EPIDEMIOLOGY/HEALTH SERVICES RESEARCH

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

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OBJECTIVE

Racial/ethnic minorities in the U.S. have a higher prevalence of type 2 diabetes mellitus (T2DM) than white adults. While many independent risk factors for T2DM have been identified, these determinants are often viewed in isolation without considering the joint contributions of competing risk factors. The objective of this study was to assess the relative contributions of six domains of influence to racial/ethnic disparities in T2DM.

RESEARCH DESIGN AND METHODS

Cross-sectional analyses were conducted using the Boston Area Community Health III Survey (2010–2012), the third wave of a population-based sample of men and women from three racial/ethnic groups (black, Hispanic, white) living in Boston, Massachusetts (*N* = 2,764). Prevalent diabetes was defined by self-report of T2DM, fasting glucose >125 mg/dL, or HbA_{1c} ≥6.5%. Structural equation models were constructed to evaluate the direct effects of each conceptual domain of influence on T2DM prevalence, as well as their indirect effects on the race/ethnicity–T2DM relationship. All direct and indirect pathways were included.

RESULTS

The final model indicated that 38.9% and 21.8% of the total effect of black race and Hispanic ethnicity, respectively, on T2DM prevalence was mediated by the socioeconomic, environmental, psychosocial, and lifestyle/behavioral risk scores. The largest mediating influence was the socioeconomic risk score, which explained 21.8% and 26.2% of the total effect of black race and Hispanic ethnicity, respectively.

CONCLUSIONS

Our study found that socioeconomic factors had the greatest impact on explaining the excess prevalence of T2DM among racial/ethnic minorities.

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Disparities in type 2 diabetes mellitus (T2DM) by race/ethnicity are an important public health problem in the United States and worldwide. Compared with white adults, the prevalence of diabetes is 77% higher among black and 66% higher among Hispanic adults in the U.S. (1). Racial/ethnic disparities in T2DM are associated with disparities in diabetes control (2), elevated rates of diabetes-related complications, and higher health care costs (3).

Many factors have been identified as contributing to these disparities (4), including variations in lifestyles and behaviors, biophysiological, psychosocial, sociodemographic, and environmental factors and biogeographic ancestry (BGA) (5,6). Research to date, however, has largely focused on individual risk factors in isolation, and the relative contribution of these influences have not been identified (4).

Since racial/ethnic differences in T2DM seem to result from a broad range of influences, a more complete understanding requires a multilevel approach. A multilevel risk model, reflecting the many factors that contribute to T2DM risk, may advance understanding and better inform the design of interventions to target the most relevant domains that disproportionately contribute to disparities.

The aim of this research is to assess the relative contributions of six domains of influence to racial/ethnic disparities in T2DM: *1*) socioeconomic, *2*) local environmental, *3*) psychosocial, *4*) lifestyle/behavioral, *5*) biophysiological, and *6*) BGA. To address this aim we developed a conceptual model within a population health/causal modeling framework (Fig. 1). This model identifies distal, intermediate, and proximate factors that influence T2DM. Distal factors, which may be population-level determinants, include individual-level social conditions (i.e., socioeconomic factors, racial/ethnic discrimination). The intermediate determinants of T2DM include neighborhood- or community- level physical (i.e., food environment, open space) and social (i.e., crime, disorder) environments. Proximate determinants of T2DM include biophysiological and genetic factors and individual health behaviors. These influences are hypothesized to directly and/or indirectly, singly and in combination, affect T2DM. For example, household income (socioeconomic) could contribute to the development of T2DM through the availability of healthy foods and places to exercise (environmental), which in turn can influence individual behaviors (lifestyle). Socioeconomic factors could also directly affect health behaviors via material constraints, limited knowledge, and/or limited opportunities to act upon health-promoting messages.

RESEARCH DESIGN AND METHODS

Study Sample

The Boston Area Community Health (BACH) Survey is a longitudinal cohort of 5,502 residents (2,301 men and 3,201 women) aged 30–79 years from three racial/ethnic groups in Boston, MA (7,8). The sampling strategy for the BACH Survey has been published previously (7). Briefly, to ensure a representative sample, a stratified, two-stage cluster sampling design was used, with census blocks as the primary sampling units and households as the secondary sampling units. Census blocks were stratified by minority density. High minority strata were oversampled to attain a sample with roughly one-third black, one-third Hispanic, and one-third white participants. BACH has conducted a total of three surveys to date: BACH I (2002–2006; N = 5,502), BACH II (2008–2010; N = 4,144), and BACH III (2010–2012; N = 3,155). This analysis uses cross-sectional data from BACH III. Only participants who had a geocodable address (99.9%) and who resided in the city of Boston during BACH III were included in the analysis because of the availability of neighborhood-level parameters, leaving 2,764 subjects. The response rate, dependent on previous participation, was 81.4%. Retention rates increased slightly with older age. Retention was not significantly related to race/ethnicity or socioeconomic status (SES) (8). Participants who moved were more likely to be white (vs. black), be younger, and have a higher income. Survey participants were interviewed in the morning after fasting overnight ($\geq$8 h) and after providing written informed consent. The interviews were conducted by trained, certified phlebotomists fluent in English and/or Spanish. The study was approved by the New England Research Institutes' Institutional Review Board.

Measures

The primary determinant of interest was self-identified 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). The racial/ethnic categories used in this research are *1*) non-Hispanic black (black), *2*) Hispanic of any race (Hispanic), and *3*) non-Hispanic white (white).

The primary outcome was prevalent T2DM. Fasting glucose (FG) was measured with a HemoCue 201 point-of-care analyzer. HbA_{1c} was measured by Quest Laboratories (Cambridge, MA). Participants who *1*) self-reported T2DM ("Have you ever been told by a doctor or other health professional that you have type 2 diabetes?"), or *2*) had FG $>$125 mg/dL or HbA_{1c} $\geq$6.5% were classified as having T2DM. Medication inventory and age at diagnosis were used to further separate type 1 diabetes versus T2DM. Eight individuals younger than 35 years at diagnosis and receiving continuous insulin therapy were considered to have type 1 diabetes and were excluded. The medication inventory confirmed over 80% of the self-reported cases of diabetes.

Six constructs were measured within each domain of our theoretical model (Fig. 1). Socioeconomic influences considered included household income, educational attainment, occupation, perceptions of everyday discrimination (9), born in the U.S. (yes/no), acculturation (10), health literacy, type of health insurance, and number of visits to a health care provider in the past year. ArcGIS 10.1 (ESRI, Redlands, CA) was used to geocode participants' residences and link participants with geographic features. Environmental influences considered included SES by census tract (11); percent living in poverty; percent non-Hispanic black or non-Hispanic white race/ethnicity; violent and property crime per 1,000 population; distance to the closest grocery store, convenience store, and fast food (miles); amount of recreational open space; perceived social and physical disorder (12); and number of years at the current address. Spatial access to health care was assessed by distance to the closest community health

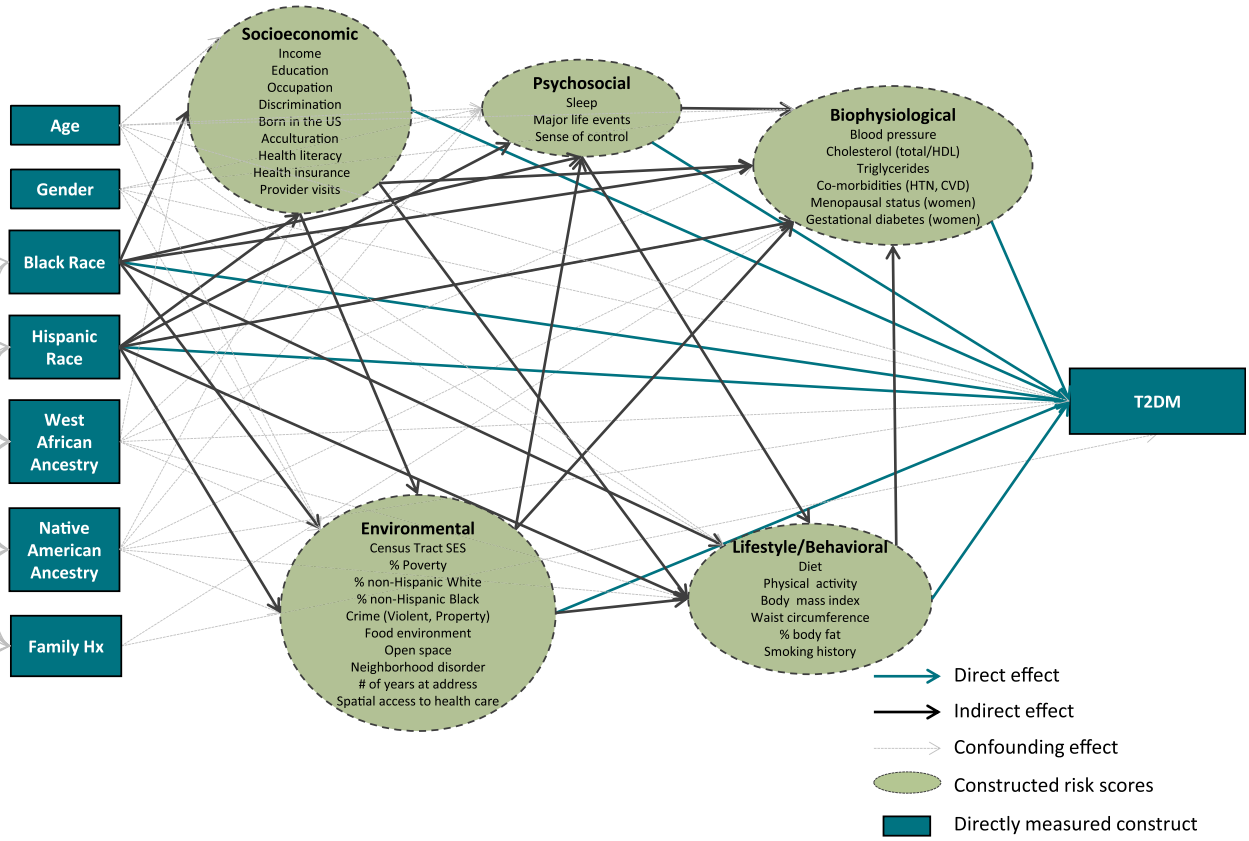


Figure 1—Conceptual model of potential factors influencing racial/ethnic disparities in T2DM. Potential factors influencing racial/ethnic disparities in T2DM are grouped into five domains of influence: socioeconomic, environmental, psychosocial, lifestyle/behavioral, biophysiological. Race/ethnicity, age, sex, and genetic constructs are considered exogenous. Constructs operationalized in the BACH III Survey are listed in the ovals (conceptual domains). CVD, cardiovascular disease; HTN, hypertension; Hx, history.

center, acute care hospital, or health care center of either kind (miles). Psychosocial influences considered included hours of sleep each night, major life events (13), and sense of personal control (14). Lifestyle/behavioral factors assessed include dietary patterns (2005 Block food frequency questionnaire [15] assessed the 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 (16); BMI, waist circumference, and body fat percentage, which were measured by trained field interviewers; and smoking history. Biophysiological influences considered 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, menopausal status (women only), and history of gestational diabetes.

To measure BGA we evaluated a panel of 63 ancestry-informative markers, including 33 autosomal single nucleotide polymorphisms differentiating Native American versus European ancestry and 30 single nucleotide polymorphisms differentiating West African versus European ancestry. The 63 markers combined can provide an estimate of the percentages of West African, Native American, and European ancestry for each participant (17). Genotyping was conducted at the Broad Institute using the Sequenom iPLEX platform. A family history of diabetes was also considered as an independent risk factor for T2DM. Race/ethnicity, age, and sex, BGA, and family history of diabetes were considered exogenous factors.

Structural Equation Modeling

We applied two-level structural equation modeling (SEM) to assess the associations between race/ethnicity, confounding and mediating characteristics, and T2DM. Two-level SEM allows us to include both direct and indirect effects of each risk domain on T2DM, as hypothesized in the conceptual model (Fig. 1), while accommodating the 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 T2DM (depicted in Fig. 1 by a single arrow from socioeconomic risk to T2DM, 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 T2DM and serves as a mediating influence. We relied on the published literature and inherent temporality to determine the direction of the effects. Correlations between the measurement errors of two variables are represented by bidirectional curves. Standardized coefficients (sβ) (18) and their P values are reported. The threshold for statistical significance was $P < 0.05$. For simplicity, age and gender effects and nonsignificant pathways ($P \geq 0.05$) are not presented here (full results are available in Supplementary Table 2). We performed a 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 (19). The overall mediated percentage 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

Data based on the five theoretical mediating domains of influence (socioeconomic, environmental, psychosocial, lifestyle/behavioral, and biophysiological) were used to create risk scores. The variables listed in Fig. 1 were reduced from those in the conceptual model using race/ethnicity-, age-, and sex-adjusted models (Supplementary Table 1). Variables that did not either *1*) meet a minimal criterion for association with T2DM ($P < 0.10$) or *2*) reduce the race/ethnic effect (odds ratio [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 (natural logarithm of the OR) to the nearest integer, using methods similar to those used by Bang et al. (20), which is the basis for the American Diabetes Association self-screening tool. For continuous variables, risk was based on clinically accepted "high-risk" criteria. If clinically accepted criteria were not available, tertiles were used. Following the construction of the final model, all variables were added to the model singly to ensure their effects were adequately captured by the risk scores.

To minimize bias and reductions in precision, multiple imputation was implemented using the Multivariate Imputation by Chained Equations (21) algorithm in R (Vienna, Austria) to account for nonmonotone item nonresponse. In general, there is no way to test whether the missing data mechanism is independent of the observed data (missing completely at random), dependent only on the observed data (missing at random), or dependent on unobserved data (missing not at random). Under the missing at random assumption, however, multiple imputation may reduce bias that would be induced in a complete case analysis (22). Fifteen multiple imputation data sets were created. Imputations were conducted separately for each racial/ethnic-by-sex combination to preserve interaction effects, and the complex survey sample design was taken into account. DNA samples were obtained from and isolated for 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 variables were low (<10%). The sampling design of BACH requires weighting observations so they are inversely proportional to their probability of selection for results to be generalizable to the base population. Sampling weights were poststratified to produce estimates representative of the black, Hispanic, and white populations in Boston, MA.

RESULTS

The prevalence of diabetes in the BACH III Survey was 23.4%; 15.0% reported a previous T2DM diagnosis and 8.4% had FG >125 mg/dL (7.2%) and/or HbA_{1c} ≥6.5% (4.2%). The demographic characteristics of the 2,746 participants the analytic sample are presented in Table 1. The sample comprised approximately one-third black (33.6%, unweighted), one-third Hispanic (33.9%), and one-third white (32.5%) participants. Hispanic participants of any race self-identified as having a number of ethnic origins, the most prevalent being the Dominican Republic (34.8%) and Puerto Rico (30.2%). The average age of the participants was 54 years. Compared with nondiabetic participants, participants with T2DM were older; had more West African genetic ancestry; were of lower SES; reported greater discrimination; had lower health literacy; lived in census tracts with lower SES/greater poverty and neighborhoods with more minority residents; reported more neighborhood disorder, short (<6 h) or long (>9 h) sleep durations, a lower sense of control, and less physical activity; had greater BMI, waist circumference, and body fat percentage; and had higher blood pressure, total cholesterol, and triglycerides and lower HDL cholesterol.

Using the results of the race/ethnic-, sex-, and age-adjusted models (Supplementary Table 1), we identified 24 variables within the 5 mediating domains that were associated with T2DM prevalence and/or racial/ethnic disparities in T2DM (Table 2). This produced the following risk scores (presented as range [mean]): socioeconomic, 0–10 (4.3); environmental, 0–1 (41.2%); psychosocial, 0–5 (1.7); lifestyle/behavioral, 0–8 (3.2); and biophysiological, 0–11 (2.7).

The SEM specified in Fig. 1 fit the data well. Age and sex had direct effects on almost all factors with the exception of environmental risk. The lifestyle/behavioral domain was the largest direct predictor of T2DM status (sβ = 0.25; $P < 0.001$), followed by biophysiological factors (sβ = 0.19; $P < 0.001$), socioeconomic factors (sβ = 0.13; $P = 0.003$), and family history of diabetes (sβ = 0.10; $P = 0.005$). There was a marginal direct effect of self-identified race/ethnicity on T2DM prevalence (black: sβ = 0.18, $P = 0.054$; Hispanic: sβ = 0.10, $P = 0.069$). The sβ represented in Fig. 2 can be interpreted as a 1-SD difference in the predictor (i.e., lifestyle/behavioral risk) is associated with a 0.25-SD difference in the outcome (i.e., T2DM). Nonstandardized coefficients are available online (Supplementary Table 2); these values suggest that for every 1-unit increase in the lifestyle/behavioral risk score, the odds of T2DM increase 35% (OR 1.35; 95% CI 1.25–1.47), and for every 1-unit increase in the biophysiological risk score, the odds increase 29% (OR 1.29; 95% CI 1.16–1.43).

Self-identified black race had a significant direct effect on only socioeconomic risk (sβ = 0.23; $P = 0.003$) and environmental risk (sβ = 0.14; $P = 0.001$). There was no direct effect of self-identified black race on psychosocial (sβ = 0.003; $P = 0.965$), lifestyle/behavioral (sβ = 0.11; $P = 0.081$); or biophysiological risk (sβ = 0.07; $P = 0.264$) or T2DM (sβ = 0.18; $P = 0.054$). 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 (Fig. 2) indicated that 38.9% of the total effect of black race was mediated by the socioeconomic, environmental, psychosocial, and 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 (sβ = 0.17; $P < 0.001$),

Table 1—Characteristics of the BACH III study population overall by diabetes status (*N* = 2,476)

	Overall (*N* = 2,764)	T2DM (*n* = 892)	No T2DM (*n* = 1,872)	*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)	
Age (years)				
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)	
Male sex	1,018 (46.46)	344 (52.00)	674 (44.82)	0.056
Genetic influences (%), mean (SE)				
West African	29.84 (1.23)	39.02 (2.27)	27.12 (1.43)	<0.001
Native American	6.85 (0.29)	6.53 (0.56)	6.95 (0.36)	0.545
European	63.31 (1.27)	54.45 (2.33)	65.93 (1.49)	<0.001
Family history of diabetes	1,483 (46.52)	602 (62.12)	882 (41.91)	<0.001
Socioeconomic influences				
Income				
<$20,000	1,234 (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	1,324 (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 U.S.	1,645 (78.97)	488 (77.50)	1,157 (79.41)	0.490
Acculturation (English not first language)				
Low	669 (8.53)	253 (10.54)	416 (7.93)	0.178
High/bicultural	2,095 (91.47)	639 (89.46)	1,456 (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	1,759 (80.32)	445 (65.60)	1,313 (84.67)	
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	2,034 (74.52)	611 (69.02)	1,423 (76.15)	
Insurance status				
Private	1,001 (51.41)	218 (33.84)	783 (56.61)	<0.001
Public	1,671 (46.03)	654 (64.14)	1,016 (40.67)	
None	92 (2.56)	20 (2.02)	73 (2.72)	
Visits to health care provider in the past year				
0–1	395 (16.71)	74 (9.49)	321 (18.85)	<0.001
2–6	1,459 (51.96)	411 (49.12)	1,048 (52.81)	
≥7	910 (31.32)	407 (41.39)	503 (28.35)	
Usual source of care	2,714 (98.75)	880 (98.48)	1,834 (98.82)	0.651
Environmental influences				
SES by CT				
Low	1,269 (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)	
Poverty by CT				
<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%	1,533 (39.40)	561 (48.01)	972 (36.86)	

Continued on p. 1213

Table 1—Continued

	Overall (*N* = 2,764)	T2DM (*n* = 892)	No T2DM (*n* = 1,872)	*P* value
Racial composition of CT (%), mean (SE)				
Black	26.80 (1.07)	32.61 (1.86)	25.09 (1.18)	<0.001
Hispanic	16.62 (0.53)	18.25 (0.88)	16.14 (0.55)	0.017
White	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	1,316 (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
Open space in CT (%), 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
Spatial access to 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
Psychosocial				
Sleep duration (h)				
<6	622 (17.53)	259 (27.98)	363 (14.44)	<0.001
6–9	2,097 (81.28)	617 (69.96)	1,480 (84.62)	
>9	45 (1.19)	17 (2.06)	28 (0.93)	
Major life events (0–10), 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				
<1,500 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 fiber	171 (7.17)	51 (5.28)	120 (7.73)	0.172
<14 g saturated fat	1,040 (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 kilocalories, mean (SE)	1,745.4 (32.02)	1,685.1 (79.47)	1,763.2 (34.47)	0.370
Physical activity				
Low	1,132 (33.21)	480 (47.31)	652 (29.03)	<0.001
Medium	1,286 (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 percentage, mean (SE)	33.96 (0.32)	36.74 (0.57)	33.13 (0.37)	<0.001
Smoking history				
Never	1,220 (44.25)	373 (37.55)	847 (46.22)	0.014
Former	1,015 (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	2,110 (70.16)	805 (89.44)	1,305 (64.46)	<0.001
Cardiovascular disease	604 (16.09)	315 (32.66)	289 (11.19)	<0.001
Women only				
Menopausal status				
Pre-/perimenopause	437 (36.63)	67 (16.14)	370 (41.90)	<0.001
Postmenopause	740 (36.79)	241 (40.54)	499 (35.83)	
Undetermined/other	569 (26.57)	240 (43.33)	329 (22.27)	
Gestational diabetes mellitus	125 (5.93)	72 (17.07)	54 (3.07)	<0.001

Data are *n* (column %) (categorical variables) unless otherwise indicated (continuous variables). *P* values were calculated using the χ^2 test. CT, census tract; DBP, diastolic blood pressure; FFQ, food frequency questionnaire; SBP, systolic blood pressure.

Table 2—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 U.S.	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		
Poverty by CT	>20%	
Psychosocial		
Sleep duration	<6 h	>9 h
Major life events	>1	
Sense of control (tertiles)	<1.0	<0.43
Lifestyle/behavioral		
Physical activity	Low	
Smoking history	Current	
BMI (20)	25–29 kg/m^2	30–39 kg/m^2 (>40 adds 3 risk points)
Waist circumference (23)	≥102 cm (men) ≥88 cm (women)	
Body fat percentage (tertiles)	>25% (men) >35% (women)	>33% (men) >42% (women)
Biophysiological		
Blood pressure (23)	SBP ≥130 or DBP ≥85 mmHg or self-report of hypertension diagnosis	
Cholesterol (total) (24)	≥200 mg/dL	≥240 mg/dL
HDL cholesterol (23)	<40 mg/dL (men) <50 mg/dL (women)	
Triglycerides (23)	≥150 mg/dL	
Cardiovascular disease		Yes
Menopausal status	Postmenopause	Surgical/undetermined
Gestational diabetes mellitus		Yes

CT, census tract; DBP, diastolic blood pressure; HCP, health care provider; SBP, systolic blood pressure.

environmental risk (sβ = 0.29; $P < 0.001$), and psychosocial risk (sβ = 0.17; P = 0.04). There was no significant direct effect of Hispanic ethnicity on lifestyle/behavioral risk (sβ = 0.04; P = 0.369), biophysiological risk (sβ = 0.04; P = 0.283), or T2DM (sβ = 0.10; P = 0.07). Mediation analyses indicate that 45.7% of the total effect of Hispanic ethnicity was explained by the risk scores. The largest mediator was the socioeconomic risk score (26.2%).

Despite the considerable differences in BGA among participants with T2DM versus those with no diabetes in the bivariate results (Table 1), neither West African ancestry (OR 1.02; 95% CI 0.92–1.14) nor Native American ancestry (OR 0.94; 95% CI 0.79–1.10) contributed to T2DM once self-identified race/ethnicity was included in the model (Supplementary Table 1). The final SEM also indicated that there was no significant direct effect of West African (sβ = −0.003; P = 0.976) or Native American ancestry (sβ = −0.02; P = 0.725) on T2DM once self-identified race/ethnicity was accounted for (Supplementary Table 2).

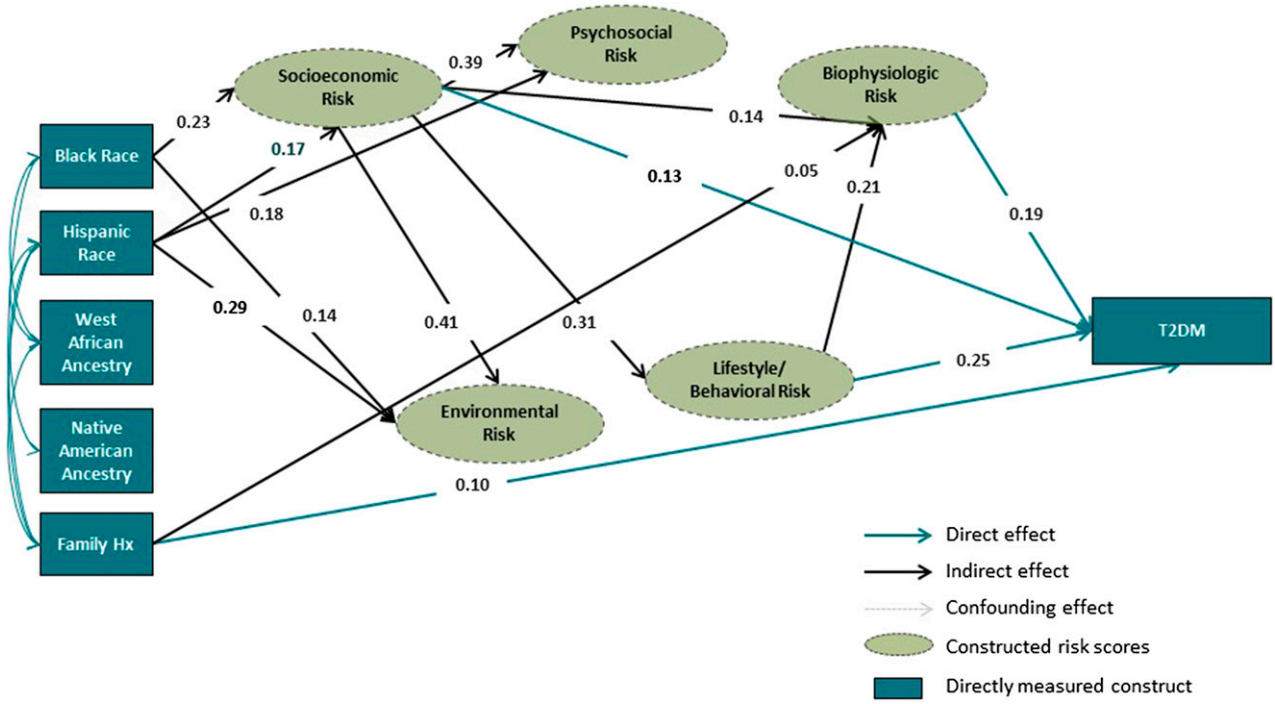
CONCLUSIONS

To our knowledge, this study presents the first examination of a multilevel risk model aimed at explaining racial/ethnic disparities in T2DM. While many authors have proposed similar conceptual frameworks with the aim of understanding and eliminating health disparities (4,25,26), to our knowledge the BACH Survey is the first to amass these data and test this model of health disparities in T2DM 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 T2DM. The data supported this temporality: individual lifestyle/behavioral risk had the largest direct effect on T2DM 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 mediated percentage was even lower. This aligns with previous research that indicates that white Americans participate in significantly more physical activity during leisure time than black and Hispanic Americans (27). However, accelerometry data indicate that Hispanics may have higher physical activity levels overall than black and white Americans, potentially because of physically demanding occupational or domestic activities (28). The latter findings may be one potential reason for the differences we see in the mediated percentage by lifestyle/behavioral factors among black and Hispanic participants; they also highlight the complex interplay between socioeconomic (occupational) and lifestyle/behavioral factors.

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 effect of socioeconomic factors on T2DM. Studies that aim to assess the role of lifestyle and behavioral factors on the socioeconomic gradient of health in T2DM 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 T2DM (29).

The mediation analyses indicate that the largest explainable proportion of the excess proportion of T2DM among black and Hispanic participants is attributable to socioeconomic risk. The socioeconomic risk score developed explains



	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%

Figure 2—Final structural equation model of factors in the pathway from race/ethnicity to T2DM. sβ are presented for all statistically significant pathways. Hx, history.

22% of the excess odds of T2DM among black participants and 26% of the excess odds among Hispanic participants. The statistical analyses indicate that while much of the excess odds of T2DM among blacks and Hispanics remains unexplained (61% and 54%, respectively), adverse socioeconomic conditions explains the largest proportion of racial/ethnic disparities in T2DM in our model. The socioeconomic risk score encompasses a variety of risk factors, including social and economic structures of society (i.e., income and occupation), acculturation, health literacy, and utilization of health care. It is likely that these risk factors individually may have differential effects on black and Hispanic individuals. These relationships could be an area for future research.

Our data, supported by our previous findings (30), suggest that the effects of BGA on T2DM are attenuated by 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 (5,6,31). However, other studies have found the effect of BGA on T2DM to be more robust with adjustment (6,32,33), including research from the BACH study, which demonstrates that the effect of BGA on prediabetic illness may be robust with adjustment for social factors (17). Race and ethnicity are complex, multidimensional constructs reflecting biogeographic origin and biological, social, cultural, and economic factors (34). Our findings suggest that while BGA may be associated with precursors to T2DM, it is likely that the social, cultural, and economic facets of race/ethnicity may better explain T2DM disparities in the BACH study. Family history of diabetes, which may have a genetic component, may also be the result of similar socioeconomic, environmental, psychosocial, lifestyle, and biophysiological risk profiles between parent(s) and offspring; family history had a direct effect on T2DM prevalence (0.10 sβ; $P = 0.005$) and was highly associated with race/ethnicity.

Each domain of the conceptual model presented here suggests a particular structural intervention. Increased socioeconomic risk suggests policy interventions affecting social conditions; environmental risk suggests community intervention; psychosocial risk suggests primary prevention aimed at reducing psychological strain and increasing coping mechanisms; lifestyle/behavioral risk suggests primary prevention directed at increasing healthy, and decreasing unhealthy, behaviors; and biophysiological risk suggests 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), suggest that interventions targeting lifestyle/behavioral and biophysiological risk may reduce T2DM risk overall. However, the results presented here demonstrate that interventions aimed at reducing disparities may need to target socioeconomic risk factors 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 been shown to increase the proportion of social inequalities that can be explained by potential modifiable risk factors.

A second limitation is that the BACH study is geographically limited to Boston, MA. The macroeconomic influences (primarily socioeconomic and environmental) of living in this urban environment in the U.S. Northeast may not be generalizable to other contexts or to the conditions in which racial/ethnic disparities in T2DM are fostered in the U.S. at large. On the other hand, the BACH Survey sample has been compared with other, large regional (Behavioral Risk Factor Surveillance System) and national (the National Health and Nutrition Examination Survey) surveys on a number of sociodemographic and health-related variables. The results suggest that the BACH estimates of key health conditions are comparable with national trends (e.g., history of diabetes, history of hypertension, history of smoking) (36). However, BACH participants are more likely to be women, foreign born, and unemployed (men).

Third, although we measured BGA markers, which are thought to estimate the contribution of genetics to increased diabetes prevalence in certain populations, we do not have comprehensive markers of genetic risk. We therefore cannot make conclusions regarding genetic contributors to racial/ethnic disparities in T2DM.

The key strengths of this study stem from the community-based, stratified, random sample design of the BACH Survey, which provided 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 wherever available. Finally, unlike many studies of T2DM, we did not rely solely on self-report for T2DM status. Participants were contacted in the morning in their home, giving a more accurate prevalence of T2DM.

Conclusion

Our study found that while lifestyle/behavioral and biophysiological risk factors had the greatest direct effect on T2DM risk, socioeconomic factors had the greatest impact on explaining racial/ethnic disparities in T2DM.

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Author Contributions. R.S.P. acquired, analyzed, and interpreted data; performed statistical analysis; supervised the study; provided administrative, technical, or material support; and wrote and critically revised the manuscript. S.V.S. analyzed and interpreted data and critically revised the manuscript. N.P. critically revised the manuscript and provided administrative, technical, or material support. J.C.F. conceived and designed the study; critically revised the manuscript; and provided administrative, technical, or material support. J.B.M. conceived and designed the study; obtained funding; acquired data; supervised the study; provided administrative, technical, or material support; and critically revised the manuscript. R.S.P. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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