Race-Ethnic Disparities in the Impact of Stroke Risk Factors

The Northern Manhattan Stroke Study

Ralph L. Sacco, MS, MD; Bernadette Boden-Albala, MPH; Gregory Abel, MD; I-Feng Lin, DrPH; Mitchell Elkind, MD; W. Allen Hauser, MD; Myunghee C. Paik, PhD; Steven Shea, MS, MD

Background and Purpose—Stroke risk factors have been determined in large part through epidemiological studies in white cohorts; as a result, race-ethnic disparities in stroke incidence and mortality rates remained unexplained. The aim in the present study was to compare the prevalence, OR, and etiological fraction (EF) of stroke risk factors among white, blacks, and Caribbean Hispanics living in the same urban community of northern Manhattan.

Methods—In this population-based incident case-control study, cases (n=688) of first ischemic stroke were prospectively matched 1:2 by age, sex, and race-ethnicity with community controls (n=1156). Risk factors were determined through in-person assessment. Conditional logistic regression was used to calculate adjusted ORs in each race-ethnic group. Prevalence and multivariate EFs were determined in each race-ethnic group.

Results—Hypertension was an independent risk factor for whites (OR 1.8, EF 25%), blacks (OR 2.0, EF 37%), and Caribbean Hispanics (OR 2.1, EF 32%), but greater prevalence led to elevated EFs among blacks and Caribbean Hispanics. Greater prevalence rates of diabetes increased stroke risk in blacks (OR 1.8, EF 14%) and Caribbean Hispanics (OR 2.1 P<0.05, EF 10%) compared with whites (OR 1.0, EF 0%), whereas atrial fibrillation had a greater prevalence and EF for whites (OR 4.4, EF 20%) compared with blacks (OR 1.7, EF 3%) and Caribbean Hispanics (OR 3.0, EF 2%). Coronary artery disease was most important for whites (OR 1.3, EF 16%), followed by Caribbean Hispanics (OR 1.5, EF 6%) and then blacks (OR 1.1, EF 2%). Prevalence of physical inactivity was greater in Caribbean Hispanics, but an elevated EF was found in all groups.

Conclusions—The prevalence, OR, and EF for stroke risk factors vary by race-ethnicity. These differences are crucial to the etiology of stroke, as well as to the design and implementation of stroke prevention programs. (Stroke. 2001;32: 1725-1731.)

Key Words: data interpretation, statistical ■ epidemiology ■ risk factors ■ stroke

Recent projections estimate that the annual incidence of stroke in the United States among all races is closer to 700 000 than the previously reported incidence of 550 000.1 Stroke continues to have a disproportionate impact on mortality rates for blacks and Hispanics compared with whites.2-5 Mortality differences have remained significant even after accounting for differences in socioeconomic status.6 Incidence data from the Northern Manhattan Stroke Study (NOMASS) have demonstrated race-ethnic differences in stroke incidence: blacks had a 2.4-fold increased annual stroke incidence and Caribbean Hispanics had a 2-fold increased annual stroke incidence compared with whites living in the same community.7 The aging and rapid growth of the black and Hispanic populations in the United States have the potential to lead to increases in these disparities.

The reasons for the race-ethnic disparities in stroke incidence and mortality are not clear. Potential explanations include variations in risk factor potency, prevalence, and treatment arising from environmental and genetic factors. Although no study has systematically examined differences in the effect of risk factors in different race-ethnic groups, the literature has consistently shown race-ethnic variations in the prevalence of cardiovascular risk factors.8,9 For example, blacks have the highest prevalence of hypertension regardless of geographic location,10 although Caribbean-born blacks have a lower prevalence of hypertension than those living in the southern or northeastern United States.11 Other studies have reported that diabetes is more common in blacks, whereas coronary artery disease and atrial fibrillation are more common in whites.12,13
The data on stroke among Hispanic Americans are scarce and inconclusive. Moreover, studies are hampered by the heterogeneity among Hispanics who originate from Mexico, South America, and the Caribbean basin. Hispanics are the most rapidly growing minority in this country. Between 1980 and 1990, the Hispanic population increased by 39% compared with 7% for the overall US population. The Hispanic population accounted for 11% of the total US population in 1998 and is estimated to be 19% in 2030. Caribbean Hispanics are 1 of the 2 major groups of Hispanics in the United States and are relatively recent immigrants from the Caribbean Islands, including the Dominican Republic and Puerto Rico. They are the fastest growing ethnic group in the northeastern United States. The northern Manhattan community provides a unique opportunity for the study of Caribbean Hispanics. According to the 1990 census, 63% of the 260 000 persons living in this area identified themselves as Hispanic.

The Hispanic population enrolled in NOMASS is representative of the underlying Caribbean Hispanic community in New York City. This population is primarily Dominican (62%), with an additional 14% being Puerto Rican, 12% being Cuban, and 12% reporting origins from various Caribbean islands and South America.

Despite the mounting evidence that stroke risk factors may differ among race-ethnic groups, public health programs and national guidelines for stroke prevention continue to focus on the identification of risk factors in the general population without addressing the specific needs of race-ethnic groups. The ability to provide targeted information to different race-ethnic groups is dependent on epidemiological studies capable of comparing risk differences in these groups; definitive differences among race-ethnic groups are best measured through a comparison of individuals from the same population or community. This population-based methodology helps to minimize biases that may arise from comparisons of groups living in different physical, social, and healthcare environments. The aim of the present study was to use a population-based case-control design to determine whether the OR, prevalence, and etiological fraction (EF) for stroke risk factors vary among whites, blacks and Caribbean Hispanics living in the same geographically defined community.

Subjects and Methods
NOMASS is an ongoing, prospective, population-based epidemiological study designed to determine stroke incidence, risk factors, and prognosis in a multiethnic, urban population. Northern Manhattan consists of the area north of 145th Street, south of 218th Street, bordered on the west by the Hudson River, and bordered on the east by the Harlem River. In 1990, ~260 000 persons lived in the region, of whom 40% were aged >39 years.

Selection of Patients
Patients eligible for the population-based case-control portion of NOMASS were prospectively enrolled if they met the following criteria: (1) diagnosed with first cerebral infarction between July 1, 1993, and June 30, 1997; (2) were aged >39 years at onset of the stroke; and (3) resided in the northern Manhattan community in a household with a telephone. Patients with TIA (ie, neurological deficits lasting <24 hours and no ischemic infarct found on brain imaging) were excluded. Prospective case surveillance consisted of daily screening of all admissions, discharges, and head CT scan logs at the Presbyterian Hospital in New York City, the only hospital in the community. Statewide Planning and Research Cooperative System (SPARCS) data indicate that ~80% of all patients in northern Manhattan with stroke are hospitalized at Presbyterian Hospital. To ensure complete incident stroke enumeration in the region, cases were also identified through discharge lists from 14 hospitals in nearby areas. Ongoing community-based surveillance for nonhospitalized patients with stroke was performed through random household telephone surveys and frequent interval contacts with community physicians, senior citizen centers, visiting nurse services, and other social and cultural community agencies. Details are enumerated elsewhere.

Patients diagnosed with stroke, as well as a variety of other neurological syndromes (eg, aphasia, hemiparesis, weakness, coma, syncope), were screened by a study research assistant, and the case was discussed with a study neurologist to confirm eligibility. With permission from the attending physician, written informed consent was obtained from the patient or the family. The study was approved by the institutional review boards at Columbia-Presbyterian Medical Center and the other 14 hospitals.

Selection of Control Subjects
Community control subjects were enrolled if they (1) had never been diagnosed with a stroke, (2) were aged >39 years, and (3) resided for ≥3 months in a household with a telephone in northern Manhattan. Stroke-free subjects were identified by random digit dialing using dual frame sampling to identify both published and unpublished telephone numbers. When a household was contacted, the research objectives were explained, and a resident older than 39 years was interviewed briefly to record age, sex, race-ethnicity, and risk factors. These telephone interviews were conducted by Audits and Surveys, Inc, using trained bilingual interviewers. The telephone response rate was 93%.

Interview data from control-eligible subjects was downloaded to the NOMASS computer system and assigned to cells defined by age, sex, and race-ethnicity. Subjects were randomly selected from cells matched to the accumulating case group by age, sex, and race-ethnicity and were recontacted by the NOMASS staff and invited to participate in the study. Appointments were made for in-person evaluations at the hospital or home for those who could not come in person (7% were conducted at home). Two concurrent control subjects were selected and matched to each stroke case by age within 5 years, sex, and race-ethnicity. The overall response rate for selected and matched control subjects was 75%. At the time of the in-person visit, written informed consent was obtained.

Definition of Race-Ethnicity
Race-ethnicity was based on self-identification through a series of interview questions modeled after the US census and conforming to the standard definitions outlined in Directive 15.22 These government directives have been mandated for all epidemiological research funded by National Institutes of Health. Two questions were asked: (1) Are you of Hispanic/Spanish origin? (no/yes), and (2) Which of the following best describes your race? (white; black or African-American; Eskimo or Aleutian (Alaskan native); Asian or Pacific Islander; other [specify]). All participants who identified themselves as Hispanic were classified as such regardless of the answer to question 2. All participants who classified themselves as white without any Hispanic origin or as black without any Hispanic origin were classified as white, non-Hispanic, or black, non-Hispanic, respectively. For this study, Caribbean Hispanics were classified as those from the Dominican Republic, Puerto Rico, Cuba, and other Caribbean basin countries.

Index Evaluation of Patients and Control Subjects
Data were collected through interview of the patients and control subjects by trained bilingual research assistants using standardized data collection instruments, review of the medical records, physical and neurological examination by study physicians, and fasting blood specimens for lipid and glucose measurements. When the subject was unable to answer questions due to aphasia, coma, dementia, or
other conditions, a proxy who was knowledgeable about the patient’s history was interviewed. Proxy respondents were used for 26% of patients and 1% of control subjects. Stroke-free control subjects were interviewed in person and evaluated in the same manner as patients.

Subjects were interviewed regarding sociodemographic characteristics, stroke risk factors, and other medical conditions. Standardized questions were adapted from the Centers for Disease Control and Prevention Behavioral Risk Factor Surveillance System regarding the following conditions: hypertension, diabetes, hypercholesterolemia, peripheral vascular disease, TIA, cigarette smoking, alcohol use, and cardiac conditions such as myocardial infarction, coronary artery disease, angina, congestive heart failure, atrial fibrillation, other arrhythmias, and valvular heart disease. Standard techniques were used to measure blood pressure, height, weight, and fasting glucose levels. Definite hypertension was defined as a systolic blood pressure recording of ≥160 mm Hg or a diastolic blood pressure recording of ≥95 mm Hg based on the average of the 2 blood pressure measurements or as a patient’s self-report of a history of hypertension or antihypertensive drug use. Diabetes mellitus was defined as a fasting glucose level of >140 mg/dL (7.7 mmol/L), the patient’s self-report of such a history, or insulin or hypoglycemic drug use. Coronary artery disease was defined as a history of either myocardial infarction, angina, CABG, or angioplasty or the current use of cardiac medications. To be included in the definition of atrial fibrillation, subjects reported they had been told they had chronic or paroxysmal atrial fibrillation by a health professional such as a physician. For this analysis, smoking was defined as currently smoking cigarettes, heavy smoking was defined as ≥40 years of smoking or smoking ≥1 pack/d for ≥20 years, and heavy alcohol use was defined as current drinking of ≥5 drinks/d. Physical activity was assessed through a questionnaire adapted from the National Health Interview Survey that recorded the frequency and duration of 14 activities during the 2 prior weeks. Body mass index was calculated as weight (kg) divided by height (meters) squared, and obesity was defined as body mass index of ≥27.8 for men and ≥27.3 for women.

Statistical Analysis
The OR, prevalence, and EF were calculated for the complete sample and for each race-ethnic group. Specifically, the risk factors evaluated among whites, blacks, and Caribbean Hispanics were hypertension, diabetes, atrial fibrillation, coronary artery disease, current smoking, heavy alcohol use, and physical inactivity. Multivariate adjusted OR and 95% CIs were calculated using conditional logistic regression. Prevalence rates were estimated from the proportion of individuals identified with the risk factor among population-based control subjects. The univariate EF, or population attributable risk, provided an estimate of the proportion of the strokes in the control subjects. The univariate EF, or population attributable risk, was calculated with the equation EF = P(OR−1)/[1 + P(OR−1)], where P is prevalence.

In addition, multivariate EFs were estimated using multivariate ORs and conditional prevalences (ie, prevalence of a risk factor given the presence of the other risk factors). Prevalence rates were compared among whites, blacks, and Caribbean Hispanics. CIs excluding 1.0 were considered to demonstrate significantly different prevalence rates. We used the bootstrap method to calculate CIs and demonstrate significance for each of the multivariate EFs.

Results
From July 1, 1993, to June 30, 1997, 688 patients with first ischemic stroke and 1156 stroke-free control subjects were enrolled (Table 1). The mean age of the subjects was 70 years, and 56% of patients and 60% of control subjects were women. The overall race-ethnic distribution of cases was 50% Caribbean Hispanic, 28% black, and 20% white; 2% were classified as “others.” Control subjects were more educated than stroke patients, with 48% of control subjects and 35% of patients having completed high school.

The multivariate model for stroke risk factors in the overall study sample demonstrated that hypertension, atrial fibrillation, diabetes, coronary artery disease, physical inactivity, and heavy alcohol use (≥5 drinks/d) were independent risk factors for ischemic stroke (Table 2). These ORs were calculated after matching by age, sex, and race-ethnicity and are adjusted for socioeconomic confounding by adding education to the multivariate model. Neither current cigarette smoking nor heavy smoking was an independent risk factor for ischemic stroke in this older cohort and was not controlled.

TABLE 1. Demographics for NOMASS Participants

<table>
<thead>
<tr>
<th></th>
<th>Patients (n=688)</th>
<th>Control Subjects (n=1156)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age, y</td>
<td>70±13</td>
<td>70±12</td>
</tr>
<tr>
<td>Women, %</td>
<td>56</td>
<td>60</td>
</tr>
<tr>
<td>Race-ethnicity, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>20</td>
<td>22</td>
</tr>
<tr>
<td>Black</td>
<td>28</td>
<td>32</td>
</tr>
<tr>
<td>Hispanic</td>
<td>50</td>
<td>45</td>
</tr>
<tr>
<td>Education completed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High school, %</td>
<td>35</td>
<td>48</td>
</tr>
</tbody>
</table>

TABLE 2. Overall Multivariate ORs for NOMASS Matched for Age, Sex, and Race-Ethnicity and Adjusted for the Listed Risk Factors

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>OR (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>2.0 (1.5–2.5)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1.7 (1.3–2.2)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>2.5 (1.6–3.9)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>1.4 (1.1–1.9)</td>
<td>0.01</td>
</tr>
<tr>
<td>Physical inactivity</td>
<td>2.7 (2.1–3.4)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Heavy alcohol (≥5 drinks/d)</td>
<td>2.3 (1.0–5.0)</td>
<td>0.04</td>
</tr>
<tr>
<td>Current smoking</td>
<td>1.1 (0.8–1.4)</td>
<td>0.7</td>
</tr>
<tr>
<td>Education</td>
<td>0.5 (0.3–0.6)</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Figure 1. Hypertension: OR and prevalence in NOMASS. Matched for age and sex and adjusted for diabetes mellitus, atrial fibrillation, coronary artery disease, physical inactivity, and education. *Significant difference in prevalence compared with whites (P<0.05).
for or included in further analysis. The prevalence of any smoking was 40%, whereas that of current smoking was only 18%. Also, we excluded heavy alcohol use from the stratified models because the number of heavy drinkers by race-ethnic group was too small.

Figures 1 through 5 show a comparison of the OR and prevalence for each risk factor by race-ethnic subgroup. Table 3 demonstrates the EF and 95% CI for each risk factor stratified by race-ethnicity. Among whites, the OR, EF, and prevalence were greatest for atrial fibrillation (OR 4.4, EF 20%) and coronary artery disease (OR 1.3, EF 16%). For atrial fibrillation, we found a significantly greater OR ($P<0.003$) and EF among whites than among Caribbean Hispanics.

The EF and prevalence of hypertension were greatest among blacks (hypertension 62%, EF 37%) and Caribbean Hispanics (hypertension 58%, EF 32%) compared with whites (hypertension 43%, EF 25%). In addition, both Caribbean Hispanics and blacks demonstrated a significantly greater prevalence of diabetes, and the OR for diabetes was significant among Caribbean Hispanics compared with that among whites ($P<0.05$). The EF for diabetes was small for the elderly white subgroup. Finally, physical inactivity was a strong stroke risk factor for all 3 race-ethnic groups, although the prevalence was significantly greater among Caribbean Hispanics, and the EF was twice as great among Caribbean Hispanics (EF 38%) as among whites (EF 18%).

**Discussion**

We found variation in the prevalence, ORs, and EFs for key stroke risk factors among whites, blacks, and Caribbean Hispanics living in northern Manhattan. Although differences in the OR for stroke risk have previously been reported between whites and blacks for factors such as cardiac disease and diabetes,8–13 this is the first study to examine such differences in a community-based, multiethnic population using the EF and controlling for common stroke risk factors. The EF estimates the proportion of strokes attributable to a specific stroke risk factor. Specifically, the use of the EF in this study provides information on the proportion of the population at risk of stroke who would be eliminated with the control of a specific risk factor. The EF is a function of both the OR and the prevalence of a risk factor and provides

![Figure 2](image2.png)

**Figure 2.** Diabetes mellitus: OR and prevalence in NOMASS. Matched for age and sex and adjusted for hypertension, atrial fibrillation, coronary artery disease, physical inactivity, and education. #Significant difference in OR compared with whites ($P<0.05$); *significant difference in prevalence compared with whites ($P<0.02$).

![Figure 3](image3.png)

**Figure 3.** Atrial fibrillation: OR and prevalence in NOMASS. Matched for age and sex and adjusted for hypertension, diabetes mellitus, coronary artery disease, physical inactivity, and education. #Significant difference in OR compared with whites ($P<0.05$); *significant difference in prevalence compared with whites ($P<0.02$).

![Figure 4](image4.png)

**Figure 4.** Coronary artery disease: OR and prevalence in NOMASS. Matched for age and sex and adjusted for hypertension, diabetes mellitus, atrial fibrillation, physical inactivity, and education. *Significant difference in prevalence compared with whites ($P<0.05$).

![Figure 5](image5.png)

**Figure 5.** Physical inactivity: OR and prevalence in NOMASS. Matched for age and sex and adjusted for hypertension, diabetes mellitus, atrial fibrillation, coronary artery disease, and education. *Significant difference in prevalence compared with whites ($P<0.04$).
important information that is useful in the planning of stroke intervention and implementing health policy in different subpopulations. The usefulness of the EF lies in the ability to use this calculation to measure the impact of the risk factor in a population and thus to estimate the benefit of risk elimination. EF is best used when risk factors are considered to be modifiable and more proximate in the causal chain of events leading to disease such as stroke. Although statistically significant differences in the EF of risk factors were found only with atrial fibrillation, the potential public health importance is no less significant. Our study demonstrates significant differences in prevalence and risk, as well as trends toward significance in EF. We demonstrate the disproportionate burden of these modifiable risk factors among whites, blacks, and Caribbean Hispanics and suggest that differential prevention strategies by race-ethnicity are needed to reduce the number of strokes.

Blacks and Caribbean Hispanics had a greater stroke EF for hypertension than did whites in the present study. It has been shown that both the prevalence and the effect of hypertension are greater among blacks than among whites using the more traditional measures of OR and prevalence. Results of preliminary ecological and cross-sectional studies of hypertension in Hispanic Americans compared with non-Hispanic whites have been conflicting for Hispanics, depending on the place of origin of Hispanics. A limitation of these studies was the inability to make direct comparisons of whites, blacks, and Hispanics living in the same community and to provide a summary measurement of differential risk for public health planning.

Our findings regarding the variation in the EF for hypertension emphasize the need for specific risk factor targeting in various communities or populations with respect to blood pressure control. Possible explanations for the greater EF of hypertension for blacks include genetic, environmental, and treatment factors. Researchers have found several gene mutations implicated in the pathogenesis of hypertension that have a higher incidence in blacks. In addition, several studies have found a correlation between the prevalence of hypertension in blacks and both higher levels of lipoprotein(a) and greater thickness of the left ventricular septal and posterior walls. It has also been shown that African Americans respond less favorably to certain antihypertensive drug therapies, particularly β-blockers and ACE inhibitors. Sociocultural, economic, and environmental determinants may also influence the potency of certain risk factors such as hypertension in blacks and Hispanics. For example, it has been demonstrated that blacks are less aware of their hypertension status, perhaps as a result of poorer education on health issues or differences in access to primary medical care. Among Mexican Americans, data from the San Antonio Heart Study demonstrated an inverse association between diastolic blood pressure and sociocultural status, especially education and “structural assimilation,” described as the “process by which minority groups enter the clubs, cliques and institutions of a broader society.”

For atrial fibrillation, the prevalence, OR, and EF differences found among whites, blacks, and Caribbean Hispanics confirm earlier studies that demonstrated a greater likelihood of atrial fibrillation as a stroke risk factor among whites versus blacks. Others have suggested that although prevalence differences in atrial fibrillation between white and blacks may be small, a differential potency of the risk factor may contribute to it having a greater impact in whites. Findings from the Cardiovascular Health Study suggested the prevalence of atrial fibrillation was similar between blacks and whites; however, this comparison was made in a very small group of blacks, and EF was not calculated. There have been no studies that assessed atrial fibrillation in Caribbean Hispanics compared with both whites and blacks. Although some of the race-ethnic differences in the burden of atrial fibrillation may be attributed to the younger age of the present study Caribbean Hispanics (mean age 66 years) and blacks (mean age 68 years) compared with the whites (mean age 77 years), age would not affect the ORs, because patients and control subjects were matched by age. The present finding that whites have a greater EF for atrial fibrillation (20%) than Caribbean Hispanics (2%) and blacks (3%) suggests that more aggressive identification and control of this risk factor are especially needed among white communities.

One of the more important findings from this study is the elevated EF for physical inactivity found in all 3 race-ethnic groups. Numerous studies have shown that physical fitness is an independent predictor of death from cardiovascular disease among healthy, middle-aged men and women. Recent findings continue to provide evidence of the overwhelming benefit of physical activity on stroke prevention. Criticisms of this literature include the idea that subjects who report a lack of physical activity may have an undiagnosed or subclinical condition that prevents such activity and that physically fit subjects may adopt other

---

**TABLE 3. EFs and 95% CIs for Stroke Risk Factors Among Whites, Blacks, and Hispanics for NOMASS Participants**

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Whites (95% CI)</th>
<th>Blacks (95% CI)</th>
<th>Hispanics (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>0.25 (0.01 to 0.45)</td>
<td>0.37 (0.16 to 0.57)</td>
<td>0.32 (0.15 to 0.43)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>0 (0.00 to 0.10)</td>
<td>0.14 (0.04 to 0.25)</td>
<td>0.10 (0.04 to 0.15)</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>0.20 (0.06 to 0.39)</td>
<td>0.03 (0.00 to 0.08)</td>
<td>0.02 (0.00 to 0.04)</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>0.16 (0.008 to 0.35)</td>
<td>0.02 (0.06 to 0.13)</td>
<td>0.06 (0.001 to 0.14)</td>
</tr>
<tr>
<td>Physical activity</td>
<td>0.18 (0.07 to 0.36)</td>
<td>0.29 (0.19 to 0.44)</td>
<td>0.38 (0.24 to 0.44)</td>
</tr>
</tbody>
</table>

*Significantly different EF attributed to atrial fibrillation in whites compared with blacks and Hispanics.*
health-promoting habits. The present work suggests that decreasing the risk burden of physical inactivity could substantially alter stroke risk among all race-ethnic groups. In addition, targeted intervention for Caribbean Hispanics is crucial, because they have the highest EF for lack of physical inactivity (38%), as well as the highest OR (3.3) and prevalence (37%).

The results from the NOMASS support the idea that effective educational and interventional stroke programs should take into account differences in OR, prevalence, and EF of stroke risk factors in different race-ethnic groups. This notion is particularly important to current public health initiatives because there continues to be a significant disparity in both stroke incidence and mortality rates among race-ethnic groups. Stroke prevention guidelines must account for these differences in statements to healthcare professionals. The failure to use information that describes the differences in risk factor potency in different race-ethnic groups when creating prevention and treatment programs may be responsible for the continued failure to achieve goals in terms of risk factor control and regulation. Targeted public health programs, when designed in conjunction with data on race-ethnic EF, may be the most cost-effective means to reduce the stroke burden in multietnic communities.

Acknowledgments
This work was supported by grants from the National Institute of Neurological Disorders and Stroke (R01-NS-27517, R01-NS-29993, and T32-NS-07153) and the General Clinical Research Center (2-M01-RR-00645). We acknowledge the support of Dr J.P. Mohr, MD, MS, Director of Cerebrovascular Research, and the help provided by Sarah Evers, Yumila Soriano, and Chris Kleeman.

References
Race-Ethnic Disparities in the Impact of Stroke Risk Factors: The Northern Manhattan Stroke Study
Ralph L. Sacco, Bernadette Boden-Albala, Gregory Abel, I-Feng Lin, Mitchell Elkind, W. Allen Hauser, Myunghee C. Paik and Steven Shea

Stroke. 2001;32:1725-1731
doi: 10.1161/01.STR.32.8.1725

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/32/8/1725

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/