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Coronary Death and Myocardial Infarction among Hispanics in the Northern Manhattan Study: Exploring the Hispanic Paradox

JOSHUA Z. WILLEY, MD, MS, **CARLOS J. RODRIGUEZ, MD, MPH**, **YESEON PARK MOON, MS**, **MYUNGHEE C. PAIK, PhD**, **MARCO R. DI TULLIO, MD**, **SHUNICHI HOMMA, MD**, **RALPH L. SACCO, MD, MS**, and **MITCHELL S.V. ELKIND, MD, MS**
Department of Neurology (J.Z.W., Y.P.M., M.S.V.E.); Department of Biostatistics, Mailman School of Public Health (M.P.); Department of Medicine, Cardiology division (M.D., S.H.); Department of Epidemiology, Mailman School of Public Health (M.S.V.E.), Columbia University, New York, NY; Department of Medicine, Division of Cardiology, and Division of Public Health Science, Wake Forest University, NC (C.J.R.); and Departments of Neurology, Public Health and Epidemiology, and Human Genetics, University of Miami, FL (R.L.S.).

Abstract

PURPOSE—Prior studies have reported that Hispanics have lower cardiovascular disease (CVD) mortality despite a higher burden of risk factors. We examined whether Hispanic ethnicity was associated with a lower risk of nonfatal myocardial infarction (MI) coronary death (CD) and vascular death.

METHODS—A total of 2671 participants in the Northern Manhattan Study without clinical CVD were prospectively evaluated. Cox models were used to calculate hazard ratios (HR) and 95% confidence intervals (CI) for the association of race–ethnicity with nonfatal MI, CD, and vascular death after adjusting for demographic and CVD risk factors.

RESULTS—Mean age was 68.8 (10.4) years; 52.8% were Hispanic (88% Caribbean-Hispanic). Hispanics were more likely to have hypertension (73.1% vs. 62.2%, $p < .001$) and diabetes (22.0% vs. 13.3%, $p < .001$), and less likely to perform any physical activity (50.1% vs. 69.2%, $p < .001$) compared to non-Hispanic whites (NHW). During a mean 10 years of follow-up there were 154 nonfatal MIs, 186 CD, and 386 vascular deaths. In fully adjusted models, Hispanics had a lower risk of CD (adjusted HR = 0.36, 95% CI: 0.21–0.60), and vascular death (adjusted HR = 0.62, 95% CI: 0.43–0.89), but not nonfatal MI (adjusted HR = 0.95, 95% CI: 0.56–1.60) when compared to NHW.

CONCLUSIONS—We found a “Hispanic paradox” for coronary and vascular deaths, but not nonfatal MI.

Keywords

Hispanic; Paradox; Mortality; Cardiovascular Disease

Address correspondence to: Joshua Z. Willey, MD, MS, 710 West 168th St, New York, NY 10032. Tel.: (212) 305-1710; Fax: (212) 305-1658. jzw2@columbia.edu..

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SUPPLEMENTARY DATA
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INTRODUCTION

Hispanics are the fastest growing segment of the population in the United States (U.S.). Prior studies have consistently identified a high prevalence of vascular disease risk factors among Hispanics, including hypertension, diabetes, physical inactivity, obesity, and the metabolic syndrome (1). Despite the higher prevalence of risk factors and a lower socio-economic status, Hispanics have been observed to have a lower risk of coronary death (CD) compared to non-Hispanic whites (NHW), leading several investigators to refer to a "Hispanic paradox" (2-8). Others, however, have not demonstrated a Hispanic paradox (9-12), particularly after adjustment for cardiovascular disease risk factors (11). One possible explanation for these differences could be differing methodology.

There are few prospective cohort studies examining the Hispanic paradox, and most have focused on Mexican-Americans (13, 14) rather than Caribbean or other foreign-born Hispanics. Prior studies have defined Hispanic ethnicity based on information in death certificates alone rather than self-identification (8, 11). Most studies have also focused on all cardiovascular disease or CD but have not compared these outcomes to other outcomes, such as nonfatal myocardial infarction (MI) (15, 16). Few studies have accounted for the possibility that competing risks for other causes of mortality.

We hypothesized that Hispanics, compared to NHW, would be at increased risk of CD, vascular death (VD), and nonfatal MI in a prospective cohort of participants free of clinical cardiovascular disease at baseline.

METHODS

Recruitment of the Cohort

The Northern Manhattan Study (NOMAS) is a population-based study designed to evaluate the impact of medical, socio-economic, and other risk factors on the incidence of VD in stroke-free cohort. Participants were identified by dual-frame random digit dialing in Northern Manhattan as previously described (17), and were eligible if they met the following criteria: (1) had never been diagnosed with a stroke; (2) were over the age of 39 years; and (3) resided in Northern Manhattan for greater than or equal to 3 months in a household with a telephone. Pre-existing coronary artery disease (CAD) was ascertained via questionnaires capturing self-reported MI, angina, or prior cardiac revascularization. Participants with pre-existing CAD were excluded from this analysis. The study was approved by the Institutional Review Boards at Columbia University Medical Center and the University of Miami. All participants gave informed consent to participate in the study.

Cohort Evaluation

Data regarding baseline status and risk factors were collected through interviews of participants by trained bilingual research assistants. Physical examinations, in-person measurements, and analysis of fasting blood specimens were carried out by study physicians. Race–ethnicity was determined by self-identification in response to a questionnaire modeled after the 2000 U.S. census and conformed to the standard definitions outlined by Directive 15(18). The majority of Hispanics in our cohort did not identify with any particular race after self-identifying as Hispanic. Education was classified as completing high school versus not completing high school. Standardized questions were adapted from the Behavioral Risk Factor Surveillance System regarding the following conditions: hypertension, diabetes, and cigarette smoking (19). Standard techniques were used to measure blood pressure, height, weight, fasting glucose, and lipid panels as described previously (20). Hypertension was defined as blood pressure greater than or equal to 140 mmHg/90 mmHg, a physician diagnosis of hypertension, or a patient's self-report (21).

Diabetes mellitus was defined as fasting blood glucose greater than or equal to 126 mg/dl or the patient's self-report. Fasting blood samples were obtained and lipid profile was measured as described previously (22). Dyslipidemia was defined as an high density lipoprotein cholesterol (HDL-C) less than 40 mg/dl for men and less than 50 mg/dl for women, total cholesterol (TC) greater than 240 mg/dl, or low density lipoprotein cholesterol (LDL-C) greater than 130 mg/dl (23). Physical activity and alcohol intake were ascertained with the use of previously validated self-report questionnaires(24, 25).

Follow-Up and Outcome Measures

Participants are followed annually via phone screening to detect any new cardiac symptoms, interval hospitalizations, medical conditions, or death. Complete loss to follow up is present in less than 1%, and is not associated with race–ethnicity (26). Any participant who screened positive for a cardiac symptom survey was scheduled for an in-person assessment. MI was defined by criteria adapted from the Cardiac Arrhythmia Suppression Trial and the Lipid Research Clinics Coronary Primary Prevention Trial requiring at least two of the three following criteria: (1) ischemic cardiac pain determined to be typical angina; (2) cardiac marker abnormalities defined as abnormal CK-MB fraction or troponin I values; and/or (3) ischemic EKG abnormalities. The presence of an MI was adjudicated by study cardiologists independently after review of all the clinical data (C.J.R., M.R.D., S.H.). CD was defined as death occurring within 30 days of an adjudicated MI, or as sudden cardiac death (27, 28). Sudden cardiac death was defined as death due to cardiac arrest, or sudden unexplained death discovered within 24 hours of the event (29). VD was defined as all deaths due to underlying heart disease (CD, congestive heart failure, other cardiac arrhythmias) or stroke. Cause of death was ascertained through phone discussion with the participant's family (including if in another country), review of medical records (from U.S. and other countries), and, when available, a copy of the death certificate.

A total of 3298 participants were recruited between 1993 and 2001 and follow up is ongoing. After excluding subjects with other race–ethnicity ($n = 69$), a baseline history of pre-existing CAD (MI [$n = 244$], angina [$n = 422$], and/or cardiac interventions [$n = 112$]; total = 548), 2671 participants were included in this analysis.

Statistical Analysis

Baseline characteristics were compared by race–ethnicity using analysis of variance or two-sided t tests for continuous variables, and the χ^2 test for proportions. The 10-year cumulative probabilities of having nonfatal MI, CD, and VD were estimated for overall and each race–ethnicity groups using Kaplan–Meier method. Cox-proportional hazard models were fitted to calculate hazard ratios (HR) and 95% confidence intervals (CI) for nonfatal MI, CD, and VD as outcomes. The parameter estimates were calculated unadjusted (model 1), after adjusting for demographics (model 2: age, sex, and education), and vascular risk factors (model 3: model 2 additionally adjusted for hypertension, diabetes mellitus, waist circumference, tobacco use, LDL-C, HDL-C, TC, moderate alcohol consumption, and physical inactivity).

The standard Cox model assumes that participants can have only one incident event by ignoring competing events and treating them as censored. Therefore we fitted subdistribution proportional hazard models of CD with nonvascular death as the competing risk to examine if the risk of CD changed when competing risk was taken into account (30). We tested for interactions between race ethnicity and age, sex, or cardiovascular disease risk factors. All statistical analyses were carried out with the use of SAS version 9.2 (SAS Institute, Cary, NC) and R2.12.2.

RESULTS

Baseline characteristics for the cohort are outlined in Table 1. The mean age of the cohort was 68.8 (10.4) years; 36.5% were men. The median follow up time for our cohort is 10 years (interquartile range, 9–13). The majority of participants were Hispanic (n = 1445, 52.8%). Among Hispanics, only 87 (6.0%) were born in the continental U.S., median residency in the continental U.S. was 22 years (interquartile range, 14–30), and 88% were Caribbean Hispanic. Dominican-Hispanics (62.5%) were younger ($p < .0001$), less likely to have completed high school ($p < .0001$) and be men ($p = .0002$), and more likely to have Medicaid or no insurance ($p < .0001$) compared to other countries of origin. Further details regarding Hispanics in our study are included in the Supplemental Table. Overall, Hispanics had a greater burden of cardiovascular disease risk factors compared to NHW. Source of information (family informant, medical records, death certificate) regarding cause of death was not statistically different between NHW and Hispanics ($p = .8$). For all our outcomes we found no evidence of an interaction of race–ethnicity with age, sex, years of residence in the U.S., or cardiovascular disease risk factors.

Association between Nonfatal MI and Hispanic Race–Ethnicity

We detected a total of 154 nonfatal MIs during the mean follow-up period of 10 years. Compared to NHW, Hispanic race–ethnicity was associated with a lower risk of nonfatal MI (HR = 0.58, 95% CI: 0.40–0.85) in an unadjusted model, though in multivariable analyses (model 3: adjusted HR = 0.95, 95% CI: 0.56–1.60) Hispanic race–ethnicity was no longer associated with risk of nonfatal MI (Table 2).

Association between CD and Hispanic Race–Ethnicity

There were 186 CDs and 386 VDs. Among the CDs, 31 were deaths from a first incident MI and 155 were sudden cardiac deaths. In unadjusted and adjusted models adjusted for socio-demographics (model 2), as well as cardiovascular disease risk factors (model 3), we found Hispanics were at lower risk of CD compared to NHW (adjusted HR = 0.36, 95% CI: 0.21–0.60) (Table 3). We found a similar association for Hispanics compared to NHW for VD (adjusted HR = 0.62, 95% CI: 0.43–0.89).

Competing Risk Models

Nonvascular deaths, such as those due to cancer or infectious disease, occurred in 521 participants, and were chosen as a competing risk of CD. In fully adjusted competing risk models, Hispanics continued to have a lower risk of CD than NHW (adjusted HR = 0.41, 95% CI: 0.25–0.69) (Table 3).

Models by Country of Origin

We found consistent results when comparing Dominican and non-Dominican Hispanics to NHW, respectively. Compared to NHW, both were not associated with nonfatal MI, whereas both were associated with a similar lower risk of fatal MI. Non-Dominican Hispanic race–ethnicity, however, was not associated with VD.

DISCUSSION

We found that despite the higher burden of cardiovascular risk factors, Hispanics were at lower risk of CD and VD compared to NHW. This association remained even after accounting for multiple confounders and the competing risk of nonvascular death; the magnitude of association was similar to other studies (12). Our results were also consistent among Dominican Hispanics, who were younger and more likely to be uninsured or have Medicaid and to not have completed high school compared to Hispanics from other

countries of origin. There was no difference in risk of nonfatal MI. Like others, we found that CD was driven by sudden cardiac death (1). Unlike others studies (31, 32), however, we found no significant difference for of the risk of CD among non-Hispanic blacks compared to NHW.

The results of our study are unique for several reasons. Most previous studies used administrative databases and focused on mortality alone. Our study is one of the first to demonstrate the existence of the Hispanic paradox in a prospective cohort with a majority of Caribbean Hispanics using a systematic follow-up mechanism of review of medical records and contact with participants, families, and physicians. The NOMAS data collection process makes it unlikely that our findings were due to failure to capture deaths outside of the U.S. We used the same adjudication process for all participants making misclassification based on race–ethnicity less likely. Our study is also one of the few examining various cardiovascular outcomes such as CD, VD, and nonfatal MIs leading to the observation that Hispanics are at a similar risk of MI as NHW, but not necessarily fatal MI. We adjudicated all MIs and sudden cardiac deaths and were therefore able to examine differences between CD and nonfatal MI. We used competing risk models to examine whether the Hispanic paradox was due to Hispanics being more likely to have nonvascular death, and noted that the mortality benefit persisted.

There are several potential explanations for the findings in our and other studies. Hispanics may be at similar risk to NHW for nonfatal MI, but may have a lower short-term and overall mortality subsequent to their MI as others have noted (7). The associations in our study remained after adjusting for differences in important confounders for CD. Moreover, we excluded all subjects with any baseline cardiac disease history to help minimize the healthy cohort effect across all race–ethnic groups. Residual confounding has been another proposed explanation and is ascribed to two phenomena: a “salmon bias” and a “healthy migrant effect” (33). The salmon bias refers to the possibility that patients may return to their country of origin when they retire or develop a fatal illness, leading to loss to follow up being associated with an apparent lower mortality (34). This was not the case in our study because the loss to follow-up was less than 1% (26). An alternative hypothesis is that Hispanics who migrate to the U.S. are overall healthier than other Hispanics, leading to the healthy migrant effect (3, 35). U.S.-born Hispanics, as opposed to foreign born, may have higher rates of cardiovascular disease compared to NHW (14, 35). Few of our Hispanic participants were born in the continental U.S. (6%), however, we found no interaction between Hispanic race–ethnicity and years of residence in the U.S. Other studies have adjusted for these sources of bias and still note the paradox, concluding that there may be an effect of unknown socio-demographic or genetic factors (36).

The results of our study are different than previous examining the Hispanic paradox. Prior studies have used variable definitions of the outcome and different study designs. The National Longitudinal Mortality Study (2) and the National Health Interview Survey (4) found lower mortality among Hispanics compared to NHW after adjusting for cardiovascular comorbidities and demographics; these studies used data from census interviews and linked them to national registries of death certificates, such as the National Death Index. Hispanics may be more likely to be assigned nonspecific and noncardiovascular disease causes of death on death certificates, leading to a potential source of bias (37); these same studies have frequently not used self-identification of race–ethnicity, thereby introducing a potential source of misclassification bias. Noncardiovascular disease related deaths may act as a competing risk that impedes the occurrence of the event of interest and leads to a biased estimate of an effect (38). In several studies, the outcome has been all cardiovascular disease (4), whereas others have been more specific to coronary

heart disease mortality (4), sudden cardiac death (7), or all myocardial infarction (13). Little consensus in addition exists as to the definition of sudden cardiac death (39).

The population of our study differs from others in that they enrolled more continental U.S.-born Hispanics or Mexican-Americans, which may limit comparability to our study. The San Luis Valley Diabetes Study (12) enrolled 1862 participants from Southern Colorado and found that only Hispanics who were diabetic had a lower cardiovascular and coronary heart disease mortality. In the Corpus Christi Heart Project, systematic collection of community wide mortality data led to no evidence of a paradox (11). Higher cardiovascular and coronary heart disease mortality was observed among Hispanics compared to NHW in the San Antonio Heart Study (10).

Our study has several limitations, including an absence of data on income or on neighborhood specific factors (40). The number of nonfatal MIs were small ($n = 154$) and we may have been thus underpowered to note an effect, although with a similar number of fatal MIs ($n = 186$) we found evidence for a paradox. Hispanics were less likely to have completed high school, which could lead to relevant lifestyle, socio-economic status, or access to care differences that we could not capture. The latter could influence access to medical records, potentially leading to information bias, though source of cause of death was not associated with race–ethnicity. We had a greater proportion of older women that could have been an important consideration in studies of cardiovascular disease. Our findings may not be generalizable to all Hispanics; 88% of our Hispanic sample consists of Caribbean-Hispanics from the Dominican Republic, Cuba, and Puerto Rico. Even though Hispanics have a common language, they have different ancestral origins, cultures, diets, and socioeconomic status, which may contribute to different cardiovascular disease outcomes. Our results may not be applicable to Hispanics from South or Central America. Most of the Hispanics in our cohort were not born in the continental U.S., and as such, our results may not be applicable to second generation Hispanics. Our participants chose not to classify themselves as Afro-Caribbean or of European descent, which may have provided an additional source for exploring the Hispanic paradox. The Hispanics in our cohort identified more strongly with country of origin, and we found no difference in risk of outcomes when stratifying by country of origin. We did not collect data regarding treatment and therefore cannot comment on any differences in outcomes driven by medical care. Our cohort is older and we may have had a selection bias toward healthier community dwellers who had not already died from MI or had CAD at baseline. This is unlikely to be a complete explanation for our findings as it would have led to nondifferential misclassification.

In conclusion we found that Caribbean Hispanics were at lower risk of CD and vascular death compared to NHW, but at no lower risk of nonfatal MI. Despite the potential “heart disease mortality advantage” our results should be interpreted with caution as our findings could be attributable to residual confounders. Our results nonetheless support that there may be a “Hispanic paradox” for CD, but not nonfatal MI with the subsequent potential long term consequences from this condition. Further studies should confirm our findings. Public health campaigns should continue to target Hispanics as cardiovascular disease remains the leading cause of morbidity and mortality (1).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Selected Abbreviations and Acronyms

US	United States
CD	coronary death
NHW	non-Hispanic white
NHB	non-Hispanic black
NOMAS	Northern Manhattan Study
HDL-C	high density lipoprotein cholesterol
LDL-C	low density lipoprotein cholesterol
VD	vascular death
MI	myocardial infarction
CVD	cardiovascular disease
CAD	coronary artery disease
TC	total cholesterol

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TABLE 1

Baseline characteristics of participants in the NOMAS without pre-existing coronary artery disease

	Overall (*n* = 2671)	Hispanic (*n* = 1445)	NHB (*n* = 681)	NHW (*n* = 545)	*p* for difference*
Mean age (SD)	68.8 (10.4)	65.9 (9.5)	71.6 (10.5)	72.9 (10.2)	<.001
Men, *n* (%)	974 (36.5)	542 (37.5)	225 (33.0)	207 (38.0)	.1
Education, completed high school, *n* (%)	1206 (45.2)	321 (22.2)	432 (63.4)	453 (83.1)	<.001
Risk factors, *n* (%)					
Hypertension§	1931 (72.3)	1056 (73.1)	536 (78.7)	339 (62.2)	<.001
Diabetes mellitus‡	543 (20.4)	317 (22.0)	154 (22.6)	72 (13.3)	<.001
Total cholesterol >240 mg/dl	421 (16.3)	223 (16.0)	101 (15.4)	97 (18.4)	.3
HDL-C (men <40 mg/dl, women <50 mg/dl)	1377 (53.5)	887 (63.6)	255 (39.0)	235 (44.6)	<.001
LDL-C (> 130 mg/dl)	1199 (47.0)	654 (47.5)	272 (41.9)	273 (52.0)	.002
Current smoking	479 (17.9)	239 (16.5)	161 (23.7)	79 (14.5)	<.001
Mean waist circumference in inches (SD)	36.7 (5.0)	36.8 (4.5)	36.8 (5.5)	36.1 (5.6)	.02
Mean body-mass index (SD)	27.8 (5.6)	28.3 (5.0)	28.1 (6.4)	26.2 (5.6)	<.0001
Any physical activity	1552 (58.1)	724 (50.1)	451 (66.2)	377 (69.2)	<.001
Moderate alcohol intake†	913 (34.2)	446 (30.9)	221 (32.5)	246 (45.1)	<.001
Framingham 10-year risk score (proportion at >20% risk)	546 (21.3)	261 (18.8)	151 (23.4)	134 (25.5)	.002
Nonfatal myocardial infarction, *n* (%)	154 (5.8)	75 (5.2)	36 (5.3)	43 (7.9)	.06
Coronary deaths, *n* (%)	186 (7.0)	56 (3.9)	66 (9.7)	64 (11.7)	<.001
Vascular deaths, *n* (%)	386 (13.7)	136 (9.4)	130 (19.1)	102 (18.7)	<.001
Nonfatal myocardial infarction, 10-year cumulative risk (%)	5.7	4.7	5.5	9.0	.01
Coronary deaths, 10-year cumulative risk (%)	5.7	2.8	8.9	10.2	<.001
Vascular deaths, 10-year cumulative risk (%)	11.5	7.3	17.7	15.7	<.001

HDL-C = high density lipoprotein cholesterol; LDL-C = low density lipoprotein cholesterol; NHB = non-Hispanic blacks; NHW = non-Hispanic whites; NOMAS = Northern Manhattan Study.

*Chi-square two degrees of freedom for categorical variables; analysis of variance for continuous variables.

†Moderate alcohol intake: 1–2 servings of alcohol/day.

‡Diabetes defined as: fasting blood glucose ≥126 mg/dl or the patient's self-report of such a history, or insulin or hypoglycemic use.

§Hypertension defined as: systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg based on the average of two measurements, a physician diagnosis of hypertension, or a patient's self-report of a history of hypertension or antihypertensive use.

TABLE 2

Association between race–ethnicity and nonfatal myocardial infarction (n = 154) in the NOMAS

	Model 1* HR (95% CI)	Model 2† HR (95% CI)	Model 3‡ HR (95% CI)
Hispanic (reference: NHW)	0.58 (0.40–0.85)	0.89 (0.54–1.46)	0.95 (0.56–1.60)
NHB (reference: NHW)	0.66 (0.43–1.03)	0.74 (0.47–1.17)	0.80 (0.49–1.29)

CI = confidence interval; HR = hazard ratio; NHB = non-Hispanic blacks; NHW = non-Hispanic whites; NOMAS = Northern Manhattan Study.

*Univariate analysis.

†Model 1 also adjusted for age, sex, and other socio-demographic factors (completing high school, having Medicaid, or no insurance).

‡Model 2 further adjusted for cardiovascular disease risk factors (hypertension, diabetes mellitus, waist circumference, tobacco use, low density lipoprotein cholesterol, high density lipoprotein cholesterol, total cholesterol, moderate alcohol consumption, and not performing any physical activity).

TABLE 3

Association between race–ethnicity and coronary (n = 186) and vascular death (including coronary death, n = 386) in the NOMAS

	Model 1* HR (95% CI)	Model 2† HR (95% CI)	Model 3‡ HR (95% CI)
Cox proportional hazards models (coronary death)			
Hispanic (reference: NHW)	0.30 (0.21–0.43)	0.42 (0.26–0.67)	0.36 (0.21–0.60)
NHB (reference: NHW)	0.84 (0.59–1.18)	0.86 (0.60–1.23)	0.73 (0.49–1.07)
Cox proportional hazards models (vascular death)			
Hispanic (reference: NHW)	0.46 (0.35–0.59)	0.65 (0.46–0.91)	0.62 (0.43–0.89)
NHB (reference: NHW)	1.04 (0.80–1.34)	1.10 (0.84–1.44)	0.95 (0.71–1.26)
Competing risk models§(ISP) (coronary death)			
Hispanic (reference: NHW)	0.32 (0.23–0.46)	0.45 (0.28–0.72)	0.41 (0.25–0.69)
NHB (reference: NHW)	0.83 (0.59–1.17)	0.85 (0.59–1.20)	0.72 (0.49–1.07)
Competing risk models§(ISP) (vascular death)			
Hispanic (reference: NHW)	0.50 (0.30–0.64)	0.69 (0.49–0.96)	0.70 (0.48–1.00)
NHB (reference: NHW)	1.03 (0.79–1.33)	1.08 (0.83–1.41)	0.95 (0.71–1.28)

CI = confidence interval; HR = hazard ratio; NHB = non-Hispanic blacks; NHW = non-Hispanic whites; NOMAS = Northern Manhattan Study.

*Univariate analysis.

†Model 1 also adjusted for age, sex, and other socio-demographic factors (completing high school, having Medicaid, or no insurance).

‡Model 2 further adjusted for cardiovascular disease risk factors (hypertension, diabetes mellitus, waist circumference, tobacco use, low density lipoprotein cholesterol, high density lipoprotein cholesterol, total cholesterol, moderate alcohol consumption, and not performing any physical activity).

§Nonvascular death as a competing risk.