Stroke Incidence and Survival Among Middle-Aged Adults
9-Year Follow-Up of the Atherosclerosis Risk in Communities (ARIC) Cohort

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Background and Purpose—Although stroke mortality rates in the United States are well documented, assessment of incidence rates and case fatality are less well studied.

Methods—A cohort of 15 792 men and women aged 45 to 64 years from a population sample of households in 4 US communities was followed from 1987 to 1995, an average of 7.2 years. Incident strokes were identified through annual phone contacts and hospital record searching and were then validated.

Results—Of the 267 incident definite or probable strokes, 83% (n = 221) were categorized as ischemic strokes, 10% (n = 27) were intracerebral hemorrhages, and 7% (n = 19) were subarachnoid hemorrhages. The age-adjusted incidence rate (per 1000 person-years) of total strokes was highest among black men (4.44), followed by black women (3.10), white men (1.78), and white women (1.24). The black versus white age-adjusted rate ratio (RR) for ischemic stroke was 2.41 (95% CI, 1.85 to 3.15), which was attenuated to 1.38 (95% CI, 1.01 to 1.89) after adjustment for baseline hypertension, diabetes, education level, smoking status, and prevalent coronary heart disease. There was a tendency for the adjusted case fatality rates to be higher among blacks and men, although none of the case fatality comparisons across sex or race was statistically significant.

Conclusions—After accounting for established baseline risk factors, blacks still had a 38% greater risk of incident ischemic stroke compared with whites. Identification of new individual and community-level risk factors accounting for the elevated incidence of stroke requires further investigation and incorporation into intervention planning. (Stroke. 1999;30:736-743.)

Key Words: cerebral infarction ■ epidemiology ■ intracerebral hemorrhage ■ racial differences

An estimated 600 000 to 731 100 strokes occurred in 1996 in the United States, making stroke the third leading cause of death and the leading cause of severe neurological disability. Although the rate of death from stroke declined 17.3% from 1985 to 1995, the aging of the US population contributed to a 3.2% increase in actual number of stroke deaths.6

Blacks are at particularly high risk for fatal stroke, with age-adjusted stroke mortality rates 2 to 4 times that of whites.1,4–8 In addition, a geographic concentration of stroke mortality exists with especially high rates in North Carolina, South Carolina, and Georgia.6,9–11 The relative contributions of stroke incidence versus survival to the greater stroke mortality rates among blacks continue to be poorly understood. While incidence rates and case fatality of stroke have been reported from numerous countries worldwide,12–17 incidence data on fatal and nonfatal stroke among blacks are limited.2,4,18–23 Reasons for reported race differences are not clear.24 In fact, a recent American Heart Association Scientific Panel concluded that “stroke incidence data on groups at high risk of stroke mortality (blacks, residents of the southeastern United States) are lacking.”7 To further the understanding of stroke incidence and case fatality, we describe a 9-year follow-up of a biracial cohort from 4 geographically diverse US communities (including 2 in the southeastern United States).

Subjects and Methods

The Atherosclerosis Risk in Communities (ARIC) Study recruited a population-based cohort of 15 792 individuals aged 45 to 64 years at baseline from a sample of households in 1987 through 1989.25 Samples were selected from Jackson, Mississippi; Forsyth County, North Carolina; Washington County, Maryland; and the northwestern suburbs of Minneapolis, Minnesota. The Jackson sample was from black residents only.

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Hypertension at baseline was defined as a systolic blood pressure of \( \geq 140 \) mm Hg or a diastolic pressure of \( \geq 90 \) mm Hg or use of antihypertensive medication. Prevalent diabetes mellitus was defined as a fasting glucose level of \( \geq 140 \) mg/dL, nonfasting glucose level of \( \geq 200 \) mg/dL, and/or a history of or treatment for diabetes. Self-reported educational achievement, a surrogate measure of socioeconomic status, was categorized into 3 levels based on years of school completed: less than high school (<12 years), high school graduate (12 years), and greater than high school (>12 years). The study was approved by an institutional review committee and subjects gave informed consent.

**Ascertainment of Clinical Stroke Events**

ARIC Study participants were contacted annually by phone, and all hospitalizations and deaths during the previous year were identified. In addition, local hospitals provided lists of cardiovascular disease discharges, which were examined for participant hospitalizations. A hospitalization was considered eligible for possible validation as a stroke if it contained a discharge diagnosis code indicative of cerebrovascular disease (International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM) codes 430 to 438) and/or 1 of the following keywords was listed in the discharge summary or mentioned in the nursing notes as occurring during the admission: stroke, transient ischemic attack, cerebrovascular disease, cerebral hemorrhage, cerebral infarction, subarachnoid hemorrhage (SAH), cerebral embolus, paralysis, aphasia, lacunar infarction, dysarthria, cerebral angiofibrinolytic therapy, and endarterectomy. A case was also eligible if review of the medical record revealed a diagnostic CT or MRI scan with cerebrovascular findings or if the patient had ever been admitted to the neurological intensive care unit. Out-of-hospital fatal strokes (n=4) were also monitored but not validated and therefore are not included in the calculation of incidence rates.

**Diagnostic Methods**

appropriate sections of the medical record of eligible hospitalizations were copied and sent to a central center (Minneapolis) for abstraction by a single trained nurse. Items recorded from the medical charts included timing, type, and duration of neurological symptoms and deficits (headache, vertigo, convulsions, aphasia, paralysis), medical history, the results of the procedures performed (catheterization, heart surgery, cerebral angiography, carotid endarterectomy, CT or MRI scan, B-mode or Doppler ultrasound, lumbar puncture), anticoagulant or thrombolytic therapy, and autopsy evidence.

Using criteria adopted from the National Survey of Stroke, strokes were classified by computer algorithm and categorized into 1 of 4 main types: SAH, intracerebral hemorrhage (ICH), thrombotic brain infarction, or embolic brain infarction. Categories of “possible stroke of undetermined type,” “out-of-hospital fatal stroke” (based on underlying cause of death from the death certificate only), and “no stroke” were also assigned. In the rare instance when a stroke case met criteria for 2 different diagnostic categories, the following hierarchy was used: ICH, SAH, embolic brain infarction, thrombotic brain infarction.

In addition to a computer-determined diagnosis, cases were independently reviewed by a physician. The physician-reviewer was provided with a detailed report of the information abstracted from the medical record as well as the full discharge summary, the CT and MRI scan reports, reports from any neurological consults, and admission history summary. The final diagnosis was determined by agreement of computer and reviewer classification. Disagreements were adjudicated by a second physician-reviewer. Although the physician-reviewers used the algorithm described below, they employed their own discretion whenever the algorithm classification clearly failed to match the clinical picture.

**Event Criteria**

The minimum criterion for definite or probable stroke was evidence of sudden or rapid onset of neurological symptoms lasting for >24 hours or leading to death, in the absence of evidence for a nonstroke cause. Exclusionary conditions for a diagnostic classification of stroke included major brain trauma, neoplasms, coma due to metabolic disorders or disorders of fluid or electrolyte balance, vasculitis involving the brain, peripheral neuropathy, hematologic abnormalities, or central nervous system infections. Presence of major symptoms (hemiparesis of 2 or more body parts, homonymous hemianopia, or aphasia) or minor symptoms (diplopia, vertigo or gait disturbance, dysarthria, dysphagia, dysphonia, or unilateral numbness or weakness of \( \geq 2 \) body parts) were also considered in event classification. Vertigo and gait disturbance occurring together were considered 1 minor symptom. The following additional criteria were used to subtype definite or probable strokes.

**Thrombotic Brain Infarction**

Definite thrombotic brain infarction (TBI) required either (1) autopsy evidence of a nonhemorrhagic infarct of the brain or (2) evidence from the hospital record of 1 major or 2 minor neurological signs or symptoms lasting at least 24 hours or until the patient died, and CT or MRI findings showing an infarct or area of decreased density (excluding evidence of hemorrhage). A case was considered a probable TBI if 1 major or 2 minor symptoms had sudden onset lasting >24 hours; and CT or MRI findings within the first 48 hours were negative or nonspecific, with no sign of hemorrhage; and a spinal tap was not done, was traumatic, or yielded clear, colorless spinal fluid.

**Cardioembolic Stroke**

Definite cardioembolic stroke (CES) required either (1) autopsy evidence of an infarcted area in the brain and a source of emboli in a vessel of any organ or presence of an embolus in the brain, or (2) 1 major or 2 minor symptoms showed rapid onset lasting >24 hours and the medical records revealed evidence of valvular heart disease, atrial fibrillation, or flutter, acute or recent (within 4 weeks) myocardial infarction, cardiac or arterial procedure intracardiac thrombus, or bacterial endocarditis. In this latter case, CT or MRI findings must have shown an area of decreased density indicative of edema or ischemia, with no evidence of hemorrhage. A diagnosis of probable CES was made if there was evidence of 1 major or 2 minor symptoms and medical record review identified a source of cerebral embolus; and if a CT or MRI within the first 48 hours was either negative or nonspecific, with no evidence of hemorrhage; and a spinal tap was either not done, was traumatic, or yielded clear, colorless spinal fluid.

**Subarachnoid Hemorrhage**

Definite SAH required either (1) angiographic identification of a saccular aneurysm as a source of bleeding and bloody or xanthochromic spinal fluid; or (2) CT or MRI findings indicating a blood clot in the fissure of Sylvius, between the frontal lobes, in the basal cisterns, or within a ventricle, with no associated intraparenchymal hematoma; or (3) autopsy or surgical procedures that uncovered a bleeding saccular aneurysm. A diagnosis of probable SAH was made if (1) angiographic evidence of a saccular aneurysm was identified as the source of bleeding and the spinal tap was not done, was traumatic, or was missing; or (2) within a few minutes or hours of symptom onset there was evidence of a severe headache, depressed state of consciousness, meningeal irritation, or retinal hemorrhages, and spinal fluid was bloody or xanthochromic.

**Intracerebral Hemorrhage**

Definite ICH required (1) an area of increased density indicative of ICH identified by CT or MRI; or (2) the demonstration of an ICH at autopsy or during surgery; or (3) in the absence of a technically adequate CT or MRI, there was 1 major or 2 minor symptoms of sudden onset lasting >24 hours, bloody (nontraumatic) or xanthochromic spinal fluid, and evidence from cerebral angiography of a vascular mass without evidence of aneurysm or arteriovenous malformation. A diagnosis of probable ICH was made if (1) a decreased level of consciousness or coma lasted at least 24 hours and
(2) a nontraumatic spinal tap was spinal fluid was bloody or xanthochromic and (3) CT or MRI imaging was not performed or was inadequate.

**Lacunar Infarcts**

All definite thrombotic brain infarctions were further classified as either lacunar or nonlacunar. Lesion size and neuroimaging reports were evaluated, and a final diagnosis of lacunar infarction was made on the basis of physician review. A definite lacunar infarction was assigned if 2 criteria were met: (1) anatomic findings typical of lacunar infarctions (basal ganglia, brain stem, thalamus, internal capsule, or cerebral white matter) and (2) estimated infarct size of ≤2 cm or an infarct of unstated size. In the rare event that these criteria were met but the neuroimaging report explicitly stated that the infarct was not lacunar, the latter prevailed. The definition of lacunar infarcts was primarily based on characteristic neuroimaging finding and may include some false-positives. Lacunar symptoms were not part of the diagnostic criteria because the recording of physical examination findings in the medical record were incomplete and inconsistent. However, they were used at the discretion of the reviewer when other criteria components were not definitive.

**Definition of Incidence**

An incident stroke occurrence was defined as a first definite or probable hospitalized stroke occurring in a participant free of a history of physician-diagnosed stroke at the baseline interview. Participants were excluded from follow-up if they had a history of stroke at baseline (n=284) or if history of stroke was unknown (n=45). Therefore, a total of 15,463 participants were followed for incident stroke occurrence through 1995, an average of 7.2 years of follow-up.

For this report, ischemic strokes were defined as either cardioembolic or thrombotic brain infarctions. Cases classified as possible stroke of undetermined type (n=17) and out-of-hospital fatal stroke (n=4) were excluded from incident rate analyses. Case fatality was defined as the percent of stroke cases in which the patient died of any cause within 30 days of hospital admission.

**Data Analysis**

For participants with an incident stroke, the follow-up time interval was defined as the time between the baseline examination and the date of stroke admission. For those without strokes, the end of follow-up time was December 31, 1995, date of death, or date of last contact if lost to follow-up. Age-adjusted (using age at event) stroke incidence rates by race, gender, and education status were computed from Poisson regression models. Because of the small number of blacks in the Minneapolis and Washington County cohorts, they were excluded when fitting these regression models. The 30-day stroke survival proportions were adjusted for age at event, race, and gender by logistic regression. Deaths were discovered via annual phone calls or through a search of health department death certificate files.

**Results**

**Validation of Hospitalized Events**

Between 1987 and 1995, 11,85 stroke-eligible hospitalizations were identified. Fifty-four percent (n=647) of medical records showed no evidence of any neurological signs lasting >24 hours or development of new signs or symptoms leading up to or during the hospitalization, and these were automatically classified as no stroke. This percentage remained stable over the study period. Of the remaining 538 hospitalizations, 84% had at least 1 CT scan, 27% had an MRI of the head, 15% had a cerebral angiography, and 6% a lumbar puncture, with no differences in these percentages between blacks and whites. Of these 538 hospitalizations, 61% (n=329) were either definite or probable strokes. The computer classification algorithm and the physician-reviewer agreed on the stroke classification in 78% of cases (κ=0.71). In 65% of the 158 adjudicated cases, the adjudicator agreed with the initial physician reviewer. In the majority (64%) of the 118 disagreements, the computer algorithm tended toward a less-severe diagnostic classification.

Among the 526 hospital discharge codes indicative of acute stroke (ICD9-CM 430 to 434), 44% were validated with a final diagnosis of definite or probable stroke by the ARIC classification system (Table 1). Cases discharged with a ICD-9-CM code of 430 (SAH) had the highest validation rate (86%). Of the 277 cases classified as ischemic strokes, the majority (78%) were discharged with an ICD-9-CM code of

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<table>
<thead>
<tr>
<th>ICD-9-CM Code</th>
<th>ICD-9-CM Classification</th>
<th>ISC</th>
<th>ICH</th>
<th>SAH</th>
<th>No Stroke*</th>
<th>Total</th>
<th>Percent Validated†</th>
</tr>
</thead>
<tbody>
<tr>
<td>430</td>
<td>Subarachnoid hemorrhage</td>
<td>0</td>
<td>0</td>
<td>19</td>
<td>3</td>
<td>22</td>
<td>86%</td>
</tr>
<tr>
<td>431</td>
<td>Intracerebral hemorrhage</td>
<td>7</td>
<td>26</td>
<td>1</td>
<td>7</td>
<td>41</td>
<td>83%</td>
</tr>
<tr>
<td>432</td>
<td>Other intracerebral hemorrhage</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>10</td>
<td>11</td>
<td>9%</td>
</tr>
<tr>
<td>433</td>
<td>Occlusion of precerebral arteries</td>
<td>36</td>
<td>0</td>
<td>0</td>
<td>230</td>
<td>266</td>
<td>14%</td>
</tr>
<tr>
<td>434</td>
<td>Occlusion of cerebral arteries</td>
<td>143</td>
<td>1</td>
<td>0</td>
<td>42</td>
<td>186</td>
<td>77%</td>
</tr>
<tr>
<td>435</td>
<td>Transient cerebral ischemia</td>
<td>14</td>
<td>0</td>
<td>0</td>
<td>105</td>
<td>119</td>
<td>12%</td>
</tr>
<tr>
<td>436</td>
<td>Acute but ill-defined cerebrovascular disease</td>
<td>73</td>
<td>3</td>
<td>0</td>
<td>32</td>
<td>108</td>
<td>70%</td>
</tr>
<tr>
<td>437</td>
<td>Other ill-defined cerebrovascular disease</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>58</td>
<td>59</td>
<td>2%</td>
</tr>
<tr>
<td>438</td>
<td>Late effects of cerebrovascular disease</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>245</td>
<td>246</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Other</td>
<td>None of above</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>124</td>
<td>127</td>
<td>2%</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>277</td>
<td>32</td>
<td>20</td>
<td>856</td>
<td>1185</td>
<td>28%</td>
</tr>
</tbody>
</table>

ISC indicates ischemic stroke.

*Includes cases classified as no or possible stroke.

†Percent of cases for each ICD-9-CM code validated as definite or probable stroke.
either 434 (occlusion of cerebral arteries) or 436 (acute but ill-defined cerebrovascular disease). We also classified as definite or probable ischemic strokes 14% (n=36) of cases discharged with ICD-9-CM 433 (occlusion and stenosis of precerebral arteries) and 12% (n=14) of cases discharged with ICD-9-CM 435 (transient cerebral ischemia). Of the 127 cases identified by key words in the discharge summary but lacking an ICD-9-CM discharge code of 430 to 438, only 3 (2%) were validated as strokes.

**Table 2. Number of Incident Clinical Strokes and Person-Years of Observation by Type, Race, and Sex: the ARIC Study, 1987–1995**

<table>
<thead>
<tr>
<th>Stroke Type</th>
<th>Men</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Black</td>
<td>White</td>
<td>Black</td>
<td>White</td>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>Ischemic</td>
<td>42</td>
<td>75</td>
<td>53</td>
<td>51</td>
<td>221</td>
<td></td>
</tr>
<tr>
<td>ICH</td>
<td>9</td>
<td>4</td>
<td>9</td>
<td>5</td>
<td>27</td>
<td></td>
</tr>
<tr>
<td>SAH</td>
<td>1</td>
<td>5</td>
<td>7</td>
<td>6</td>
<td>19</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>52</td>
<td>84</td>
<td>69</td>
<td>62</td>
<td>267</td>
<td></td>
</tr>
<tr>
<td>Person-years of observation*</td>
<td>10,411</td>
<td>37,939</td>
<td>17,724</td>
<td>43,256</td>
<td>109,330</td>
<td></td>
</tr>
</tbody>
</table>

*Person-years of follow-up for entire cohort.

### Age-Adjusted Stroke Incidence

Table 2 shows that of the 329 hospitalizations with a validated definite or probable stroke, 267 (81%) were incident events. Two persons who had both a first-time ischemic stroke and a first-time hemorrhagic stroke are included in this table. Of the incident events, 83% (n=221) were ischemic strokes, 10% (n=27) were hemorrhagic strokes, and 7% (n=19) were SAH. Among the 178 definite thrombotic brain infarctions, 38% (n=68) were classified as lacunar. Forty-seven lacunar strokes occurred in blacks and 21 in whites.

As Table 3 shows, the age-adjusted incidence rate (per 1000 person-years) of total hospitalized stroke was highest among black men (5.32), followed by black women (3.96), white men (2.00), and white women (1.49). The difference in age-adjusted stroke incidence was statistically significant between blacks and whites but not between men and women. Stroke incidence rates were higher among those with less than a high school education and among the all-black cohort from Jackson. These patterns were similar for both ischemic as well as hemorrhagic stroke incidence rates.

The black versus white age-adjusted rate ratio (RR) for ischemic stroke was 2.41 (95% CI, 1.85 to 3.15) (Figure 1). The high incidence among blacks was greater among those aged ≤55 years (RR=2.77; 95% CI, 1.37 to 5.62) compared with those ≥55 (RR=2.23; 95% CI, 1.66 to 3.00).

**Table 3. Age-Adjusted Incidence Rate (per 1000 Person-Years) and 95% CI of Hospitalized Clinical Stroke by Sex, Race, Educational Achievement, and Center: the ARIC Study, 1987–1995**

<table>
<thead>
<tr>
<th>Group</th>
<th>Ischemic Stroke*</th>
<th>Hemorrhagic Stroke†</th>
<th>Total Stroke‡</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rate</td>
<td>95% CI</td>
<td>Rate</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Women</td>
<td>1.80</td>
<td>(1.40–2.32)</td>
<td>0.24</td>
</tr>
<tr>
<td>Men</td>
<td>2.39</td>
<td>(1.86–3.07)</td>
<td>0.27</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>3.57</td>
<td>(2.77–4.61)</td>
<td>0.65</td>
</tr>
<tr>
<td>White</td>
<td>1.48</td>
<td>(1.16–1.90)</td>
<td>0.11</td>
</tr>
<tr>
<td>Race-Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black men</td>
<td>4.44</td>
<td>(3.31–5.97)</td>
<td>0.77</td>
</tr>
<tr>
<td>White men</td>
<td>1.78</td>
<td>(1.35–2.34)</td>
<td>0.12</td>
</tr>
<tr>
<td>Black women</td>
<td>3.10</td>
<td>(2.34–4.10)</td>
<td>0.58</td>
</tr>
<tr>
<td>White women</td>
<td>1.24</td>
<td>(0.93–1.65)</td>
<td>0.09</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than HS</td>
<td>3.69</td>
<td>(2.80–4.86)</td>
<td>0.35</td>
</tr>
<tr>
<td>HS graduate</td>
<td>1.66</td>
<td>(1.22–2.27)</td>
<td>0.24</td>
</tr>
<tr>
<td>Greater than HS</td>
<td>1.65</td>
<td>(1.25–2.18)</td>
<td>0.22</td>
</tr>
<tr>
<td>Center</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Forsyth County, NC</td>
<td>1.41</td>
<td>(0.99–2.01)</td>
<td>0.14</td>
</tr>
<tr>
<td>Jackson, Miss</td>
<td>3.49</td>
<td>(2.67–4.56)</td>
<td>0.69</td>
</tr>
<tr>
<td>Minneapolis, Minn</td>
<td>1.56</td>
<td>(1.11–2.19)</td>
<td>0.10</td>
</tr>
<tr>
<td>Washington County, Md</td>
<td>1.78</td>
<td>(1.29–2.46)</td>
<td>0.10</td>
</tr>
</tbody>
</table>

*Age at event was used for age adjustment. HS indicates high school.

*Includes definite and probable embolic and thrombotic brain infarction.

†Includes definite and probable ICH (excludes SAH).

‡Total stroke combines ischemic, ICH, and SAH.
After additional adjustment for prevalent hypertension and diabetes mellitus, the excess in ischemic stroke incidence in blacks was attenuated (RR=1.57; 95% CI, 1.18 to 2.09) but was still statistically significant. The addition of educational status to the model decreased the black excess to 38%, which remained statistically significant (95% CI, 1.01 to 1.89). Further adjustment for baseline smoking status and prevalent coronary heart disease had little or no effect on the rate ratio. A similar pattern was seen with hemorrhagic stroke (data not shown).

When all stroke types are combined (Figure 2), the age-adjusted black-to-white incidence rate ratio was 2.58 (95% CI, 2.02 to 3.29). After adjusting for gender, hypertension, diabetes, center, education, smoking, and history of coronary heart disease, the relative risk among blacks versus whites was attenuated to 1.62, which also remained statistically significant (95% CI, 1.22 to 2.14).

**Survival After Stroke**

The unadjusted 30-day case fatality rate for incident and recurrent hospitalized stroke combined (n=327) was 7.6% for ischemic and 37.5% for hemorrhagic events (data not shown). The age-, sex-, and gender-adjusted case fatality rates shown in Table 4 were similar (7.3% and 33.1%, respectively). There was a trend for the adjusted case fatality rates from ischemic stroke to be higher among blacks and men (except total stroke), although none of the comparisons across sex or race was statistically significant. Hemorrhagic strokes were 4.5 times as fatal as ischemic strokes in the overall study group.

**Discussion**

We found that blacks in the ARIC Study had incidence rates of ischemic stroke 2- to 3-fold higher than those of whites. However, the differences in 30-day case fatality rates differed by only 44%, and this difference was not statistically significant. These data support the conclusion that the well-documented excess in mortality among blacks is primarily due to elevated incidence rather than diminished survival.

This study adds to the growing literature on race/ethnicity-specific stroke incidence rates in 3 important ways. It confirms findings from other studies of an elevated incidence rate...
of stroke among blacks, provides a partial explanation for the observed excess, and contributes new information about the association of ethnicity with survival after a stroke.

Previous studies have reported that blacks have a higher risk of stroke than whites. Two of the most recent studies to report stroke incidence rates in blacks compared with whites come from northern Manhattan and from Cincinnati. In the Northern Manhattan Stroke Study, the average annual age-adjusted incidence rate for stroke was 223 per 100,000 population for blacks and 93 per 100,000 for whites, a 2.4-fold greater stroke incidence among blacks. The black-to-white age-adjusted incidence rate ratio was greatest in younger ages. Among 45- to 54-year-olds, the RR was 3.8 compared with 1.7 among those 55 to 64 years of age. In ARIC, a similar pattern was seen, although the magnitude of the age-dependent difference in the incidence rate ratio was smaller (2.8 and 2.2, respectively), a difference that did not achieve statistical significance between age strata. We also found that even though the number of events was small, the difference in age-adjusted incidence rates of hemorrhagic stroke among blacks (0.65 per 1000 person-years) and whites (0.11 per 1000 person-years) was large and statistically significant. This increased risk of hemorrhagic stroke among blacks in the ARIC study also agrees with findings from the Cincinnati. In the Northern Manhattan Stroke Study, the average annual age-adjusted incidence rate for stroke was 223 per 100,000 population for blacks and 93 per 100,000 for whites, a 2.4-fold greater stroke incidence among blacks. The black-to-white age-adjusted incidence rate ratio was greatest in younger ages. Among 45- to 54-year-olds, the RR was 3.8 compared with 1.7 among those 55 to 64 years of age. In ARIC, a similar pattern was seen, although the magnitude of the age-dependent difference in the incidence rate ratio was smaller (2.8 and 2.2, respectively), a difference that did not achieve statistical significance between age strata. We also found that even though the number of events was small, the difference in age-adjusted incidence rates of hemorrhagic stroke among blacks (0.65 per 1000 person-years) and whites (0.11 per 1000 person-years) was large and statistically significant. This increased risk of hemorrhagic stroke among blacks in the ARIC study also agrees with findings from the Northern Manhattan study, in which black women had >3 times the rate of incident intracerebral hemorrhage as white women, and is consistent with other previous reports.

Blacks in the Cincinnati region had an incidence rate of stroke greater than that of the white population in Rochester for all age groups except ≥75 years. Among those aged <65, blacks in the Cincinnati region had ischemic stroke incidence rates 2 to 4 times greater than those of similarly aged whites in Rochester. The age-and-sex-adjusted incidence rate of first-ever stroke among blacks in the Cincinnati region in 1993 was found to be 288 per 100,000 (95% CI, 250 to 325) compared with 179 per 100,000 (95% CI, 164 to 194) in Rochester whites during 1985 to 1989. Of the black population in the Cincinnati region, the incidence rate of SAH remained stable from 1988 to 1993, while the incidence rate of ICH increased significantly during this same period. However, Broderick et al did not report comparisons of hemorrhagic strokes between blacks in Cincinnati and whites in Rochester.

In addition to the previously mentioned studies, we found our data consistent with national data from the National Health and Nutrition Examination Survey (NHANES) Epidemiologic Follow-up, which indicates that when compared with whites of the same age, younger blacks had 2.6 times the risk of cerebral infarction as whites. However, older blacks (>64 years) were not at increased risk.

Although further understanding of the reasons for the observed excess in stroke risk among blacks is needed, this study provides a partial explanation for the observed excess. While we found that differences in smoking history and prevalent heart disease explained only part of the race/ethnicity stroke difference, we found that much of the excess stroke risk in blacks was attributable to differences in hypertension and diabetes, with a further proportion attributable to differences in educational achievement. This finding contrasts with reports based on the NHANES follow-up study which showed that a black-to-white RR among 34- to 44-year-olds attenuated only from 2.6 to 2.1 after accounting for sex, education, history of heart disease, diabetes, systolic blood pressure, treatment for hypertension, Quetelet index, and serum hemoglobin and magnesium levels. However, the NHANES study did not validate stroke events, and contrary to the ARIC study, it did not report a black excess in stroke incidence among those older than 64 years. An attenuation of the race/ethnicity difference in stroke incidence after accounting for hypertension and diabetes is consistent with what has also been shown for stroke mortality rates.

The independent contribution of educational status in explaining differences in stroke incidence agrees with recent investigations into the role of social class as a contributing factor to the stroke mortality belt in the United States. Third, although several studies have reported greater survival rates after stroke for men than women (which we did not observe), less is known about differences in survival between ethnic groups. In New Zealand, the age- and sex-adjusted risk of death within 28 days was higher in Maori
(OR, 1.4) and Pacific Islands people (OR, 1.6) than in Europeans, the latter being statistically significant. A review by Gillum et al. of reports from hospital mortality studies suggests that although firm conclusions about ethnic/race differences in survival after a stroke cannot be drawn due to the sparse number of studies, blacks suffering from stroke may have a less favorable acute prognosis than whites. We found that while case fatality after either ischemic or ICH was 44% greater among blacks than whites (12.5% and 8.7%, respectively), the difference was not statistically significant with our sample size. This may partly be due to greater proportion of hemorrhagic strokes in blacks. Continued follow-up of this cohort is needed to determine whether these differences in survival are statistically meaningful.

Although the validation system of stroke events in ARIC is based in part on diagnostic procedures such as CT and MRI scans, this dependence was unlikely to have accounted for the observed differences in event rates between blacks and whites. The use of CT and MRI scans was high among those with stroke discharge codes and was similar between groups and to that in other studies. In the Greater Cincinnati/Northern Kentucky study, 98% of incident cases had a CT scan and 22% had an MRI of the brain.2 A strength of this study is the use of validated events rather than reliance on discharge diagnosis coding, which has been shown to be inaccurate in diagnosing ischemic cerebrovascular disease in patients in this study and others.33,34

Although our classification system is not error free, it was designed to achieve consistency across study centers and over time. Our case definition of stroke was primarily clinical, founded on sufficiently unambiguous neurological findings in the absence of another apparent cause. Neuroimaging reports were used to classify stroke cases into subtypes. This method of case classification was checked by an independent physician’s review of the discharge summary, neuroimaging reports, and other relevant material. Although the reviewer followed strict classification rules, clinical judgment was used whenever these rules clearly failed to match the clinical picture. In cases of discrepancy with the computer classification, a second reviewer served as an adjudicator. This system, while not perfect, was well standardized, ensuring high specificity of incident stroke diagnosis in the cohort. Our subtype classification into hemorrhagic stroke and ischemic stroke is probably accurate in most, if not all, cases. However, the categories of embolic stroke and lacunar stroke were more prone to errors.

Another strength is that the prospective follow-up procedures in ARIC are less prone to missing hospitalized cases than are retrospective event identification methods. It is possible, however, that nonhospitalized events (not investigated in ARIC) could influence the results. Still, the magnitude of any potential underestimation of stroke rates is likely to be small (<5%), especially given ARIC’s youthful (aged 45 to 64 at baseline) cohort. Furthermore, the ARIC cohort was drawn from 4 geographically diverse communities, including 1 from the stroke belt.

The validity of baseline risk status is also imperfect. For example, defining hypertension status from a single baseline measurement of blood pressure or antihypertensive medica-

tion use does not capture the full extent of a person’s exposure to the risks of elevated blood pressure over many years. Accordingly, adjustment of incidence rate ratios for baseline risk factors may not entirely account for the effects of those risk factors.

In conclusion, we found that after accounting for several established risk factors, blacks still have >30% more first-time ischemic strokes than whites. It is likely that this difference in incidence explains much of the excess stroke mortality long associated with blacks. Identification of new individual and community-level risk factors accounting for the elevated incidence of stroke requires further investigation and incorporation into intervention planning.

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References


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