Stroke Incidence Is Decreasing in Whites But Not in Blacks

A Population-Based Estimate of Temporal Trends in Stroke Incidence From the Greater Cincinnati/Northern Kentucky Stroke Study

Dawn O. Kleindorfer, MD; Jane Khoury, PhD; Charles J. Moomaw, PhD; Kathleen Alwell, RN; Daniel Woo, MD; Matthew L. Flaherty, MD; Pooja Khatri, MD; Opeolu Adeoye, MD; Simona Ferioli, MD; Joseph P. Broderick, MD; Brett M. Kissela, MD

Background and Purpose**—**Although other studies (in largely white populations) have found that stroke incidence declined during the 1990s, we previously reported that stroke incidence in our population (18% of which was black) did not change during that decade and that incidence rates in blacks were significantly higher than in whites. We sought to update temporal trends in stroke incidence by adding new data obtained from our large, biracial population in 2005. The objective of this study was to examine temporal trends in stroke incidence and case-fatality within a large biracial population over time by comparing stroke incidence rates from 1993 to 1994, 1999, and 2005.

Methods**—**Within the Greater Cincinnati/Northern Kentucky population of 1.3 million, all strokes among area residents were ascertained at all local hospitals during July 1993 to June 19/94 and calendar years 1999 and 2005. A sampling scheme was used to ascertain cases in the out-of-hospital setting. Only first-ever strokes were included in this analysis. Race-specific incidence rates, standardized to the 2000 US Census population, and case-fatality rates were calculated.

Results**—**The number of physician-confirmed first-ever strokes in patients ≥20 years of age was 1942 in 1993 to 1994, 2041 in 1999, and 1921 in 2005. In all study periods, blacks had higher stroke incidence than whites, and case-fatality rates were similar between races. In contrast to previous study periods, we found a significant decrease in overall stroke incidence in 2005. When stratified by race and stroke subtype, this change was driven by a decrease in ischemic stroke incidence among whites, whereas ischemic stroke incidence in blacks was unchanged. Hemorrhagic stroke incidence was unchanged in both races.

Conclusions**—**For the first time, we report a significant decrease in stroke incidence within our population, which is consistent with other reports in the literature. This decrease was found only among whites, which suggests a worsening of the racial disparity in stroke incidence. **(*Stroke*. 2010;41:00-00.)**

Key Words: acute stroke ■ intracerebral hemorrhage ■ intracranial hemorrhage ■ women and minorities

Stroke is the third leading cause of death and a leading cause of major adult disability in the United States.[1,2] Understanding trends in stroke incidence over time is of major public health concern, especially as newer prevention and risk factor modification strategies are developed. A few population-based studies in the literature have tracked stroke incidence during recent decades. Rothwell et al reported a decrease in stroke incidence of nearly 40% between the 1980s and 2002 in Oxfordshire, UK.[3] Lakshminarayan et al reported that the incidence of stroke in Minnesota was stable in both men and women between 1990 and 2000.[4] The Framingham cohort study also found that the incidence of stroke was declining over the past 50 years.[5]

Many of the previous population-based studies have not included a significant proportion of minorities.[3–7] It has been documented that stroke incidence and mortality are different in minority populations when compared with whites both in the United States and in other countries.[1,8–10] Of note, Carter et al found divergent trends in stroke incidence over time in Auckland, New Zealand, in that white rates were decreasing but other race/ethnic minority rates were stable or increasing between 1981 and 2003. Therefore, stroke incidence trends derived from a US population with minorities are needed.

The Greater Cincinnati/Northern Kentucky Stroke Study (GCNKSS) is designed to investigate stroke incidence rates and case-fatality in the biracial population of a 5-county region that includes the city of Cincinnati. Our study population of 1.3 million is similar to the United States with regard to median age, percent black, median household income, and education level; however, it does not contain a substantial

Received December 8, 2009; final revision received March 11, 2010; accepted March 15, 2010.

From the Department of Neurology (D.O.K.), University of Cincinnati College of Medicine, Cincinnati, Ohio; and the University of Cincinnati (J.K., C.J.M., K.A., D.W., M.L.F., P.K., O.A., S.F., J.P.B., B.M.K.), Cincinnati, Ohio.

Correspondence to Dawn O. Kleindorfer, University of Cincinnati, Department of Neurology, MSB #0525, Cincinnati, OH 45242. E-mail dawn.kleindorfer@uc.edu

Stroke **is available at http://stroke.ahajournals.org** **DOI: 10.1161/STROKEAHA.109.575043**

proportion of persons of Hispanic ethnicity.[11,12] Previously, the GCNKSS reported that a comparison of the incidence of stroke in the study periods July 1993 to June 1994 versus calendar year 1999 showed that incidence did not significantly change for either blacks or whites, which suggests that stroke incidence was stable during the 1990s.[13] We now present data from 2005, our most recent study period, and compare incidence and case-fatality of stroke in 2005 with prior study periods of 1999 and 1993 to 1994.

Methods

The Greater Cincinnati/Northern Kentucky (GCNK) region includes 2 southern Ohio counties and 3 contiguous northern Kentucky counties that border the Ohio River. The study population of the GCNK region consists of <3% Hispanic and other minorities; thus, we calculated rates only for black and white patients. Only residents of the 5 study counties are considered for case ascertainment. In the GCNK region, 19 hospitals were active in 1993 to 1994, 18 in 1999, and 17 in 2005. Previous studies have documented that residents of the 5 counties who have a stroke exclusively seek care at these hospitals rather than at hospitals in the outlying region.[11] This study was approved by the Institutional Review Board at all participating hospitals for each study period.

The GCNKSS involved ascertainment of all stroke events that occurred in the population between July 1, 1993, and June 30, 1994, and again in the calendar years of 1999 and 2005. Details of the previous study periods' case ascertainment have been previously published.[13] In 2005, screening was virtually identical to those techniques used in previous study periods. Study nurses screened the medical records of all inpatients with primary or secondary stroke-related International Classification of Diseases, 9th Revision discharge diagnoses (430 to 436) from the 17 acute care hospitals in the study region. In addition, strokes not found by inpatient screening were ascertained by monitoring all stroke-related visits to hospital emergency departments (with the exception of Cincinnati Children's Hospital) and to the region's 9 public health clinics and 7 hospital-based outpatient clinics and family practice centers. Cases for which stroke was listed as the primary or secondary cause of death by 1 of the 5 county coroners' offices were also included. Further monitoring was performed by examining the records of potential stroke cases in a random sample of 51 of the 832 primary care physicians' offices and 25 of the 126 nursing homes in the GCNK region. Sampling was necessary given the large number of physician offices and nursing homes in the region. Sites were selected randomly by the study statistician from a list generated from a combination of the local yellow pages and the American Medical Association listing of physicians in the region. Events found by out-of-hospital monitoring were crosschecked against inpatient records to prevent double counting.

To qualify as a GCNKSS incident case, a patient must have met the criteria for 1 of the clinically based stroke categories adapted from the Classification for Cerebrovascular Diseases III[14] and from epidemiological studies of stroke in Rochester, Minn[6]: cerebral ischemia, intracerebral hemorrhage (ICH), subarachnoid hemorrhage (SAH), or stroke of uncertain cause. Imaging results were not considered in this clinical definition except for the presence of hemorrhage on CT or MRI for hemorrhagic events. Only first-ever-in-a-lifetime events were included in this analysis. Transient ischemic attacks, defined as symptoms lasting <24 hours regardless of imaging results, were not included in this analysis. The onset of stroke symptoms must have occurred within the study time periods. Charts were screened for an additional 60 days beyond the end of the study periods to capture patients who had a stroke during the study period but had not been discharged. A study physician reviewed every abstract to verify whether a stroke or transient ischemic attack had occurred. The physician assigned a stroke category and mechanism to each event based on all available information using definitions listed previously.

In addition, for the 2005 study period, physicians were asked to give a separate clinical judgment about whether a stroke had occurred after taking into account all available information, including imaging results. To have consistent case identification for all 3 study periods, only cases identified by the strict clinical definition were included in the present analysis.

Potential cases were excluded if they had a discharge/autopsy diagnosis or neuroimaging consistent with stroke but no clinical history consistent with stroke or if they had a clinical diagnosis of stroke and died within 24 hours of symptom onset but had no focal neurological deficit and no confirmatory neuroimaging or autopsy. Once potential cases were identified, a study research nurse abstracted information regarding stroke symptoms, physical examination findings, medical/surgical history, medication use before stroke, social history/habits, prehospital evaluation, vital signs and emergency room evaluation, neurological evaluation, diagnostic test results (including laboratory testing, electrocardiographic and cardiac testing, and neuroimaging of any type), treatments, outcome, type of insurance, and current address. Stroke severity was estimated through a validated method of retrospective National Institutes of Health Stroke Scale score obtained from review of the physician examination as documented in the emergency department evaluation. Classification of race/ethnicity was as self-reported in the medical administrative record. The research nurse made a determination as to whether a stroke or transient ischemic attack had occurred. Nurse abstractors were instructed to consult with study physicians for any questionable cases. If the nurse abstractor was unsure whether a stroke occurred, the event was abstracted so a study physician could determine of whether the event was a stroke. In general, the study personnel remained constant across the 3 study periods. When new personnel were introduced, they underwent an extensive training period that was supervised by either the study principal investigator or the lead study nurse coordinator. This, in conjunction with our detailed physician and research nurse study manuals that describe screening, abstraction, and reviewing procedures, ensured a continuity of methods across the 3 study periods.

Population-Based Survey

Our general population was surveyed regarding self-reported stroke risk factors and medication use in 1995, 2000, and again in 2005. The detailed methods for this random digit-dial telephone survey have been previously published and are available in an online supplement to this article (available at http://stroke.ahajournals.org).[15–17] The phone survey is designed to ensure that the respondents represent a randomly selected group of individuals whose demographics (age, race, and gender) closely match the expected demographics of the population of patients with stroke.

Calculation of Incidence and Case-Fatality

Incidence rates for first-ever stroke were calculated using 2 measures: strokes ascertained in hospital settings only and "all" strokes (ascertained in either hospital or out-of-hospital settings such as nursing homes, clinics, and physician offices). The numerator for calculation of the hospital-ascertained incidence rate was the number of first-ever strokes confirmed by physician review, ascertained through inpatient records, coroners' offices, or emergency departments. The numerator for the incidence rate for "all strokes" also included the number of first-ever strokes ascertained through public health clinics, hospital-based outpatient clinics and family practice centers, and coroners' offices plus a weighted estimate of the number of strokes ascertained only in the physician's office or nursing home. Events ascertained in physicians' offices and nursing homes were multiplied approximately 16- and 5-fold, respectively, for 2005 events, to account for the sampling methods. Cases ascertained from hospital-based clinics were not weighted because all the clinics in the region were screened. Events were considered to be noncases if medical records could not be located (0.2% to 0.7% of events in previous study periods).

The denominator for the calculation of incidence rates was extracted from the US Census Bureau web site (www.census.gov). The estimates are based on interpolation and extrapolation of county population between enumerated census years accounting for births,

Table 1. Adult Overall Annual Incidence of First-Ever Hospital-Ascertained Stroke per 100 000 in 1993 to 1994, 1999, and 2005*

	1993 to 1994 (95% CI)	1999 (95% CI)	2005 (95% CI)
All stroke subtypes	214 (204, 223) [n=1907]	215 (206, 225) [n=1995]	189* (181, 198) [n=1888]
Ischemic	188 (179, 197) [n=1672]	189 (180, 198) [n=1748]	167* (159, 175) [n=1664]
ICH	28 (25, 32) [n=255]	32 (28, 36) [n=299]	32 (29, 36) [n=321]
SAH	9 (7, 11) [n=85]	10 (8, 12) [n=95]	9 (7, 11) [n=91]
Whites only			
All stroke subtypes	201 (191, 211) [n=1564]	203 (194, 213) [n=1646]	175† (166, 184) [n=1496]
Ischemic	178 (169, 188) [n=1388]	180 (170, 189) [n=1453]	154† (146, 162) [n=1319]
ICH	25 (22, 29) [n=197]	29 (25, 33) [n=233]	30 (27, 34) [n=259]
SAH	9 (6, 11) [n=68]	9 (7, 11) [n=74]	8 (6, 10) [n=64]
Blacks only			
All stroke subtypes	325 (290, 360) [n=343]	305 (273, 338) [349]	308 (277, 339) [n=392]
Ischemic	271 (239, 303) [n=284]	263 (233, 293) [n=395]	275 (246, 305) [n=345]
ICH	54 (40, 68) [n=58]	58 (44, 72) [n=66]	48 (36, 60) [n=62]
SAH	15 (8, 22) [n=17]	15 (8, 21) [n=21]	20 (12, 27) [n=27]

*$P<0.001$ for overall trend over time and for 2005 versus 1999.
†$P<0.001$ for overall trend over time and for 2005 versus 1999.
*Age- and gender-adjusted to the 2000 US Census.

deaths, and migration. The at-risk population included 218 906 blacks and 1 100 950 whites for 2005. The 95% CIs for the incidence rates were calculated assuming a Poisson distribution. Age-, race-, and gender-specific rates were also determined. All adjusted rates were standardized to the 2000 US population. Rates were calculated for adults only (age ≥20 years).

Case-fatality was defined as death from any cause within 30 days of stroke. To verify vital status for case-fatality calculations, death certificate data from the Ohio and Kentucky Departments of Vital Statistics were reviewed first. The national Social Security Death Index was queried for any patient who did not have an Ohio or Kentucky death record. Death during hospitalization, as recorded in the medical record abstract, was confirmed in at least 1 of the mortality sources. Patients for whom a death record could not be found were presumed to be alive.

Trends over time were tested by comparing age- and gender-adjusted incidence rates from 1 time period to the next and from the first study period to the last using a Bonferroni correction for multiple comparisons. We confirmed these results by a generalized linear model (using PROC GENMOD in SAS). The model included year, race, age, and gender and the interaction between year and race (results not shown).

Results

There were 1942 first-ever strokes among residents of the 5-county area ≥20 years of age in 1993 to 1994, 2041 in 1999, and 1921 in 2005. The demographics of these defined stroke populations were similar across study periods: in 1993 to 1994, the stroke population was 18.2% black and 57.0% female, average age 71.2±13.5 years (mean±SD); in 1999, the population was 17.5% black and 57.6% female, age 70.9±14.5 years; and in 2005, the population was 20.9% black and 55.1% female, age 68.4±15.4 years. The 2005 stroke population had a statistically significantly higher proportion of blacks and was younger than both the 1993 to 1994 and 1999 populations.

The overall annual incidence of adult, first-ever, hospital-ascertained stroke significantly decreased in 2005 when compared with previous study periods: 189 per 100 000 in 2005 compared with 214 in 1993 to 1994 and 215 in 1999 ($P<0.001$). Race-specific incidence rates of first-ever hospitalized stroke in adults are presented in Table 1. In whites, the incidence of all first-ever stroke and first-ever ischemic stroke significantly decreased in 2005 compared with prior study periods; however, we did not find a similar decline among blacks. The inclusion of cases ascertained in out-of-hospital settings (Table 2) did not change these findings. For both races, the incidence of ICH and SAH did not change across study periods.

Table 2. Adult Race-Specific Annual Incidence of First-Ever Stroke per 100 000 in 1993 to 1994, 1999, and 2005, Hospital Plus Out-of-Hospital Ascertainment*

	Blacks			Whites		
	1993 to 1994 (95% CI)	1999 (95% CI)	2005 (95% CI)	1993 to 1994 (95% CI)	1999 (95% CI)	2005 (95% CI)
All stroke subtypes	357 (320, 394)	334 (300, 368)	331 (299, 364)	228 (217, 238)	265 (253, 276)	208 (199, 218)
Ischemic	303 (269, 337)	291 (259, 323)	294 (164, 325)	206 (195, 216)	241 (230, 252)	179 (170, 188)
ICH	54 (40, 68)	59 (45, 73)	56 (43, 70)	25 (22, 29)	32 (28, 35)	30 (27, 34)
SAH	15 (8, 22)	15 (8, 21)	20 (12, 27)	9 (6, 11)	9 (7, 11)	8 (6, 10)

*Age- and gender-adjusted to the 2000 US Census.

Table 3. Adult 30-Day Case-Fatality Rates After First-Ever Stroke in 1993 to 1994, 1999 and 2005 by Race and Stroke Subtype, Inpatient Plus Out-of-Hospital Ascertainment

	1993 to 1994			1999			2005		
	All†	Black*	White*	All†	Black*	White*	All†	Black*	White*
All stroke subtypes	13.8%	12.9%	14.8%	14.3%	12.8%	16.0%	15.0%	14.1%	16.0%
Ischemic	9.3%	7.8%	11.2%	9.5%	8.5%	10.7%	10.2%	10.1%	10.4%
ICH	34.6%	34.1%	34.6%	38.6%	37.3%	39.9%	39.6%	35.7%	43.9%
SAH	33.7%	34.0%	33.3%	31.9%	32.4%	31.3%	23.4%	20.8%	26.2%

*Adjusted for age and gender.
†Adjusted for age, gender, and race.

Thirty-day case-fatality rates for overall stroke (13.8% in 1993 to 194, 14.3% in 1999, 15.0% in 2005), and for each stroke subtype, were not significantly different among the study periods. Table 3 presents the case-fatality rates, stratified by race and stroke subtype, for the 3 study periods. A numeric decrease in SAH case-fatality was not statistically significant, although the number of SAH cases was relatively smaller compared with other stroke subtypes.

Stroke risk factor disease prevalences within the population, as ascertained from the population-based telephone survey, are presented in Table 4 stratified by race. In every study period, the blacks surveyed had higher prevalences of hypertension, diabetes, and current smoking and very similar prevalences of hypercholesterolemia, prior stroke, and heart disease when compared with whites. Regarding temporal trends, white rates of hypertension significantly increased over time, whereas blacks remained stable, and conversely, rates of diabetes increased among blacks but were stable in whites. Hypercholesterolemia increased in both races, and prior stroke, heart disease, and current smoking remained unchanged in both races over time.

Medication use is also presented in Table 4. In all 3 study periods, blacks were more likely to report use of antihypertensive medication, insulin, and oral medications for diabetes (except for in 1995). Whites were significantly more likely to report aspirin use than blacks in all 3 study periods, and there were no significant racial differences in reported use of lipid-lowering agents. Regarding temporal trends, both blacks and whites reported significantly increased rates of aspirin and lipid-lowering agents over time. Whites reported increasing rates of antihypertensive use, whereas black rates were stable; conversely oral medication for diabetes increased among blacks but was stable in whites over time.

Table 4. Population-Based Survey Results: Demographics, Stroke Risk Factor Prevalence, and Medication Use in 1995, 2000, and 2005

	1995 Survey (n=1855)		2000 Survey (n=2111)		2005 Survey (n=2156)		Overall Trend *P* Value	
	White (N=1348)	Black (N=507)	White (N=1557)	Black (N=554)	White (N=1547)	Black (N=529)	White (N=4452)	Black (N=1590)
Medication use (%)								
Antihypertensive medication	408 (30)	232 (46)*	570 (37)	267 (48)*	638 (41)	270 (51)*	<0.0001	0.09
Insulin for diabetes	37 (3)	37 (7)*	45 (3)	43 (8)*	51 (3)	45 (8)*	0.38	0.47
Oral medication for diabetes	87 (6)	40 (8)	144 (9)	84 (15)†	132 (8)	92 (17)*	0.08	<0.0001
Lipid-lowering agent	127 (9)	56 (11)	316 (20)	109 (20)	468 (30)	145 (27)	<0.0001	<0.0001
Aspirin use	393 (29)	87 (17)*	587 (38)	165 (30)†	611 (40)	189 (36)	<0.0001	<0.0001
Risk factor prevalence (%)								
Hypertension	520 (39)	289 (57)*	650 (42)	307 (55)*	713 (46)	311 (59)*	<0.0001	0.55
Hypercholesterolemia	436 (32)	168 (33)	604 (39)	199 (36)	694 (45)	211 (40)	<0.0001	0.02
Current smoker	264 (20)	135 (27)†	283 (18)	139 (25)†	283 (18)	133 (25)*	0.39	0.59
Diabetes	166 (12)	86 (17)†	210 (13)	129 (23)*	220 (14)	138 (26)*	0.13	0.0004
Heart disease	132 (10)	53 (10)	157 (10)	46 (8)	139 (9)	46 (9)	0.44	0.33
Prior stroke or transient ischemic attack	89 (7)	39 (8)	103 (7)	48 (9)	118 (8)	54 (10)	0.27	0.15
Demographics								
Mean age (SD)	62.8 (16.4)	63.3 (15.5)	61.3 (17.1)	60.1 (17.7)	62.3 (17.1)	59.4 (17.9)†	0.54	0.0003
No. of women (%)	790 (59)	319 (63)	912 (59)	393 (71)*	917 (59)	356 (67)†	0.71	0.14
Education >12 years, no. (%)	598 (44)	144 (28)*	819 (53)	233 (42)*	862 (56)	255 (48)*	<0.001	<0.0001

*$P<0.001$ black vs white.
†$P<0.05$ black vs white.

Discussion

For the first time, we report a decline in stroke incidence among whites within our population. This was driven by a decrease in ischemic stroke incidence, because the hemorrhagic stroke incidence remained stable. This decline was also present when out-of-hospital ascertainment was included, which suggests that the decrease is not likely due to changes in admission rates versus outpatient management of patients with stroke.

We did not find a similar decline within blacks in our community, which suggests that the well-described substantial racial disparity in stroke incidence is worsening. Given the aging of our population, even with stable incidence rates of stroke among blacks, the total number of stroke events should continue to increase over time and could become an even larger burden for the black community.[18]

Thirty-day all-cause case-fatality remained constant across all study periods for both blacks and whites. Previously we reported that the greater "stroke mortality" reported at the national level in blacks is due to greater stroke incidence.[11–13] This finding is confirmed in the present study, because blacks continue to have a similar case-fatality but nearly twice the stroke incidence when compared with whites.

There was a striking decline in the point estimates of overall SAH case-fatality, from 34% in 1993 to 1994 to 23% in 2005. Although this was not statistically significant, likely due to small numbers, it fits with previous reports in the literature of declining case-fatality related to SAH.[19,20] Further analysis of this trend, and potential underlying factors, is underway.

The lack of a significant change in the age-adjusted incidence rates of stroke for blacks in the GCNK region is disappointing. This is especially true given the primary and secondary prevention efforts for cardiovascular disease within our community during the 1990s, which included a focus on racial disparity in stroke incidence. Our population-based telephone survey of the general population describing the prevalence of risk factors and self-reported medication use does not easily explain this racial disparity. Blacks continue to have higher rates of many stroke risk factors, but the use of medications to treat these diseases is also higher among blacks. Although diabetes appears to be increasing over time in blacks, hypertension does not. These population-level trends in prevalence and medication use are complex and cannot easily explain the trends seen in stroke incidence. Furthermore, there are many other considerations for which our study cannot provide data, including but not limited to access to care, age of onset of disease, severity of disease, and medication compliance, among others. It is well documented that blacks have earlier onset of vascular risk factors, which tend to be more severe and refractory to treatment.[21,22] Less effective control of stroke risk factors in blacks could then explain the difference in stroke incidence rates. We are unable to address this possibility, because we do not have populationwide data regarding the quality of care or medication compliance for risk factors such as hypertension, diabetes, and smoking. Another potential explanation is that there is a time delay in the effects of improved treatment of stroke risk factors and that improved risk factor management among blacks will eventually lead to a similar decrease in stroke incidence as whites in the future.

Although our method for identification of hospitalized strokes remained consistent across the 3 study periods, the potential for bias of incomplete case ascertainment is important to consider in any study that examines temporal trends in the incidence of a disease. Our additional use of passive surveillance of emergency rooms, nursing homes, physician offices, and clinics should reduce chances of incomplete ascertainment. In addition, the random sampling of offices and nursing homes assumes a uniform distribution of strokes by region; this, of course, may not be the case, particularly because differences by race may impact the assumption of uniformity. Our method for identification of hospitalized strokes, however, remained consistent across the 3 study periods. Although changes in clinical practice such as increased frequency of MRI may have an effect the detection and diagnosis of stroke, the case definition for stroke used in this analysis was based strictly on the presence and duration of focal clinical symptoms rather than imaging findings and was consistent across study periods. This is confirmed by the similar case-fatality across study periods. Therefore, we believe that our consistent methods and clinical case definition over the 3 study periods has minimized possible ascertainment biases for hospitalized strokes. In addition, any incidence study that relies on medical contact for counting of events risks missing events that were not recognized by the general public as needing medical attention. Our study does not have the ability to track changes over time in mortality from other causes. It is possible that improvement in treatment or mortality of other diseases such as heart disease could potentially impact stroke incidence rates, but only if those patients who have now survived are at higher risk for stroke. Further analysis of age-specific incidence rates are underway. Finally, the power we have to detect small changes in incidence among blacks is more limited than it is among whites. Nevertheless, our study is the largest population-based study of stroke incidence in the United States, and it is 1 of the few studies in the world that includes a substantial number of black patients. All of these considerations suggest that several counterbalancing biases may influence the final incidence rates of stroke that we observed.

Clearly, there is still much work to be done to understand racial disparities and temporal trends in stroke incidence. Population-based studies of temporal trends in stroke incidence rates are critical for providing a report card of our overall progress in primary stroke prevention as well as our efforts to reduce the continuing disparity in incidence rates between black and white populations in the United States.

Disclosures

None.

References

1. Disparities in deaths from stroke among persons aged <75 years—United States, 2002. *MMWR Morb Mortal Wkly Rep.* 2005;54:477–481.
2. Centers for Disease Control and Prevention. Prevalence of disabilities and associated health conditions—United States, 1991–92. *MMWR Morb Mortal Wkly Rep.* 1994;43:730–739.

3. Rothwell PM, Coull AJ, Giles MF, Howard SC, Silver LE, Bull LM, Gutnikov SA, Edwards P, Mant D, Sackley CM, Farmer A, Sandercock PA, Dennis MS, Warlow CP, Bamford JM, Anslow P. Change in stroke incidence, mortality, case-fatality, severity, and risk factors in Oxfordshire, UK from 1981 to 2004 (Oxford Vascular Study). *Lancet*. 2004;363:1925–1933.
4. Lakshminarayan K, Anderson DC, Jacobs DR Jr, Barber CA, Luepker RV. Stroke rates: 1980–2000: the Minnesota stroke survey. *Am J Epidemiol*. 2009;169:1070–1078.
5. Carandang R, Seshadri S, Beiser A, Kelly-Hayes M, Kase CS, Kannel WB, Wolf PA. Trends in incidence, lifetime risk, severity, and 30-day mortality of stroke over the past 50 years. *JAMA*. 2006;296:2939–2946.
6. Brown R, Whisnant J, Sicks J, O'Fallon W, Wiebers D. Stroke incidence, prevalence, and survival: secular trends in Rochester, Minnesota, through 1989. *Stroke*. 1996;27:373–380.
7. Shahar E, McGovern PG, Pankow JS, Doliszny KM, Smith MA, Blackburn H, Luepker RV. Stroke rates during the 1980s. The Minnesota stroke survey. *Stroke*. 1997;28:275–279.
8. Carter K, Anderson C, Hacket M, Feigin V, Barber PA, Broad JB, Bonita R. Trends in ethnic disparities in stroke incidence in Auckland, New Zealand, during 1981 to 2003. *Stroke*. 2006;37:56–62.
9. Howard G, Howard VJ. Ethnic disparities in stroke: the scope of the problem. *Ethn Dis*. 2001;11:761–768.
10. White H, Boden-Albala B, Wang C, Elkind MS, Rundek T, Wright CB, Sacco RL. Ischemic stroke subtype incidence among whites, blacks, and Hispanics: the Northern Manhattan study. *Circulation*. 2005;111:1327–1331.
11. Broderick J, Brott T, Kothari R, Miller R, Khoury J, Pancioli A, Gebel J, Mills D, Minneci L, Shukla R. The Greater Cincinnati/Northern Kentucky stroke study: preliminary first-ever and total incidence rates of stroke among blacks. *Stroke*. 1998;29:415–421.
12. Kissela B, Schneider A, Kleindorfer D, Khoury J, Miller R, Alwell K, Woo D, Szaflarski J, Gebel J, Moomaw C, Pancioli A, Jauch E, Shukla R, Broderick J. Stroke in a biracial population: the excess burden of stroke among blacks. *Stroke*. 2004;35:426–431.
13. Kleindorfer D, Broderick J, Khoury J, Flaherty M, Woo D, Alwell K, Moomaw CJ, Schneider A, Miller R, Shukla R, Kissela B. The unchanging incidence and case-fatality of stroke in the 1990s: a population-based study. *Stroke*. 2006;37:2473–2478.
14. National Institute of Neurological Disorders and Stroke. Classification of neurological disorders III. *Stroke.* 1990;21:637–676.
15. Schneider AT, Pancioli AM, Khoury JC, Rademacher E, Tuchfarber A, Miller R, Woo D, Kissela B, Broderick JP. Trends in community knowledge of the warning signs and risk factors for stroke. *JAMA*. 2003;289:343–346.
16. Kleindorfer D, Khoury J, Broderick JP, Rademacher E, Woo D, Flaherty ML, Alwell K, Moomaw CJ, Schneider A, Pancioli A, Miller R, Kissela BM. Temporal trends in public awareness of stroke: warning signs, risk factors, and treatment. *Stroke*. 2009;40:2502–2506.
17. Pancioli AM, Broderick J, Kothari R, Brott T, Tuchfarber A, Miller R, Khoury J, Jauch E. Public perception of stroke warning signs and knowledge of potential risk factors. *JAMA*. 1998;279:1288–1292.
18. Broderick JP. William M. Feinberg lecture: stroke therapy in the year 2025: burden, breakthroughs, and barriers to progress. *Stroke*. 2004;35: 205–211.
19. Goldacre MJ, Duncan M, Griffith M, Rothwell PM. Mortality rates for stroke in England from 1979 to 2004: trends, diagnostic precision, and artifacts. *Stroke*. 2008;39:2197–2203.
20. Qureshi AI, Suri MF, Nasar A, Kirmani JF, Divani AA, He W, Hopkins LN, Trends in hospitalization and mortality for subarachnoid hemorrhage and unruptured aneurysms in the united states. *Neurosurgery*. 2005; 57:1–8; discussion 1–8.
21. Sacco RL, Boden-Albala B, Abel G, Lin IF, Elkind M, Hauser WA, Paik MC, Shea S. Race–ethnic disparities in the impact of stroke risk factors: the Northern Manhattan stroke study. *Stroke*. 2001;32:1725–1731.
22. Howard G, Prineas R, Moy C, Cushman M, Kellum M, Temple E, Graham A, Howard V. Racial and geographic differences in awareness, treatment, and control of hypertension: the reasons for geographic and racial differences in stroke study. *Stroke*. 2006;37:1171–1178.
23. Ezzati TM, Massey JT, Waksberg J, Chu A, Maurer KR. Sample design: Third National Health and Nutrition Examination Survey. *Vital Health Stat 2*. 1992:1–35.

Supplemental Material

Population-Based Telephone Survey Methods

We used random-digit selection of telephone numbers and random respondent selection within a household. Details of this method have been previously described.[15,17] Because 97.8% of the households in Cincinnati reported having telephone service in 2000, only a small proportion of the population would have been omitted from this sampling technique.

The 64 professional telephone interviewers for the University of Cincinnati Institute for Policy Research who administered the survey were monitored for quality and comparability. The supervisor randomly monitored 20% of the interviews by using special telephone lines or direct computer screen viewing.

To ensure that the demographic characteristics of the respondents approximated those of the ischemic stroke population, we created a demographic table that contained the desired number of respondents in each of the demographic categories of age, race, and gender. To maintain consistency, the age, race, and gender demographics were determined by the same method in both study periods. We interviewed only those respondents whose demographic characteristics matched an unfilled category.

The survey instrument consisted of 29 questions divided into 3 sections. Respondents were asked questions designed to assess the prevalence of stroke risk factors. These questions were identical to questions used in the Third National Health and Nutrition Examination Survey.[23] A copy of the questionnaire is available on request from the authors.

Ascertainment Quality Assurance Supplemental Methods

In addition to retrospective ascertainment by International Classification of Disease codes, in 1999 and 2005, we also implemented a prospective case ascertainment technique. Admission logs of all local emergency departments were screened by study nurses for stroke-like symptoms (eg, weakness, numbness, vision change, problems speaking). Charts for patients suspected of having a stroke were abstracted regardless of International Classification of Diseases, 9th Revision code assignment. In a cohort of 502 cases gathered prospectively, only 19 were found to have International Classification of Diseases, 9th Revision codes other than the screened 430 to 436. This suggests that our retrospective methods may have missed approximately 3% to 4% of stroke cases. Cases ascertained only by prospective monitoring were not included in calculation of incidence rates in 1999 and 2005 for this report to keep methods consistent with that of 1993 to 1994.

Stroke
JOURNAL OF THE AMERICAN HEART ASSOCIATION

American Heart Association | American Stroke Association

Stroke Incidence Is Decreasing in Whites But Not in Blacks. A Population-Based Estimate of Temporal Trends in Stroke Incidence From the Greater Cincinnati/Northern Kentucky Stroke Study

Dawn O. Kleindorfer, Jane Khoury, Charles J. Moomaw, Kathleen Alwell, Daniel Woo, Matthew L. Flaherty, Pooja Khatri, Opeolu Adeoye, Simona Ferioli, Joseph P. Broderick and Brett M. Kissela

Stroke. published online May 20, 2010;
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0039-2499. Online ISSN: 1524-4628