Ischemic Stroke Subtype Incidence Among Whites, Blacks, and Hispanics
The Northern Manhattan Study

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Background—Stroke incidence is greater in blacks than in whites; data on Hispanics are limited. Comparing subtype-specific ischemic stroke incidence rates may help to explain race-ethnic differences in stroke risk. The aim of this population-based study was to determine ischemic stroke subtype incidence rates for whites, blacks, and Hispanics living in one community.

Methods and Results—A comprehensive stroke surveillance system incorporating multiple overlapping strategies was used to identify all cases of first ischemic stroke occurring between July 1, 1993, and June 30, 1997, in northern Manhattan. Ischemic stroke subtypes were determined according to a modified NINDS scheme, and age-adjusted, race-specific incidence rates calculated. The annual age-adjusted incidence of first ischemic stroke per 100 000 was 88 (95% CI, 75 to 101) in whites, 149 (95% CI, 132 to 165) in Hispanics, and 191 (95% CI, 160 to 221) in blacks. Among blacks compared with whites, the relative rate of intracranial atherosclerotic stroke was 5.85 (95% CI, 1.82 to 18.73); extracranial atherosclerotic stroke, 3.18 (95% CI, 1.42 to 7.13); lacunar stroke, 3.09 (95% CI, 1.86 to 5.11); and cardioembolic stroke, 1.58 (95% CI, 0.99 to 2.52). Among Hispanics compared with whites, the relative rate of intracranial atherosclerotic stroke was 5.00 (95% CI, 1.69 to 14.76); extracranial atherosclerotic stroke, 1.71 (95% CI, 0.80 to 3.63); lacunar stroke, 2.32 (95% CI, 1.48 to 3.63); and cardioembolic stroke, 1.42 (95% CI, 0.97 to 2.09).

Conclusions—The high ischemic stroke incidence among blacks and Hispanics compared with whites is due to higher rates of all ischemic stroke subtypes. (Circulation. 2005;111:1327-1331.)

Key Words: cerebral infarction ■ epidemiology ■ ethnic groups ■ stroke

The demographic profile of the US population is changing, with increasing numbers of elderly and minority groups. According to the 2000 American census, 12.3% of Americans identify their race as black, and 12.5% identify their ethnicity as Hispanic.1 The burden of disease, particularly cerebrovascular disease, is greater among black Americans than among whites; blacks have a shorter life expectancy, higher prevalence of cardiovascular risk factors, higher stroke incidence, and higher stroke mortality than whites. Data on stroke risk in Hispanics are more limited and inconsistent. Studies of Caribbean American Hispanics demonstrate a stroke incidence higher than in whites.3 Past studies of Mexican American Hispanics have shown stroke incidence rates that were similar to or lower than those of whites,4 but recent studies have also shown higher incidence rates.5

Prior studies have examined the relative proportions of ischemic subtypes among races.6–10 Ischemic stroke subtype incidence may also vary by race-ethnicity, but these differences have not been well documented,11–13 and no current work includes whites, blacks, and Hispanics living in the same region. The aim of this population-based study was to determine ischemic stroke subtype incidence and distribution in a multiethnic community.

Methods

The Northern Manhattan Study (NOMAS) is an ongoing, prospective, population-based epidemiological study designed to determine stroke incidence, risk factors, and outcomes in a multiethnic, urban population. Northern Manhattan consists of a well-defined area of New York City; in 1990, 71% of the 210 000 individuals who resided this area were 20 years of age; 22% were white, 13% were black, and 64% were Hispanic.

Identification of Subjects

An active, prospective community surveillance program was used to find all cases of first stroke occurring in northern Manhattan between July 1, 1993, and June 30, 1997. All individuals who were ≳20 years of age, had resided in the area for ≳3 months, and had had a first stroke (as defined by the National Institute of Neurological Disorders and Stroke [NINDS] classification of cerebrovascular diseases III)14
within the study time period were included. Patients with transient ischemic attack (TIA; ie, a neurological deficit lasting <24 hours with no stroke found on brain imaging) or prior stroke were excluded. In the present analysis, patients with primary intracerebral or subarachnoid hemorrhage were also excluded.

Multiple strategies were used to capture all cases of hospitalized and nonhospitalized stroke occurring in northern Manhattan. All strategies were approved by the relevant institutional review boards and are detailed elsewhere. In brief, the Columbia University Medical Center and 14 other area hospitals were monitored continuously for any northern Manhattan residents with possible stroke through screening of patient admissions, discharge codes, and referrals for neuroimaging. Cases were also obtained from regular contact with community physicians in the area, the Visiting Nurses Service of New York City, and a community outreach program that encouraged self-referral of patients who had not been otherwise identified. Random-digit dialing of 14,810 households performed to recruit subjects for another study was used to identify possible missing cases. Randomly dialed individuals who responded affirmatively when questioned about a prior history of stroke were contacted, and a medical record review and an in-person interview were performed when appropriate.

Of 3,617 possible cases screened, 2,892 were ineligible, including 304 with prior stroke, 2,294 who had suffered a TIA or other neurological event that was not an ischemic stroke, 61 who lived outside northern Manhattan, 10 who were <20 years of age, 11 with insufficient information, and 212 who had been enrolled previously. Only 11 subjects were not white, black, or Hispanic; hence, they were also excluded from the present analysis.

Most cases (82%) were identified within the Columbia University Medical Center, 13% from outside hospitals, 3% from the visiting nurse services, and 2% from community physicians, random-digit dialing, and community outreach programs. Random-digit dialing of 14,810 households identified only 3 cases of first stroke that had occurred in the study period that had otherwise not been identified.

**Assessment of Risk Factors and Race-Ethnicity**

Sociodemographic and risk factor data were obtained by chart review, accompanied by in-person interview of the subject or family. In 71% of cases. During the in-person interview, questions adapted from the Behavioral Risk Factor Surveillance System by the Centers for Disease Control and Prevention were used to determine any prestroke history of hypertension, diabetes, hypercholesterolemia, TIA, current smoking, heavy alcohol use, atrial fibrillation, and myocardial infarct as previously described. During chart review, history of these conditions as reported by the patient or previously diagnosed in the medical record was recorded. As in the US census, race and ethnicity were determined by self-identification. Participants who responded affirmatively the question “Are you of Hispanic/Spanish origin?” were deemed Hispanic. All other participants were then asked to classify their race into 1 of 6 categories: white, black, American Indian, Eskimo, Asian/Pacific Islander, or other. A proxy family member or caregiver was used for patients who were unable to answer questions because of coma, aphasia, etc.

**Ischemic Stroke Subtype Classification**

The participants' history, neurological examination, and diagnostic evaluation (CT, MRI, MR angiography, carotid Doppler, transcranial Doppler, ECG, transesophageal or transthoracic echocardiogram, Holter monitoring, and conventional catheter angiography) were abstracted from the medical record. A panel of NOMAS neurologists blinded to patient identifiers, gender, race-ethnicity, risk factors (except history of TIA, atrial fibrillation, and any other heart condition), and outcome classified each case according to a modified NINDS scheme, summarized below. Consensus was reached by discussion, and disagreements were adjudicated by the principal investigator (R.L.S.).

**Lacunar Infarct**

Lacunar infarcts were defined by focal neurological symptoms and signs with brain image evidence of a small, deep infarct (<1.5 cm in diameter) or a normal repeated brain image, normal or minimal large-artery stenosis by noninvasive vascular imaging or angiography, and no source of cardiac embolism. Ipsilateral cerebral arteries had to be free of hemodynamically significant lesions on angiography if obtained. A lacunar syndrome such as pure motor hemiplegia, pure sensory syndrome, sensorimotor syndrome, ataxic-hemiparesis or dysarthria–clumsy hand syndrome was the presentation in 90%.

**Extracranial Atherosclerotic Infarct**

Focal neurological symptoms and signs with brain image evidence of infarction and extracranial carotid or vertebral stenosis (>60%) or occlusion documented by noninvasive vascular imaging or angiography defined extracranial atherosclerotic infarcts.

**Intracranial Atherosclerotic Infarct**

Focal neurological symptoms and signs with brain image evidence of infarction (including small, deep infarcts) and intracranial large-artery stenosis or occlusion documented by noninvasive vascular imaging or angiography identified intracranial atherosclerotic infarcts.

**Cardioembolic Infarct**

This type of infarct was determined from focal neurological symptoms and signs with brain image evidence of infarction (including small, deep infarcts) and a definite cardiac source of embolism without evidence of large-artery disease by noninvasive vascular testing. Cardiac sources included atrial fibrillation or flutter, recent (<6 months) myocardial infarct, valvular heart disease, cardiac intraluminal thrombus, cardiomyopathy, bacterial or marantic endocarditis, atrial myxoma, and pulmonary vein thrombosis.

**Cryptogenic Infarction/Conflicting Mechanisms**

If there were focal neurological symptoms and signs, no definite cardioembolic source, and no ipsilateral atherosclerotic vascular disease, strokes were assigned this classification. Presentations were usually with a nonlacunar syndrome and an unexplained infarct that was not small or deep. They did not meet any of the above criteria or may have had inadequate evaluation so that reasonable diagnostic classification was difficult. Large (>1.5 cm), deep infarcts presenting with a lacunar syndrome with neither a cardiac source of embolus nor large-artery atheroma were classified as cryptogenic. Small, deep infarcts presenting with a lacunar syndrome and no obvious embolic or arterial cause were classified as cryptogenic if the infarct location or arterial involvement was not typical for lacunes (eg, anterior choroidal artery territory, midbrain), if there was incidental radiological evidence of previous asymptomatic nonlacunar infarct in the same vascular territory as the index lesion, or if a potential source of embolism was possible although not definite (eg, spontaneous echo contrast, multiple filamentous strands).

**Other**

This category included rare but known causes of stroke such as dissection, fibromuscular dysplasia, vasculitis, sickle cell anemia, and stroke in the setting of migraine.

**Statistical Analysis**

SAS version 8.02 (SAS Institute) was used for all statistical analyses. Average annual incidence rates stratified by age, gender, and ethnicity were calculated as follows: number of first stroke patients identified divided by the white/black/Hispanic northern Manhattan population according to 1990 American census. Direct age-adjusted rates with standard errors were calculated using age-specific proportions from the northern Manhattan population for each race-ethnicity subgroup. Relative incidence rates for each ischemic stroke subtype were obtained by division of the rate in the Hispanic or black population by the white reference group. The delta method was used to obtain 95% CIs. We used χ² and Student t tests to compare...
race-ethnic differences in risk factors, sociodemographics, and proportions of ischemic subtypes.

Results
Over 4 years, 714 individuals with first ischemic stroke were identified within the northern Manhattan population; 29% were white, 22% were black, and 49% were Hispanic (55% from the Dominican Republic, 13% from Puerto Rico, 11% from Cuba, 21% other/unknown). The sociodemographic characteristics and risk factor profiles of this group are shown in Table 1. Of these subjects, 95% were hospitalized. All characteristics and risk factor profiles of this group are shown in Table 1. Of these subjects, 95% were hospitalized. All characteristics and risk factor profiles of this group are shown in Table 1.

For statistical comparisons of risk factors between race-ethnic groups, Student t test and x² test were used as appropriate.

*Baseline data are missing in >10%. For high school education, n = 543; for TIA, n = 526.
†Significantly different from the white population at P<0.0001.
‡Significantly different from the white population at P<0.01.
§Significantly different from the white population at P<0.05.

The annual age-adjusted incidence of first ischemic stroke per 100 000 was 88 (95% CI, 75 to 101) in whites, 149 (95% CI, 132 to 165) in Hispanics, and 191 (95% CI, 160 to 221) in blacks. The age-adjusted annual incidence rate of each ischemic stroke subtype was lowest in whites, higher in

<table>
<thead>
<tr>
<th>Subtype-Specific Age-Adjusted Annual Ischemic Stroke Incidence Rates per 100 000 Among Whites, Blacks, and Hispanics in Northern Manhattan 1993–1997</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whites (95% CI)</td>
</tr>
<tr>
<td>(n=207)</td>
</tr>
<tr>
<td>----------------</td>
</tr>
<tr>
<td>Atherosclerotic</td>
</tr>
<tr>
<td>Intracranial</td>
</tr>
<tr>
<td>Extracranial</td>
</tr>
<tr>
<td>Lacunar</td>
</tr>
<tr>
<td>Cardioembolic</td>
</tr>
<tr>
<td>Cryptogenic</td>
</tr>
<tr>
<td>Other</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>
TABLE 3. Relative Age-Adjusted Rates With 95% CIs of Each Ischemic Stroke Subtype in Hispanics and Blacks Compared With Whites

<table>
<thead>
<tr>
<th>Subtype</th>
<th>Hispanic vs White (95% CI)</th>
<th>Black vs White (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n=352)</td>
<td>(n=155)</td>
</tr>
<tr>
<td>Atherosclerotic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intracranial</td>
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<td>5.85 (1.82–18.73)</td>
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<td>1.58 (0.99–2.52)</td>
</tr>
<tr>
<td>Cryptogenic</td>
<td>1.44 (1.09–1.92)</td>
<td>1.82 (1.31–2.52)</td>
</tr>
<tr>
<td>Other</td>
<td>2.27 (0.42–51.91)</td>
<td>4.66 (0.25–20.50)</td>
</tr>
</tbody>
</table>

**Discussion**

To the best of our knowledge, this is the first population-based study to determine and compare the incidence of ischemic stroke subtypes among whites, blacks, and Hispanics living in one community. Our findings suggest that the high ischemic stroke incidence in blacks and Hispanics compared with whites is due to increased incidence rates of all ischemic stroke subtypes. Differences in incidence, however, were not equal across all subtypes. The greatest race-ethnic disparities in incidence were observed for intracranial atherosclerotic and lacunar stroke. Extracranial atherosclerotic stroke incidence was greater in blacks than whites, but the differences were less pronounced for Hispanics. The smallest race-ethnic disparities in incidence were seen for cardioembolic and cryptogenic stroke.

Prior studies that examined cerebrovascular disease location and the relative proportions of ischemic stroke subtypes among races included non–population-based angiogram, carotid Doppler, autopsy, and hospital patient series. Despite methodological limitations, these studies have shown a higher proportion of lacunar and intracranial atherosclerotic stroke in blacks and Hispanics and a higher proportion of cardioembolic and extracranial atherosclerotic stroke in whites. In contrast to some prior studies, we found no significant difference in the proportion of lacunar or extracranial atherosclerotic stroke among the 3 race-ethnic groups, although the proportion of lacunar stroke was somewhat lower in whites compared with blacks and Hispanics. Furthermore, our study, which was population based, demonstrates a greater incidence of extracranial atherosclerotic stroke in blacks than suggested by these previous, more selected series and may refute the argument that the low rates of carotid endarterectomy in blacks are caused by a low incidence of extracranial carotid disease. Inconsistencies among studies comparing race-ethnic ischemic subtype proportions may stem from different risk factor profiles among participants in each study and from selection bias when studies are not population based.

There are few population-based studies of ischemic stroke subtype incidence; fewer still have examined race ethnicity, none have included Hispanics, and some have included only white subjects. The Greater Cincinnati and Northern Kentucky Stroke Study (GCNKSS) found a significantly greater proportion of cardioembolic stroke in whites than in blacks and no other significant race-ethnic differences in ischemic stroke subtype proportions. The GCNKSS also found that blacks had significantly higher incidence rates of all ischemic stroke subtypes compared with whites and that the elevations in relative rates were largest for small-vessel and cryptogenic stroke, smaller for large-vessel stroke, and smallest for cardioembolic stroke. The incidence rates obtained from the northern Manhattan black population were very comparable to figures from the GCNKSS. In another GCNKSS study, the incidence rates of cardioembolic, small-vessel, and uncertain-cause stroke were significantly higher in blacks, and the incidence of large-vessel stroke was higher in whites.

With Whites

### FIGURE

Proportion of ischemic stroke subtypes among whites, blacks, and Hispanics in northern Manhattan.
ated with previous cardiac disease and atrial fibrillation, and northern Manhattan whites had a significantly greater proportion of cardioembolic stroke than blacks and Hispanics. Furthermore, race-ethnic differences in the proportions of ischemic stroke subtypes were attenuated after adjustment for sociodemographics and risk factors.

The strengths of our study include the use of many overlapping capture methods for complete case ascertainment. The study also includes Hispanics, who have not been represented in any previous population-based studies of ischemic stroke subtype incidence. Our population-based surveillance system included hospitalized and nonhospitalized cases, although the 5% proportion of nonhospitalized strokes may be smaller than that of other communities. Finally, study subjects were thoroughly investigated, and stroke subtype assignment was performed by a team of neurologists who reached a consensus agreement, therefore minimizing classification errors.

Limitations of our study include inadequate information on the risk factor profile of the underlying northern Manhattan population to allow for adjustment of relative rates for risk factors and the fairly small numbers of individuals in each race-ethnic ischemic stroke subtype subgroup, which led to decreased CI precision. This was an observational study, which limits the inferences that can be made about etiological relationships. In addition, although great measures were taken to include all incident stroke cases, in any incidence study, complete capture of all cases cannot be substantiated. The fact that not all subjects underwent a systematic investigation could be a source of information bias; however, diagnostic investigations were undertaken on the basis of the subject’s syndrome, rather than race, as evidenced by the similar proportions of inadequate workup by race-ethnicity. Moreover, the diagnostic committee was blinded to race-ethnicity. Another source of bias may be the use of census figures from 1990 to calculate incidence rates, but the use of projected figures for 1995 (the study midpoint) could have introduced even greater bias into the study. Whether our study provides accurate data for minority populations, in any previous population-based studies of ischemic stroke subtype incidence. Our population-based surveillance system included hospitalized and nonhospitalized cases, although the 5% proportion of nonhospitalized strokes may be smaller than that of other communities. Finally, study subjects were thoroughly investigated, and stroke subtype assignment was performed by a team of neurologists who reached a consensus agreement, therefore minimizing classification errors.

Acknowledgments

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