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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 to 2014 (n = 2430). 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 risk of diabetes. Unique approaches should be considered to reduce diabetes as traditional RF may not be as influential in minority populations.

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## 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 (Centers for Disease Control and Prevention, 2003; Centers for Disease Control and Prevention, 2014). The prevalence and incidence of type 2 diabetes have been steadily increasing, particularly among minority racial–ethnic groups (Kanjilal et al., 2006; Geiss et al., 2006; Mainous et al., 2007). The cost of diabetes surpassed $245 billion dollars in 2011, making it one of the most expensive chronic diseases in the United States today (Economic costs of diabetes in the U.S. in, 2012, 2013; Ariza et al., 2010). This is predominantly due to the fact that diabetes is a major risk factor for other diseases, notably cardiovascular disease and stroke (Sarwar et al., 2010; Banerjee et al., 2012). Recent research has also shown that diabetes might be an independent risk factor for dementia, Alzheimer disease, and both cognitive and functional decline (Luchsinger, 2010; Luchsinger, 2012; Dhamoon et al., 2014).

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%) (Centers for Disease Control and Prevention, 2014; Beckles et al., 2011; Lopez et al., 2014). 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 (Rosenstock et al., 2014; Roglic and Unwin, 2010; Freeman et al., 2011).

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

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factors and co-morbidities (hypertension (HTN), high-density lipoprotein cholesterol (HDL), and C-reactive protein levels (CRP)) (Lee et al., 2011; Ford, 1999; King et al., 2003; Chatterjee et al., 2014; Laaksonen et al., 2010). Several studies have also listed CRP as an independent predictor of the disease (Freeman et al., 2002; Mugabo et al., 2010). 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 (Sacco et al., 2001).

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 to 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 3298 total NOMAS participants, a sub-cohort of 2498 participants had no diagnosis of diabetes at baseline. The final analytical cohort (n = 2430) 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 (Gentry et al., 1985) and validated in the NOMAS cohort (Kargman et al., 1999).

### *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 1676 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 (Banerjee et al., 2012). 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 were 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 (Banerjee et al., 2012).

### *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 the 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 the 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 ± 0.6). Of the cohort, 37% were men; 23% were non-Hispanic white, 24% were non-Hispanic black, and 53% were 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 2430 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

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