94.23 (0.55) | < 0.001 |
| Body Fat %, Mean (SE) | 33.96 (0.32) | 36.74 (0.57) | 33.13 (0.37) | < 0.001 |
| Smoking History |  |  |  |  |
| Never | 1220 (44.25%) | 373 (37.55%) | 847 (46.22%) | 0.014 |
| Former | 1015 (38.80%) | 346 (39.62%) | 669 (38.56%) |  |
| Current | 529 (16.95%) | 173 (22.83%) | 356 (15.21%) |  |
| **Biophysiological Influences** |  |  |  |  |
| SBP, Mean (SE) | 130.57 (0.61) | 138.83 (1.32) | 128.13 (0.66) | < 0.001 |
| DBP, Mean (SE) | 80.38 (0.37) | 81.86 (0.79) | 79.94 (0.43) | 0.034 |
| Total Cholesterol, Mean (SE) | 187.03 (1.29) | 176.10 (2.31) | 190.26 (1.45) | < 0.001 |
| HDL Cholesterol, Mean (SE) | 54.89 (0.68) | 50.39 (1.00) | 56.22 (0.82) | < 0.001 |
| Triglycerides, Mean (SE) | 129.05 (3.88) | 148.94 (5.87) | 123.17 (4.67) | < 0.001 |
| Hypertension | 2110 (70.16%) | 805 (89.44%) | 1305 (64.46%) | < 0.001 |
| Cardiovascular Disease | 604 (16.09%) | 315 (32.66%) | 289 (11.19%) | < 0.001 |
| **Women Only** |  |  |  |  |
| Menopausal Status |  |  |  |  |
| Pre/Peri- menopause | 437 (36.63%) | 67 (16.14%) | 370 (41.90%) | < 0.001 |
| Post-menopause | 740 (36.79%) | 241 (40.54%) | 499 (35.83%) |  |
| Undetermined/Other | 569 (26.57%) | 240 (43.33%) | 329 (22.27%) |  |
| Gestational Diabetes | 125 (5.93%) | 72 (17.07%) | 54 (3.07%) | < 0.001 |
|  |  |  |  |  |

1 n and column percent presented for categorical variables, *P-*value from a chi-squared test

2 Mean and standard error presented for continuous variables, *P-*value from a t-test

CT= Census Tract

**eAPPENDIX A**

**Measures**

***Race/ethnicity***

Race/ethnicity was self-reported by survey participants according to two separate survey questions: “Do you consider yourself to be Spanish, Hispanic, or Latino (Latina)?” and “What do you consider yourself to be? Select one or more of the following” with response categories of American Indian or Alaska Native, Asian, Black or African American, Native Hawaiian or other Pacific Islander, White or Caucasian, and Other (Specify). These questions are the standard used in the United States as recommended by the Office of Management and Budget.[38](#_ENREF_67) The racial/ethnic labels used in this research are 1) non-Hispanic Black (black), 2) Hispanic of any race (Hispanic), and 3) non-Hispanic White (white).

***Socio-economic Influences***

*Socioeconomic Status (SES)*

The individual SES indicators considered were: household income, educational attainment and occupation. Household income, originally grouped into 12 ordinal categories, was collapsed into the following three categories of US dollars: <20,000, 20-49,999, and ≥50,000. Educational attainment was categorized as less than high school, high school graduate or equivalent, some college, and college or advanced degree were combined due to smaller numbers. Current or former occupationwas categorized into four groups: (1) management, professional, sales and office occupations; (2) service occupations; (3) manual labor which includes construction, maintenance, farming, production, and transportation occupations; and (4) never worked. We use the broader term ‘SES’ when referring to these three distinct socioeconomic factors in the aggregate, all of which are strongly related to overall health.[39](#_ENREF_68)

*Discrimination*

We measured perceptions of everyday discrimination using the Every Discrimination Scale (Short Version), a five item scale that attempts to measure chronic and/or routine experiences of unfair treatment without direct reference to the influence of race and a four item scale that measures the influence .[11](#_ENREF_32) Metrics on this scale have been published previously.[11](#_ENREF_32) The five items capture the frequency of the following experiences in the day-to-day lives of the participants: being treated with less courtesy than others; less respect than others; receiving poorer service than others in restaurants or stores; people acting as if you are not smart; they are afraid of you; and being threatened or harassed. Scores for each item ranged from 0 to 5 with 0 indicating the event never occurred and 5 indicating an almost every day occurrence. Scores for the total scale ranged from 0 to 45, with a high score indicating greater perceived discrimination.

*Acculturation*

The Bidimensional Acculturation Scale (BAS) was used to assess the extent to which the individuals participate in the cultural domains of both their original culture and the culture of contact. The domains assessed are language use, language proficiency, and electronic media. The BAS has been tested in several Hispanic populations (Central and Mexican Americans populations) and correlates well with generational status, length of residence in the US, and ethnic self-identification.[12](#_ENREF_33)

*Health Literacy*

To measure health literacy, we used the abbreviated form of the s-TOFHLA, Spanish or English version.[40](#_ENREF_34) The abbreviated s-TOFHLA is a 36-item, timed reading comprehension test. Every fifth to seventh word in a reading passage is omitted, and 4 multiple-choice options are

provided. The abbreviated s-TOFHLA contains two health care passages. The abbreviated s-TOFHLA is scored on a scale of 0 to 36. Using established convention, we categorized patients

as having inadequate health literacy if the s-TOFHLA score was 0 to 16, marginal health literacy if it was 17 to 22, and adequate health literacy if it was 23 to 36.

***Environmental Influences***

*Neighborhood Socioeconomic Status (SES)*

Census-tract (neighborhood) SES was based on methods used by Diez-Roux. [13](#_ENREF_35) A composite index Z-score was created for census tract based on six measures including: log median household income; log median value of owner occupied housing; percent of household receiving interest, dividend or net rental income; percent of adults 25 and over with high school degree; percent of adults 25 and over with a college degree; and percent of individuals ages 16 and over in management and professional occupations. An increasing score signifies increasing neighborhood socioeconomic advantage. Census tracts were designated as low, middle, or high SES according to the tertiles of the z-score.[41](#_ENREF_69)

*Safety*

Crime incident reports provided by the Boston Police Department were downloaded from the City of Boston website at <https://data.cityofboston.gov/>. Data was coded using conventions described by others.[42](#_ENREF_70) The property crime rates in 2010-2011 were calculated as the number of offenses of burglary, larceny, and motor vehicle theft per 1,000 population. Violent crime was calculated as murder, robbery, and assault (including sexual assault) per 1,000 population.

*Built Environment*

We used ArcGIS (Environmental Systems Research Institute, Redlands, CA) to draw circular buffers with 3 different radii (0.25mi, 0.5mi, and 1.0 miles) centered at each research participant’s residence. A distance of 0.25 mi is approximately a 5 minute walk which has been estimated to be the average distance walked to a grocery store in large metropolitan cities with available public transit.[43](#_ENREF_71) The food stores within each radii were categorized as supermarkets, grocery stores, fast food, and convenience stores. Information on food establishments located in the Boston metro area were purchased from InfoUSA Inc, a proprietary information service. Supermarkets and grocery stores were identified by a primary SIC code of 541105. Following precedents set by previous work,[44](#_ENREF_72) supermarkets were differentiated from grocery stores on the basis of chain name recognition or annual payroll of greater than 50 employees. Convenience stores were identified as businesses with a primary or secondary SIC code of 541102 or 541103. Following standards set by other researchers[45](#_ENREF_73) fast food restaurants including restaurants, delicatessens, pizza shops and coffee shops (SIC codes: 581206,07,08,09,14,19,22,24,28) had to meet the following criteria: 1) be a franchised vendor, 2) ability to purchase food without wait staff, and 3) sale of both food and beverage. To ensure that our database was both comprehensive and appropriate for Boston, we gathered information on fast-food restaurants in the city from several other sources. In addition to the InfoUSA list, we also referred to a privately run Web site, Fast Food Source (http://www.fastfoodsource.com), to identify fast-food restaurants in Boston. We thus defined food environment exposures into three category measures defined as high access (<0.25 miles), medium (0.25-0.50 miles), and low access (>0.50 miles) to convenience stores, grocery stores, supermarkets, and fast food.

*Neighborhood disorder*

Social and physical neighborhood order and disorder were measured using the “Perceived Neighborhood Disorder” scale developed by Ross and Mirowsky.[14](#_ENREF_37) Social disorder refers to people hanging around on the streets, drug and alcohol use, trouble with neighbors, and a general perception of lack of safety. Physical disorder refers to graffiti, vandalism, abandoned buildings, cleanliness, and maintenance of homes and apartments. The physical and social disorder indices were created by reverse coding “order” items and summing the six items in each subscale with higher scores indicating higher perceived disorder.46, 47 Number of years at current address was used to assess residential mobility and was considered as a potential mediator between neighborhood determinants and downstream health effects.

The locations of community health centers and acute care hospitals were obtained from the Massachusetts Office of Geographic Information (MassGIS).[48](#_ENREF_36) Distance to the closest community health center, acute care hospital, and community health center or hospital were calculated in miles. In addition, we assessed whether the participants had a usual source of care (Yes/No) and their perceived difficulty in getting to their primary care provider (Very/Somewhat/Not too/Not at all difficult).

***Psychosocial Influences***

*Sleep*

Hours of sleep each night was captured continuously and categorized as < 6, 6-9, >9 hours over the referent period of the past month.

*Sense of Control*

The Mirowsky and Ross sense of control index[16](#_ENREF_39) contains 8 items that assess internal sense of control over positive and negative outcomes (e.g. “I am responsible for my own successes” [positive] and “I am responsible for my failures” [negative]) as well as a sense of powerlessness over positive and negative outcomes (e.g. “the really good things that happen to me are mostly luck” [positive] and “most of my problems are due to bad breaks” [negative]). This 2x2 design eliminates defense and agreement bias from the measure. All items are coded -2 to 2 (external items are reverse coded). The sense of control score is calculated as the sum of the responses to the 8 items, and ranges from maximally denying (−16) to maximally claiming control (+16). Metrics for this scale on black and white populations indicate high test-retest reliability and robust confirmatory factor analysis validation. 46, 49

***Lifestyle/behavioral influences***

*Dietary patterns*

Participants completed the self-administered Block Food Frequency Questionnaire (FFQ) in English or Spanish. This FFQ has been validated to obtain data on usual dietary intake over the past year.[17](#_ENREF_40) Based upon the USDA and AHA guidelines for healthy eating,[50](#_ENREF_77) we calculated a healthy eating score composed of FFQ data on average daily intake of sodium, vegetables, fruits, meats/beans, grains, fiber and saturated fat.The healthy eating score was adjusted for total kilocalories.

**eTable 1. Results from logistic regression models (each potential variable added one at a time to race/ethnicity, age, and gender model)**

|  | **Odds Ratio (OR)**  **(95% CI)** | **p-value** | **Black vs. White**  **OR (95% CI)1** | **Hispanic vs. White**  **OR (95% CI)1** | Score Assigned |
| --- | --- | --- | --- | --- | --- |
| **Base Model (age, gender adjusted)** |  |  | **2.86 (1.96, 4.19)** | **1.98 (1.34, 2.94)** |  |
| **Genetic Influences** |  |  |  |  |  |
| West African Ancestry | 1.02 (0.92, 1.14) | 0.658 | **2.44 (1.09, 5.46)** | **2.10 (1.18, 3.76)** | N/A2 |
| Native American Ancestry | 0.94 (0.79, 1.10) | 0.428 | **2.44 (1.09, 5.46)** | **2.10 (1.18, 3.76)** | N/A2 |
| Family History of Diabetes | 2.11 (1.54, 2.90) | <0.001 | **2.51 (1.71, 3.69)** | **1.80 (1.21, 2.69)** | N/A2 |
| **Socio-economic Influences** |  |  |  |  |  |
| Income |  |  | **2.30 (1.56, 3.39)** | **1.34 (0.87, 2.06)** |  |
| <$20,000 | 3.13 (2.08, 4.71) | < 0.001 |  |  | 2 |
| $20,000-$49,999 | 1.63 (1.06, 2.51) |  |  |  | 1 |
| ≥ $50,000 | Reference |  |  |  | 0 |
| Education |  |  | **1.94 (1.30, 2.88)** | **1.06 (0.68, 1.65)** |  |
| Less than high school | 4.56 (2.74, 7.61) | < 0.001 |  |  | 2 |
| High school or GED | 2.60 (1.75, 3.86) |  |  |  | 1 |
| Some college | 1.93 (1.22, 3.05) |  |  |  | 1 |
| College or advanced degree | Reference |  |  |  | 0 |
| Occupation |  |  | **2.59 (1.77, 3.80)** | **1.54 (1.01, 2.35)** |  |
| Professional | Reference | 0.005 |  |  | 0 |
| Service | 1.34 (0.91, 1.99) |  |  |  | 0 |
| Manual labor | 1.78 (1.15, 2.76) |  |  |  | 1 |
| Never worked | 2.23 (1.28, 3.89) |  |  |  | 1 |
| Discrimination (log transformed) | 1.03 (0.89, 1.21) | 0.667 | 2.82 (1.92, 4.16) | 2.00 (1.35, 2.96) | -- |
| Born in US (Yes vs. No) | 1.10 (0.68, 1.76) | 0.705 | 2.89 (1.95, 4.27) | **2.14 (1.20, 3.79)** | 1\* |
| Acculturation |  |  | 2.88 (1.96, 4.21) | **1.68 (0.94, 2.99)** |  |
| Low | Ref | 0.396 |  |  | 0 |
| High/Bicultural | 1.35 (0.67, 2.72) |  |  |  | 1\* |
| Health Literacy |  |  | **2.46 (1.67, 3.63**) | **1.40 (0.90, 2.18)** |  |
| Inadequate | 2.06 (1.33, 3.19) | 0.002 |  |  | 1 |
| Marginal | 1.81 (1.10, 2.96) |  |  |  | 1 |
| Adequate | Ref |  |  |  | 0 |
| **Access to/Use of health care** |  |  |  |  |  |
| Insurance Status |  |  | **2.56 (1.74, 3.77)** | **1.71 (1.14, 2.56)** |  |
| Private | 0.87 (0.33, 2.29) | 0.004 |  |  | 0 |
| Public | 1.69 (0.63, 4.54) |  |  |  | 1 |
| Other | Ref |  |  |  | 0 |
| Visits to HCP in the past year |  |  | 2.94 (2.00, 4.33) | 2.06 (1.39, 3.06) |  |
| 0-1 | 0.37 (0.23, 0.62) | < 0.001 |  |  | 0 |
| 2-6 | 0.62 (0.45, 0.86) |  |  |  | 0 |
| 7+ | Ref |  |  |  | 1 |
| **Environmental Influences (random intercept models)** |  |  | **2.89 (2.08, 4.01)** | **2.53 (1.86, 3.45)** |  |
| CT SES |  |  | 2.71 (1.91, 3.83) | 2.39 (1.72, 3.30) |  |
| Low | 1.27 (0.89, 1.82) | *0.193* |  |  | -- |
| Middle | 1.18 (0.83, 1.68) | *0.356* |  |  | -- |
| High | *Reference* |  |  |  | -- |
| CT Poverty |  |  | **2.63 (1.84, 3.75)** | **2.29 (1.65, 3.19)** |  |
| < 5% | *Reference* |  |  |  | 0 |
| 5-9.9% | 1.73 (0.88, 3.39) | *0.109* |  |  | 0 |
| 10-19.9% | 1.15 (0.62, 2.13) | 0.648 |  |  | 0 |
| ≥ 20% | 1.72 (0.93, 3.18) | 0.086 |  |  | 1 |
| CT Racial Composition |  |  |  |  |  |
| % Black (log transformed) | 1.04 (0.92, 1.18) | 0.494 | 2.73 (1.86, 4.02) | 2.45 (1.76, 3.41) | -- |
| % White (log transformed) | 0.95 (0.82, 1.10) | 0.484 | 2.74 (1.87, 4.00) | 2.45 (1.77, 3.40) | -- |
| % Hispanic (log transformed) | 1.09 (0.92, 1.29) | 0.324 | 2.82 (2.02, 3.92) | 2.42 (1.75, 3.35) | -- |
| Property crime (log transformed) | 1.11 (0.86, 1.43) | 0.419 | 2.82 (2.02, 3.94) | 2.48 (1.81, 3.40) | -- |
| Violent crime (log transformed) | 1.16 (0.94, 1.43) | 0.169 | 2.72 (1.93, 3.82) | 2.42 (1.76, 3.33) | -- |
| Access to Supermarkets |  |  | 2.91 (2.10, 4.03) | 2.53 (1.86, 3.45) | -- |
| Low Access (> 0.5 miles) | 0.76 (0.53, 1.08) | 0.169 |  |  | -- |
| Medium Access (0.25-0.5 miles) | 0.77 (0.52, 1.12) | 0.169 |  |  | -- |
| High Access (< 0.25 miles) | *Reference* |  |  |  | -- |
| Access to Grocery Stores |  |  | 2.90 (2.09, 4.03) | 2.50 (1.83, 3.41) | -- |
| Low Access (> 0.5 miles) | 0.69 (0.43, 1.10) | 0.121 |  |  | -- |
| Medium Access (0.25-0.5 miles) | 0.88 (0.68, 1.14) | 0.331 |  |  | -- |
| High Access (< 0.25 miles) | *Reference* |  |  |  | -- |
| Access to Convenience Stores |  |  | 2.89 (2.08, 4.01) | 2.50 (1.83, 3.42) | -- |
| Low Access (> 0.5 miles) | 0.94 (0.56, 1.56) | 0.804 |  |  | -- |
| Medium Access (0.25-0.5 miles) | 0.81 (0.60, 1.09) | 0.159 |  |  | -- |
| High Access (< 0.25 miles) | *Reference* |  |  |  | -- |
| Access to Fast Food |  |  | 2.93 (2.11, 4.06) | 2.54 (1.87, 3.45) | -- |
| Low Access (> 0.5 miles) | 0.80 (0.59, 1.08) | 0.15 |  |  | -- |
| Medium Access (0.25-0.5 miles) | 0.95 (0.72, 1.26) | 0.739 |  |  | -- |
| High Access (< 0.25 miles) | *Reference* |  |  |  | -- |
| CT % Open Space |  |  | 2.85 (2.05, 3.95) | 2.52 (1.85, 3.43) | -- |
| ≤ 5% | 1.14 (0.73, 1.78) | 0.57 |  |  | -- |
| 5.1-10% | 0.94 (0.57, 1.53) | 0.798 |  |  | -- |
| 10.1-20% | 1.11 (0.67, 1.85) | 0.678 |  |  | -- |
| > 20% | *Reference* |  |  |  | -- |
| Neighborhood Disorder |  |  | 2.78 (2.00, 3.87) | 2.45 (1.80, 3.35) |  |
| Social (log transformed) | 1.09 (0.58, 2.05) | 0.79 |  |  | -- |
| Physical (log transformed) | 1.37 (0.79, 2.38) | 0.26 |  |  | -- |
| Years Lived at Current Address (log transformed) | 0.90 (0.78, 1.04) | 0.16 | 2.79 (2.01, 3.88) | 2.39 (1.74, 3.29) | -- |
| Access to Community Health Center (miles) | 0.96 (0.66, 1.41) | 0.851 | 2.84 (1.94, 4.15) | 1.97 (1.32, 2.93) | -- |
| **Psychosocial Influences** |  |  |  |  |  |
| Sleep Duration |  |  | **2.57 (1.74, 3.80)** | **1.83 (1.21, 2.76)** |  |
| < 6 | 2.07 (1.45, 2.96) | < 0.001 |  |  | 1 |
| 6-9 | Ref |  |  |  | 0 |
| > 9 | 3.32 (0.92, 11.95) |  |  |  | 2 |
| Major Life Events (log transformed) | 1.18 (0.83, 1.69) | 0.349 | 2.82 (1.93, 4.11) | 1.97 (1.32, 2.92) | 1 (> 1 MLE) |
| Sense of Control | 0.63 (0.45, 0.87) | 0.006 | 2.83 (1.93, 4.14) | **1.68 (1.10, 2.56)** | 1 (< 1.0), 2 (< 0.43) |
| **Lifestyle/Behavioral Influences** |  |  |  |  |  |
| Dietary Influences2 |  |  |  |  |  |
| < 1500 mg Sodium | 0.91 (0.55, 1.51) | 0.702 | 2.84 (1.93, 4.17) | 1.94 (1.30, 2.88) | -- |
| 3-4 Servings of Vegetables | 0.72 (0.39, 1.33) | 0.293 | 2.80 (1.91, 4.12) | **1.87 (1.25, 2.79)** | -- |
| 2-3 Servings of Fruit | 0.96 (0.61, 1.51) | 0.845 | 2.81 (1.90, 4.14) | 1.91 (1.28, 2.84) | -- |
| 2-3 Servings of Meat/Beans | 0.87 (0.58, 1.28) | 0.470 | 2.81 (1.91, 4.13) | 1.92 (1.30, 2.85) | -- |
| 6-11 Servings of Grain | 0.77 (0.47, 1.27) | 0.301 | 2.78 (1.88, 4.10) | 1.91 (1.28, 2.85) | -- |
| 25-30 g of Fiber | 0.82 (0.44, 1.53) | 0.529 | 2.80 (1.91, 4.11) | 1.92 (1.29, 2.85) | -- |
| < 14 g Saturated Fat | 0.84 (0.55, 1.28) | 0.422 | 2.84 (1.93, 4.18) | 1.96 (1.33, 2.90) | -- |
| FFQ Score (log) | 0.70 (0.48, 1.03) | 0.068 | 2.80 (1.90, 4.11) | 1.97 (1.33, 2.91) | -- |
| Total Kcal (log) | 0.87 (0.65, 1.17) | 0.357 | 2.82 (1.92, 4.14) | 1.92 (1.29, 2.85) | -- |
| Physical activity |  |  | 2.83 (1.92, 4.15) | 1.91 (1.28, 2.84) |  |
| Low | 1.77 (0.99, 3.17) | 0.004 |  |  | 1 |
| Medium | 1.00 (0.59, 1.72) |  |  |  | 0 |
| High | Ref |  |  |  | 0 |
| Smoking status |  |  | 2.73 (1.87, 3.99) | 1.96 (1.32, 2.91) |  |
| Never | Ref | 0.014 |  |  | 0 |
| Former | 1.03 (0.73, 1.47) |  |  |  | 0 |
| Current | 1.81 (1.19, 2.75) |  |  |  | 1 |
| BMI (log transformed) | 31.42 (14.07, 70.15) | < 0.001 | **2.51 (1.68, 3.77)** | **1.88 (1.25, 2.84)** | 1 (25-29), 2 (30-39), 3 (> 40) |
| Waist Circumference | 1.05 (1.04, 1.06) | < 0.001 | 2.78 (1.86, 4.15) | **2.54 (1.66, 3.87)** | 1 (≥ 102 cm, men; ≥ 88 cm, women) |
| Body Fat % | 1.06 (1.04, 1.09) | < 0.001 | **2.54 (1.70, 3.80)** | 1.91 (1.28, 2.87) | 1 (> 25%, men; > 35% women), 2 (> 33%, men; > 42%, women) |
| **Biophysiological Influences** |  |  |  |  |  |
| SBP | 1.02 (1.01, 1.03) | < 0.001 | **2.50 (1.70, 3.70)** | 1.92 (1.29, 2.87) | 1 (SBP ≥ 130) |
| DBP | 1.01 (1.00, 1.03) | 0.101 | 2.73 (1.86, 4.00) | 1.97 (1.33, 2.93) | 1 (DBP ≥ 85) |
| Total Cholesterol | 0.99 (0.98, 0.99) | < 0.001 | 3.01 (2.05, 4.41) | 2.05 (1.37, 3.06) | 1 (≥ 200 mg/dL) |
| HDL Cholesterol | 0.97 (0.96, 0.99) | < 0.001 | 3.00 (2.04, 4.41) | **1.83 (1.22, 2.75)** | 1 (< 40 mg/dL, men; < 50 mg/dL women) |
| Triglycerides | 1.00 (1.00, 1.00) | < 0.001 | **3.12 (2.14, 4.55)** | 1.97 (1.31, 2.95) | 1 (≥ 150) |
| Hypertension | 3.21 (1.98, 5.19) | < 0.001 | **2.41 (1.64, 3.55)** | 1.90 (1.26, 2.85) | 1 |
| Cardiovascular Disease | 2.86 (1.99, 4.12) | < 0.001 | 2.72 (1.84, 4.01) | 1.92 (1.28, 2.88) | 2 |
| **Women Only** |  |  | **3.09 (1.88, 5.09)** | **2.46 (1.48, 4.10)** |  |
| Menopausal Status |  |  | **2.83 (1.71, 4.70)** | 2.34 (1.39, 3.93) |  |
| Pre/Peri | Reference | 0.017 |  |  | 0 |
| Post | 2.14 (0.95-4.81) |  |  |  | 1 |
| Undetermined/Unknown | 3.11 (1.36-7.11) |  |  |  | 2 |
| Gestational Diabetes | 8.46 (4.03-17.74) | <0.001 | **3.34 (1.99, 5.62)** | **2.28 (1.32, 3.93)** | 2 |

1 **Bold** indicates racial/ethnic effect was reduced by ≥ 10%

2 Genetic factors were treated as exogenous (not predicted by any other variables in the model) variables and therefore not incorporated into the mediating risk scores

3 All dietary influences are also adjusted for total kcal

\* Point was added to risk score due to mediation of the effect of race/ethnicity rather than a direct effect on T2D

**eTABLE 2. Final Structural Equation Model of Factors in the Pathway from Race/Ethnicity to T2D (full standardized and non-standardized results)**

| Predictor | Outcome | Standardized Coefficient | Unstandardized Coefficient | *p*-value |
| --- | --- | --- | --- | --- |
| Black vs. White | Socioeconomic Risk | 0.226 | 0.969 | 0.003 |
| Hispanic vs. White | Socioeconomic Risk | 0.173 | 0.900 | <0.001 |
| West African ancestry | Socioeconomic Risk | 0.105 | 0.057 | 0.135 |
| Native American ancestry | Socioeconomic Risk | 0.008 | 0.013 | 0.801 |
| % Black participants1 | Environmental Risk | 0.140 | 0.269 | 0.001 |
| % Hispanic participants1 | Environmental Risk | 0.289 | 0.689 | <0.001 |
| West African ancestry | Environmental Risk | ND2 |  |  |
| Native American ancestry | Environmental Risk | ND2 |  |  |
| Socioeconomic Risk | Environmental Risk | 0.410 | 0.225 | <0.001 |
| Black vs. White | Psychosocial Risk | 0.003 | 0.006 | 0.965 |
| Hispanic vs. White | Psychosocial Risk | 0.173 | 0.496 | <0.001 |
| West African ancestry | Psychosocial Risk | 0.031 | 0.009 | 0.653 |
| Native American ancestry | Psychosocial Risk | 0.023 | 0.019 | 0.477 |
| Socioeconomic Risk | Psychosocial Risk | 0.391 | 0.207 | <0.001 |
| Environmental Risk | Psychosocial Risk | 0.009 | 0.008 | 0.903 |
| Black vs. White | Lifestyle/behavioral Risk | 0.113 | 0.451 | 0.081 |
| Hispanic vs. White | Lifestyle/behavioral Risk | 0.037 | 0.178 | 0.369 |
| West African ancestry | Lifestyle/behavioral Risk | -0.034 | -0.017 | 0.605 |
| Native American ancestry | Lifestyle/behavioral Risk | -0.034 | -0.051 | 0.312 |
| Socioeconomic Risk | Lifestyle/behavioral Risk | 0.312 | 0.291 | <0.001 |
| Environmental Risk | Lifestyle/behavioral Risk | 0.114 | 0.165 | 0.178 |
| Psychosocial Risk | Lifestyle/behavioral Risk | 0.008 | 0.014 | 0.785 |
| Black vs. White | Biophysiologic Risk | 0.069 | 0.259 | 0.264 |
| Hispanic vs. White | Biophysiologic Risk | 0.044 | 0.200 | 0.283 |
| West African ancestry | Biophysiologic Risk | -0.072 | -0.034 | 0.218 |
| Native American ancestry | Biophysiologic Risk | 0.015 | 0.021 | 0.602 |
| Socioeconomic Risk | Biophysiologic Risk | 0.136 | 0.120 | <0.001 |
| Environmental Risk | Biophysiologic Risk | 0.083 | 0.116 | 0.297 |
| Psychosocial Risk | Biophysiologic Risk | 0.049 | 0.082 | 0.069 |
| Lifestyle/behavioral risk | Biophysiologic Risk | 0.205 | 0.194 | <0.001 |
| Family history | Biophysiologic Risk | 0.047 | 0.169 | 0.056 |
| Black vs. White | T2D | 0.177 | 0.875 | 0.054 |
| Hispanic vs. White | T2D | 0.100 | 0.600 | 0.069 |
| West African ancestry | T2D | -0.003 | -0.002 | 0.976 |
| Native American ancestry | T2D | -0.016 | -0.029 | 0.725 |
| Socioeconomic Risk | T2D | 0.132 | 0.152 | 0.003 |
| Environmental Risk | T2D | -0.022 | -0.037 | 0.861 |
| Psychosocial Risk | T2D | 0.039 | 0.085 | 0.283 |
| Lifestyle/behavioral risk | T2D | 0.248 | 0.306 | <0.001 |
| Family history | T2D | 0.102 | 0.485 | 0.005 |
| Biophysiologic Risk | T2D | 0.192 | 0.251 | <0.001 |

1 The % of black and Hispanic participants within each census tract were used to predict neighborhood-level outcomes

2 ND = Not determined, model would not converge with this path present