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Racial-Ethnic Disparities in the Association between Risk Factors and Diabetes: the Northern Manhattan Study

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Abstract

Purpose—To identify risk factors (RF) for diabetes within a multiethnic cohort and to examine whether race-ethnicity modified their effects.

Methods—Participants in the Northern Manhattan Study without diabetes at baseline were studied from 1993–2014 (n=2, 430). Weibull regression models with interval censoring data were fit to calculate hazard ratios and 95% confidence intervals for incident diabetes. We tested for interactions between RF and race-ethnicity.

Results—During a mean follow-up period of 11 years, there were 449 diagnoses of diabetes. Being non-Hispanic black (HR 1.69 95%CI 1.11–2.59) or Hispanic (HR 2.25 95%CI 1.48–3.40) versus non-Hispanic white, and body mass index (BMI; HR 1.34 per SD 95%CI 1.21–1.49) were associated with greater risk of diabetes; high-density lipoprotein cholesterol (HR 0.75 95%CI 0.66–0.86) was protective. There were interactions by race-ethnicity. In stratified models, the effects of BMI, current smoking, and C-reactive protein (CRP) on risk of diabetes differed by race-ethnicity (p for interaction <0.05). The effects were greater among non-Hispanic whites than non-Hispanic blacks and Hispanics.

Conclusions—Although Hispanics and non-Hispanic blacks had a greater risk of diabetes than whites, there were variations by race-ethnicity in the association of BMI, smoking, and CRP with

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Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

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risk of diabetes. Unique approaches should be considered to reduce diabetes as traditional RF may not be as influential in minority populations.

Keywords

Diabetes Mellitus; Health Status Disparities; Epidemiology; Risk Factors; Urban Health

Introduction

Type 2 diabetes mellitus is a significant public health problem in the United States; approximately 21 million people have been diagnosed with the disease while another 8.1 million remain undiagnosed [1,2]. The prevalence and incidence of type 2 diabetes have been steadily increasing, particularly among minority racial-ethnic groups [3–5]. The cost of diabetes surpassed \$245 billion dollars in 2011, making it one of the most expensive chronic diseases in the United States today [6,7]. This is predominantly due to the fact that diabetes is a major risk factor for other diseases, notably cardiovascular disease and stroke [8,9]. Recent research has also shown that diabetes might be an independent risk factor for dementia, Alzheimer disease, and both cognitive and functional decline [10–12].

Significant racial-ethnic disparities are seen in the age-adjusted prevalence of diagnosed diabetes in the United States; 9% of non-Hispanic black and 13% of Hispanic individuals aged 20 years or older have been diagnosed with the disease, a rate almost double that of non-Hispanic whites (8%) [2,13,14]. Racial-ethnic disparities are also present in mortality attributed to the disease, with non-Hispanic black and Hispanic mortality rates far surpassing those of non-Hispanic whites [15–17].

Despite well-documented disparities in the morbidity and mortality of diabetes across racial-ethnic groups, there has been limited examination of racial-ethnic variation in influence of risk factors for the disease. Risk factors for diabetes include sociodemographic factors (age, sex, socioeconomic status (SES)); behavioral factors (physical inactivity, smoking, alcohol consumption, and overweight); and physiological factors and co-morbidities (hypertension (HTN), high-density lipoprotein cholesterol (HDL), and C-reactive protein levels (CRP)) [18–22]. Several studies have also listed CRP as an independent predictor of the disease [23,24]. There is little research to indicate whether these risk factors have similar magnitudes of effect across racial-ethnic groups. This study set out to identify risk factors for incidence of diabetes within an ethnically diverse cohort. Further, we explored whether race-ethnicity modified the effects of these factors on the risk of developing diabetes.

Methods

Research Design and Methods

The Northern Manhattan Study (NOMAS) is an ongoing, prospective, population-based cohort study, originally designed to measure stroke incidence, cardiovascular risk factors, and cardiovascular outcomes in a multiethnic urban population in Northern Manhattan. Full methodology has been described previously [25].

All activities pertaining to NOMAS were approved by the Institutional Review Boards at Columbia University Medical Center and the University of Miami. Written consent was obtained for each participant upon enrollment.

Selection of NOMAS Cohort

Cohort recruitment occurred from 1993–2001. Residents of Northern Manhattan were considered eligible if they had never been diagnosed with stroke, resided in Northern Manhattan with a working telephone for three or more months, and were at least 40 years old. Participants were contacted using random digit dialing. The overall enrollment rate was 68%.

Among the 3,298 total NOMAS participants, a sub-cohort of 2,498 participants had no diagnosis of diabetes at baseline. The final analytical cohort (n=2,430) excluded those with race-ethnicity defined as ‘other’ or missing (n=68). The current analysis included data from baseline assessment through 2014.

Baseline Evaluation

Participants were invited for an in-person baseline interview and health assessment for risk factors of stroke and cardiovascular disease. All interviews were conducted by trained bilingual interviewers in the primary language of the participant. Questions for the baseline interview were adapted from the Centers for Disease Control and Prevention Behavioral Risk Factor Surveillance System Questionnaire [26] and validated in the NOMAS cohort [27].

Definition of Race-Ethnicity

Data on race-ethnicity were collected through self-identification. Response options were modeled after the categories used by the 1990 census: white, black, American Indian, Eskimo, Asian or Pacific Islander, and other. Ethnicity was self-defined as Hispanic or non-Hispanic. In this population, participants who identified themselves as Hispanic or of Spanish origin were classified primarily as Hispanic. Racial-ethnic categories were classified as ‘non-Hispanic white’, ‘non-Hispanic black’, ‘Hispanic’, or ‘other’. Due to small sample size (n=68), ‘other’ race-ethnicities were excluded from all analyses.

Risk Factor Assessment and Follow-up

Education was dichotomized into less than high school or greater than or equal to high school education. Health insurance status was categorized as Medicare/private insurance or Medicaid/no insurance. Any physical activity was defined as engaging in any leisure activity during the past 10 days. Moderate alcohol intake was defined as one drink per month up to two per day. Current smoking was characterized as having smoked within the last year. Body Mass Index (BMI) was calculated from weight in kilograms divided by height squared in meters, and assessed as a continuous variable. Hypertension (HTN) was having a systolic blood pressure > 140 mm/Hg or a diastolic blood pressure ≥ 90 mm Hg (average of two measurements), physician diagnosis, or the patient’s self-report of a history of HTN. High-density lipoprotein cholesterol levels (HDL) levels were analyzed continuously. Concentration of high-sensitivity C-Reactive Protein (CRP) was available for a sub-cohort

of 1, 676 participants, log-transformed to achieve linearity, and assessed as a continuous variable. Diabetes at baseline was defined as having at least one of the following: fasting blood glucose greater or equal to 126 mg/dL, self-reported medical diagnosis of diabetes mellitus, or treatment with insulin or oral hypoglycemic [9]. Those with diabetes at baseline were excluded from this analysis.

Participants underwent annual follow-up telephone interview in order to assess changes in vital status, neurologic events, cardiac symptoms, risk factor status, medications, and overall functional status. If the subject had been admitted to the hospital, confirmation of telephone responses and additional clinical information was garnered using medical records.

Participants who reported a neurological or cardiovascular event, or whose medical records indicated an event had occurred, underwent an additional in-person examination by a staff neurologist.

Assessment of onset of Diabetes during follow-up

Self-Reported diabetes status was updated at each annual follow-up interview. The primary outcome, onset of diabetes was defined as responding positively to at least one of the following questions: “Since we last contacted you, have you been newly diagnosed with diabetes or high blood sugar?” or “Do you currently take any of the following medications: Insulin or Oral Hypoglycemics”. Interval change in diabetes status was confirmed by review of medical records using methodology previously described and validated [9].

Statistical Analyses

Distributions of sociodemographic characteristics and cardiovascular risk factors at baseline were calculated as means for continuous variables and proportions for categorical variables. Characteristics were further stratified and tested for differences by race-ethnicity using Wilcoxon rank sum test for continuous variables, and the chi-squared test for proportions. Weibull regression models with interval censoring failure time were fitted to calculate hazard ratios (HR) and 95% confidence intervals (CI) for time from baseline evaluation to onset of self-reported diabetes. Weibull models were used to accommodate the interval censored nature of this data, as the onset of diabetes occurred at an unknown time within the interval of two known follow-up time points. The models were adjusted for sociodemographic (age, sex, race-ethnicity, education, health insurance status) and cardiovascular risk factors (smoking, physical activity, HTN, alcohol use, BMI, and HDL) (Model 1). A secondary analysis introduced CRP as a predictor (Model 2). To evaluate potential effect modification by race-ethnicity, we tested for multiplicative interaction between all risk factors and race-ethnicity (non-Hispanic white as a reference), and reported stratified results when there was a significant interaction based on chi-square test with two degrees of freedom ($p < 0.05$). As sensitivity analyses, we fitted sub-distribution proportional hazard models with death as a competing risk to assess if the effect of risk factors on risk of incident diabetes was affected by competing risk of death. All analyses were performed using SAS Version 9.3 (SAS Institute Inc., Cary, NC, USA).

Results

Description of the Cohort

Characteristics for the cohort free of diabetes at baseline ($n = 2,430$), overall and by race-ethnicity are outlined in Table 1. Mean age at time of enrollment was 69 years ($SD \pm 10.6$). Of the cohort, 37% were men; 23% were non-Hispanic white, 24% non-Hispanic black, and 53% Hispanic. Compared to non-Hispanic blacks or Hispanics, non-Hispanic whites were more likely to have completed high school education and be physically active, and were less likely to have Medicaid or no insurance, be hypertensive and currently smoking. Overall, non-Hispanic blacks and Hispanics had a greater burden of risk factors in comparison to non-Hispanic whites.

Of the 2,430 participants free of diabetes at baseline, 18.5% ($n=449$) were diagnosed with diabetes during a mean follow-up period of 11.0 years (range 0.25–20.5). The cumulative incidence of diabetes differed by race-ethnicity; there were 43 (7.6%) diagnoses of diabetes in non-Hispanic whites, 77 (13.4%) in non-Hispanic blacks, and 329 (25.5%) in Hispanics.

Association of sociodemographic and risk factors with the risk of developing diabetes during follow-up

In model 1, adjusted for sociodemographic and cardiovascular risk factors, non-Hispanic black (HR 1.69, 95% CI 1.11–2.59) or Hispanic (HR 2.25, 95% CI 1.48–3.40) versus non-Hispanic white was associated with a greater risk of developing diabetes. BMI (HR per standard deviation (SD) increase 1.34, 95% CI 1.21–1.49) was also associated with greater risk while HDL levels were associated with a lower risk of diabetes (HR per SD increase 0.75, 95% CI 0.65–0.86). In secondary analyses in the sub-cohort with CRP available ($n=1724$), CRP concentration was also associated with a greater risk of diabetes (HR per SD log-CRP 1.25, 95% CI 1.08–1.44) (Table 2). Additional analyses were run using waist-hip ratio in place of BMI in all models with minimal changes in effect size (data not shown). In sensitivity analyses with mortality as a competing risk, we found that the magnitude of association remained unchanged with the exception of age (supplemental table).

Effect modification by the association of risk factors and risk of developing diabetes by race-ethnicity

The effect of BMI, current smoking, and CRP on the risk of diabetes differed by race-ethnicity (chi-square with two degrees of freedom, p -values<0.05). Those effects were greater among non-Hispanic whites than non-Hispanic blacks or Hispanics.

In models stratified by race-ethnicity (Table 3), BMI was associated with risk of diabetes in non-Hispanic whites (HR per SD increase 1.62 95% CI 1.28–2.05) and Hispanics (HR per SD increase 1.37 95% CI 1.22–1.55), but not in non-Hispanic blacks. CRP was also associated with increased risk of diabetes in non-Hispanic whites (HR per SD log-CRP 1.66 95% CI 1.05–2.61) and Hispanics (HR per SD log-CRP 1.32, 95% CI 1.12–1.56), but not in non-Hispanic blacks (HR per SD log-CRP 0.94 95% CI 0.71–1.24). The non-Hispanic white population was the only group for whom current smoking was a significant risk factor (HR

3.11, 95% CI 1.50–6.47) for diabetes. None of the risk factors analyzed were found to be significantly associated with risk of diabetes in non-Hispanic black participants.

Discussion

In this multi-ethnic cohort study, we report on two key findings. First, we identified disparities in the development of diabetes, finding that minority populations (i.e., non-Hispanic blacks and Hispanics) were at increased risk of developing diabetes. The pattern that we see with diabetes onset in our cohort has been observed in other studies assessing the influence of racial-ethnic disparities in pre-diabetes [28], control of diabetes [29,30], and stroke [31–33]. Increased BMI and CRP were also associated with an increased risk of diabetes. These results echoed earlier studies which reported a strong relationship between both BMI and CRP and the risk of diabetes [14,17,18,34]. Second, we found evidence of a differential effect of these risk factors on the onset of diabetes among race-ethnic subgroups, such that there was an attenuation of strength of association among non-Hispanic blacks and Hispanics, compared to non-Hispanic whites. While BMI, smoking, and CRP all were associated with risk of diabetes among non-Hispanic whites, none of these factors were associated among non-Hispanic blacks. In the Hispanic population, BMI and CRP were associated with risk of diabetes, while smoking was not.

Our results suggest that traditional risk factors do not equally explain the risk of diabetes across all racial-ethnic groups. Race-ethnicity is a multifaceted construct which not only reflects race, but also biological processes, geographical origins, and social, cultural, and socioeconomic factors [35–37]. This makes the study of racial-ethnic disparities in health a complex task. Studies have suggested that the underlying biological and genetic mechanisms shown to cause diabetes may contribute to the variation of risk factor influence across racial-ethnic groups [38,39]. It is possible that these mechanisms are more advanced at the point of baseline measurement in minority populations. In this study, we did not adjust for a marker of insulin resistance or pre-diabetes since the information was available only for a subgroup of our cohort. An earlier sub-cohort analysis of 1,509 non-diabetic NOMAS participants found variation in Homeostatic Model Assessment -Insulin Resistance (HOMA-IR) index scores with 15% of non-Hispanic whites, 22% of non-Hispanic blacks, and 30% of Hispanics ($p<0.05$) in a state of insulin resistance [40]. This may indicate that our findings reflect that non-Hispanic blacks and Hispanics were already at higher risk of diabetes at baseline and the further additive impact of the risk factors on disease progression was lessened.

A study by Piccolo et al. [41] examined the association of geographical origin with the incidence of diabetes and found that while African ancestry was found to be significantly associated with the risk of diabetes in comparison to European ancestry, the effects of SES accounted for over 70% of the total effect attributed to ancestry, concluding that cultural and socioeconomic factors that define race-ethnicity may better explain disparities in disease prevalence. In our study, the classification of SES depended on a combination of education and health insurance status; we did not include factors that specifically capture income or neighborhood-specific factors, many of which have been shown to more strongly influence disease prevalence than race-ethnicity itself [42–44]. We have used education as a proxy for

SES, as studies have shown that variations in education even after adjusting for other factors are associated with poorer health behaviors and outcomes [45,46]. Within our cohort, non-Hispanic blacks and Hispanics are more likely to have less than a high school education and Medicaid/no insurance, indicating lower SES. It is therefore very difficult to tease apart the separate effects of race-ethnicity and SES.

The use of the NOMAS cohort to assess diabetes risk factors has several important strengths. The loss-to-follow-up in the NOMAS study is less than one percent due to a vigorous follow-up methodology. In coordination with the prospective population-based study design, this allows for well-documented risk factor and outcome data. The use of a large, multiethnic, heterogeneous cohort selected through a random sampling methodology is generalizable to other multiethnic urban populations, allowing for more valid comparisons across populations and racial-ethnic categories in the future.

There were several limitations to this study. First, there may be selection bias, as the analytic cohort included only those who had not already been diagnosed with diabetes at baseline. This, in addition to the older age of the cohort as a whole, may indicate that the study participants may be a healthier population and have lower rates of risk factors than those who were diagnosed with diabetes at a younger age. Second, the different age structure of the race-ethnic groups when enrolled in NOMAS may also limit the analysis; whites were on average 10 years older than Hispanics. Knowing that age is highly correlated with increase in risk factors for diabetes as well as mortality, it is plausible that the differential effects on risk of diabetes across race-ethnic groups could be due to this uneven age distributions. However, adjusting for age didn't change the results, and there did not appear to be any association between risk of diabetes and age in this cohort. Moreover, the results remained unchanged even when mortality were taken into account as a competing risk. Third, there may also be some misclassification bias, primarily in regards to self-reported risk factors like alcohol consumption and smoking status. The possibility of under-reporting these less socially desirable behaviors would likely lead to an under-estimate of the effect of these factors on the development of diabetes. We, however, have no evidence that this bias would differ across races so the impact is expected to be slight. The outcome in this study, diabetes, is also based off a series of self-reported questions. Currently studies have begun to use measures such as Hemoglobin A1C to diagnose and measure diabetes, however the NOMAS cohort has been followed for many years before such testing was standard for screening of diabetes. Prior studies within the NOMAS cohort have determined the validity of this self-reported diabetes measure is high. Using medical records, over 90% of participants responding yes to the use of medication and 100% of patients who self-reported a medical diagnosis of diabetes were confirmed to have the outcome [9]. A limitation remaining with a self-reported outcome measure is that participants who were unaware of their condition or had not yet been diagnosed would have been misclassified as not having diabetes. There is conflicting evidence as to whether having a missing diagnosis may be influenced by race-ethnicity, with some studies finding that black non-Hispanics and Hispanics were less likely to receive both diabetes and overall preventive care [47,48], while another study found that beliefs of discrimination and subsequent preventive service utilization for diabetes testing were not significantly different across racial-ethnic groups [49]. All participants in this study live within a single community in northern Manhattan

with access to a major medical center, which may help to lessen any disparity in access to preventive care utilization that may exist. A final potential limitation is that this study may not have addressed the full influence of exercise, diet and nutrition on the risk of diabetes. BMI was used in this analysis to assess overweight and obesity status while other anthropometric variables such as waist circumference and waist-hip ratio have also been used as determinants of insulin resistance in prior studies [50–52]. Minimal differences in effects using BMI compared to waist-hip ratio suggests that the use of one measurement over the other would not influence our final results. Although we evaluated ‘any physical activity’ as a predictor of diabetes, other levels of more vigorous physical activity that were less prevalent in this elderly cohort were not evaluated. Dietary factors, particularly in relation to racial-ethnic variation in diet within the cohort, may also have a significant influence on diabetes status [53–55], and were not analyzed here.

The findings of this study are important in order to begin to develop effective focused risk reduction strategies on specific racial-ethnic groups. It is important that we not imply the need to focus less on behavioral risk factors such as smoking, obesity, physical activity, and alcohol use as these are well-confirmed risk factors for a plethora of other diseases and such a recommendation is not beneficial for a population’s overall health status. These results more strongly imply that risk reduction strategies should be developed to intervene earlier in order to address these higher-risk populations at a younger age.

Conclusion

We found significant variations in the effects of previously identified diabetes risk factors across racial-ethnic groups, with many of these effects weaker in the non-Hispanic black and Hispanic populations. Greater emphasis should be placed on the varying influence of risk factors on the development of diabetes across racial-ethnic groups. More research is also needed to identify non-traditional risk factors in these populations. Further confirmation of these findings in different populations as well as the examination of new risk factors and biological mechanisms for diabetes will allow at-risk populations to be effectively targeted for public health interventions aimed at reducing the burden of disease.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Distribution of baseline sociodemographic and cardiovascular characteristics, overall and by race/ethnicity, Northern Manhattan Study, 1993–2014

		Race/Ethnicity			
	Overall (n=2,430)	White non-Hispanic (n=563)	Black non-Hispanic (n=576)	Hispanic (n=1,291)	p-value *
Sociodemographic Characteristics					
Mean age (y), (SD)	69.0 (10.6)	76.4 (10.2)	71.7 (10.8)	65.9 (9.68)	<0.0001
Men, % (n)	36.6 (889)	39.8 (224)	31.9 (184)	37.3 (481)	0.0175
Completed High School, % (n)	47.2 (1146)	82.6 (462)	64.4 (371)	24.0 (310)	<0.0001
Medicare or private insurance, % (n)	57.9 (1398)	90.1 (503)	76.5 (463)	35.7 (459)	<0.0001
Medicaid or no Insurance, % (n)	42.1 (1017)	9.86 (55)	23.5 (134)	64.3 (828)	<0.0001
Cardiovascular Risk Factors					
Current Smoker	16.8 (407)	12.6 (71)	22.4 (129)	16.2 (209)	<0.0001
Any Physical Activity, % (n)	59.6 (1449)	71.4 (402)	61.2 (387)	51.1 (660)	<0.0001
History of HTN, % (n)	71.0 (1725)	63.1 (355)	75.9 (437)	72.3 (933)	<0.0001
Moderate Alcohol Intake, % (n)	35.4 (861)	35.4 (883)	45.8 (258)	35.8 (206)	<0.0001
Mean Body Mass Index, (SD)	27.5 (5.35)	25.9 (5.09)	27.7 (6.14)	28.1 (4.92)	<0.0001
Mean High-Density Lipoprotein Cholesterol, (SD)	47.5 (14.6)	49.0 (14.6)	53.6 (16.3)	44.1 (12.8)	<0.0001
Mean C-Reactive Protein, (SD) **	4.73 (8.17)	4.36 (9.41)	5.52 (9.64)	4.54 (6.88)	0.0008

* testing for the differences across race-ethnicity. Chi-square 2 degrees of freedom for categorical variables; Wilcoxon test for continuous variables

** Based on a sub-cohort with laboratory blood available (n = 1676)

Table 2

Associations of sociodemographic and cardiovascular risk factors with risk of incident diabetes, Northern Manhattan Study, 1993–2014

	Model 1 *		Model 2 **	
Sociodemographic Factors				
	HR	95% CI	HR	95% CI
Non-Hispanic black (vs. non-Hispanic white)	1.69	1.11–2.59	2.04	1.16–3.59
Hispanic (vs. non-Hispanic white)	2.25	1.48–3.40	2.69	1.54–4.70
Age (per SD)	0.94	0.83–1.07	0.90	0.77–1.06
Male	1.15	0.91–1.45	1.19	0.89–1.59
Completed High School	0.91	0.71–1.16	0.90	0.66–1.22
Medicaid/no Insurance (vs. Medicare or private insurance)	1.22	0.97–1.53	1.09	0.83–1.44
Cardiovascular Factors				
Body Mass Index (per SD)	1.34	1.21–1.49	1.27	1.11–1.46
Moderate Alcohol Intake	0.86	0.68–1.08	1.02	0.77–1.33
Current Smoker (vs. non- or past smokers)	1.22	0.94–1.58	1.00	0.72–1.41
Any Physical Activity	0.85	0.69–1.04	0.85	0.66–1.09
Hypertension	1.23	0.97–1.58	1.14	0.85–1.53
Mean High-Density Lipoprotein Cholesterol (per SD)	0.75	0.66–0.86	0.73	0.61–0.87
Log C-Reactive Protein (per SD)	-		1.25	1.08–1.44

*
n=2,430

**
Model 2 included those with CRP available (n=1676)

Table 3

Associations with risk of incident diabetes stratified by race-ethnic groups, Northern Manhattan Study, 1993–2014

	Body Mass Index (per SD) **		Current Smoker (vs. non and past smokers) **		Log C-Reactive Protein (per SD) **	
	HR (95% CI)	p-value *	HR (95% CI)	p-value *	HR (95% CI)	p-value *
Non-Hispanic White	1.64 (1.27–2.04)	ref.	3.12 (1.50–6.47)	ref.	1.66 (1.05–2.61)	ref.
Non-Hispanic Black	1.07 (0.85–1.35)	0.012	0.78 (0.42–1.44)	0.004	0.94 (0.7–1.24)	0.035
Hispanic	1.37 (1.22–1.55)	0.213	1.21 (0.89–1.64)	0.018	1.32 (1.12–1.56)	0.348

* p-value for pair-wise comparison to test for effect modification, non-Hispanic white as a reference.

** p-values for global testing of the differences across race-ethnicity using Chi-square with 2 degrees of freedom were 0.039 for BMI, 0.019 for smoking and 0.048 for log-CRP