Ethnic Group Disparities in 10-Year Trends in Stroke Incidence and Vascular Risk Factors
The South London Stroke Register (SLSR)

Peter U. Heuschmann, MD, MPH; Andy P. Grieve, PhD, DSc; Andre Michael Toschke, MD, MPH, MSc; Anthony G. Rudd, FRCP; Charles D.A. Wolfe, MD, FFPHM

Background and Purpose—Data monitoring trends in stroke risk among different ethnic groups are lacking. Thus, we investigated trends in stroke incidence and modifiable stroke risk factors over a 10-year time period between different ethnic groups.

Methods—Changes in stroke incidence were investigated with the South London Stroke Register (SLSR). The SLSR is a population-based stroke register, covering a multiethnic population of 271,817 inhabitants in South London with 63% white, 28% black, and 9% of other ethnic group (2001 Census).

Results—Between 1995 and 2004, 2,874 patients with first-ever stroke of all age groups were included. Total stroke incidence decreased over the 10-year study period in men (incidence rate ratio 1995 to 1996 versus 2003 to 2004 [IRR] 0.82, 95% CI 0.69 to 0.97) and in women (IRR 0.76, 95% CI 0.64 to 0.90). A similar decline in total stroke incidence could be observed in whites for men and women (IRR 0.76, 95% CI 0.62 to 0.93 versus IRR 0.73, 95% CI 0.59 to 0.89, respectively); in blacks, total stroke incidence was reducing only in women (IRR 0.48, 95% CI 0.31 to 0.75). In whites, the prevalence of prior-to-stroke hypertension (P<0.0017), atrial fibrillation (P=0.0113), and smoking (P=0.0177) decreased; no statistically significant changes in prior-to-stroke risk factors were observed in blacks. Total stroke incidence was higher in blacks compared to whites (IRR 1.27, 95% CI 1.10 to 1.46 in men; IRR 1.29, 95% CI 1.11 to 1.50 in women), but the black-white gap reduced during the 10-year time period (IRR 1.43, 95% CI 1.13 to 1.82 in 1995 to 1996 to 1.18, 95% CI 0.93 to 1.49 in 2003 to 2004).

Conclusions—Stroke incidence decreased over a 10-year time period. The greatest decline in incidence was observed in black women, but ethnic group disparities still exist, indicating a higher stroke risk in black people compared to white people. Advances in risk factor reduction observed in the white population were failed transferring to the black population. (Stroke. 2008;39:2204-2210.)

Key Words: stroke ■ epidemiology ■ risk factors ■ ethnicity

In the United States, the direct and indirect costs for stroke in 2006 were estimated at $58 billion; within an aging society the lifetime direct costs are expected to increase dramatically in the next few decades. Monitoring present trends in stroke incidence in the population can inform health policy on decisions about effectiveness of current prevention strategies and prioritization of future actions. Stroke incidence studies without upper age restriction have shown a decline in stroke incidence until the early 1980s with a stabilization or even an increase in stroke incidence in the late 1980s and early 1990s. Few studies on trends in stroke incidence have been published since then and they report conflicting results: rates were found to be stable in France, to decrease in the United Kingdom and in New Zealand, and to increase slightly in the United States. Most of these studies reported changes in stroke incidence between 2 or 3 different time points, with only 1 documenting stroke incidence continuously over the whole time period in the same study population.

In recent years, substantial differences in stroke incidence between different ethnic groups have been revealed. Higher stroke incidence rates were found in black compared to white populations in the United States and the United Kingdom; contrary time trends in stroke incidence have been identified for different ethnic groups, with declining rates in Whites compared to stable or increasing rates in ethnic minorities in New Zealand. Data on continuous monitoring of stroke incidence among different ethnic groups in the same study population and linking the observed trends to changes in vascular risk factors are lacking.
We have investigated time trends in incidence and modifiable risk factors in different ethnic groups over a decade within a population-based stroke register observing a multi-ethnic inner city population in South London.

Methods

Study Population
The South London Stroke Register (SLSR) is a prospective ongoing population-based stroke register set up in January 1995, recording all first-ever strokes in patients of all age groups for an inner area of South London based on 22 electoral wards in Lambeth and Southwark. Data collected between 1995 and 2004 were used in this analysis.

The total source population of the SLSR area was 271,817 with 63% white, 28% black (9% black Caribbean, 15% black African, and 4% black Other), and 9% of other ethnic group at the Census 2001. Between the most recent Censuses 1991 and 2001 the proportion of ethnic groups other than white increased from 28% to 37%; in 1991 the greatest ethnic minority group were black Caribbeans (11%), shifted to black Africans (15%) in 2001. In January 2004 the register area was extended after the revision of electoral ward boundaries in South London; this expansion increased the population covered by the SLSR by about 15%.

Case Ascertainment
Standardized criteria were applied for ensuring completeness of cases ascertainment, including multiple overlapping sources of information.11 Stroke was defined according to WHO criteria.12 All patients with the diagnosis suspected to first or recurrent stroke or to TIA documented in one of the following sources of notification were investigated for eligibility of study inclusion. Patients admitted to hospitals serving the study area (2 teaching hospitals within and 3 hospitals outside the study area) were included by daily reviews of acute wards serving stroke patients, weekly checks of brain imaging referrals, and monthly reviews of bereavement officers and of bed manager records. For detecting patients not admitted to hospital all general practitioners (GPs) within and on the borders of the study area were contacted regularly and asked to notify all stroke patients. Telephone contact, posters, and quarterly newsletters were established for a regular communication with GPs. Referral of nonhospitalized stroke patients to a neurovascular outpatient clinic (since 2003) or domiciliary visit of the patients by the study team was also available to GPs. The neurovascular outpatient clinic was reviewed weekly since 2003. Community therapists were contacted every 3 months. Death certificates were checked regularly. Case ascertainment methods were stable during the study period, except the establishment of the neurovascular outpatient clinic. Completeness of case ascertainment was estimated from a multinominal-logit capture-recapture model using methodology described in detail elsewhere.13

Data Collection
Special trained study nurses and field workers collected all data prospectively. A study clinician verified the diagnosis of stroke. Patients were examined within 48 hours of referral to SLSR where possible. The following information on sociodemographic characteristics and prior-to-stroke risk factors was collected at initial assessment: self-definition of ethnic origin (1991 Census question), stratified into white, black (black-Caribbean, black African, and black Other), and other ethnic group; hypertension (general practice or hospital records of high blood pressure); and other ethnic group; hypertension (general practice or hospital records of high blood pressure [>140 mm Hg systolic or >90 mm Hg diastolic]); diabetes mellitus; smoking; atrial fibrillation. Classification of pathological stroke subtype (ischemic stroke [IS], primary intracerebral hemorrhage [PICH], and subarachnoid hemorrhage [SAH]) was based on results from at least 1 of the following: brain imaging performed within 30 days of stroke onset (computerised tomography or MRI), cerebrospinal fluid analysis (in all living cases of SAH where brain imaging was not diagnostic), or necropsy examination. Cases without known pathological confirmation of stroke subtype were classified as undefined (UND).

Statistical Analysis
The t test was used to test differences in continuous variables, and the χ2 test was used for differences in proportions. The source population of the SLSR from 1995 to 2004 was estimated based on the UK Census figure for 1991 and 2001 by assuming a linear trend. The source population of the SLSR for 2004, taking the extension of the study area into account, was calculated by extrapolation from the extended area population in the 2001 UK Census and assuming the same linear increase of the study population as in the original SLSR area. Crude incidence rates were calculated for age group, gender, ethnic groups, and pathological stroke subtypes: sex, ethnic group, and stroke subtype incidence rates were age-adjusted to the standard European population.14 Confidence intervals (CI) for incidence rate estimates were calculated using the Poisson distribution.15 A multinominal-logit capture-recapture was used for determining the completeness of case ascertainment. The model used a prespecified set of covariates: age, sex, ethnicity, stroke severity (Barthel-Index at day 5 to 10), living conditions prestroke, and prior-to-stroke comorbidities (hypertension, diabetes mellitus, smoking, atrial fibrillation). To increase the number of patients among the different subgroups, data were reported in 2-year time intervals. For investigating potential time trends during the 10-year observation period, the first period was compared with the last period by calculating Incidence Rate Ratios (IRR); 95% CI for the direct standardized IRR were calculated by the delta method.16 Test for trend for changes in risk factors was performed by including biannual time period as ordinal variable in a logistic regression model adjusted for age, sex, and ethnic group as appropriate; potential gender differences for changes in prior-to-stroke risk factors in Whites and Blacks were explored by running separate logistic regression models for men and women. Analyses of trends in risk factors were restricted to patients without missing values in the respective variable; number of missing values ranged from 186 (6.47%) for atrial fibrillation and diabetes mellitus to 224 (7.79%) for smoking. Statistical analyses were performed with SAS software version 9.1 (SAS Institute Inc).

Ethics
Patients or their relatives gave written informed consent to participate in the study. The design of the study was approved by the ethics committees of Guy’s and St Thomas’ Hospital Trust, King’s College Hospital, Queens Square, and Westminster Hospital (London).

Results
Between January 1995 and December 2004, 2874 patients with first-ever stroke were registered in the SLSR. Median age was 72 years (Inter-Quartile-Range 62 to 81), 1447 (50.35%) were females; 2126 (73.97%) of the stroke patients were of white, 525 (18.27%) of black, 153 (5.32%) of other, and 70 (2.44%) of unknown ethnic origin. 427 (14.86%) of the patients were not admitted to hospital. The distribution of pathological subtypes was as follows: ischemic stroke (IS) 2092 (72.79%); primary intracerebral hemorrhage (PICH) 395 (13.74%); subarachnoid hemorrhage (SAH) 171 (5.95%); and 216 (7.52%) were undefined (UND). Information on brain imaging was available for 2826 patients (98.33%). A CT or a MRI scan was done in 89.84% of these patients (white: 88.62%, black: 94.21%; P = 0.0002); the proportion of patients receiving brain imaging increased significantly from 85.49% in 1995/96 to 95.14% in 2003/2004 (P < 0.001); these trends were observed for both white (P < 0.0001) and black (P = 0.0476) stroke patients. Independent capture-recapture models were fitted in 2-year time intervals and completeness was estimated to be 84% in 1995 to 1996, 83% in 1997 to 1998, 76% in 1999 to 2000, 75% in 2000 to 2001, and 81% in 2003 to 2004.
Prior-to-Stroke Vascular Risk Factors

Changes in demographic characteristics, stroke subtypes, and vascular risk factors over the 10-year study period are shown in Table 1. The proportion of patients of ethnic group other than Whites increased; no significant variations were observed for sex. Overall, the prevalence of hypertension, atrial fibrillation, and smoking decreased over the 10-year time period; prior-to-stroke diabetes mellitus increased slightly, although the increase did not reach statistical significance.

Ethnic Disparities in Prior-to-Stroke Risk Factors

Table 2 reports trends in demographic characteristics, stroke subtypes, and vascular risk factors stratified for white and black ethnic group. In white and black patients, no significant changes in sex distribution were observed. In both ethnic groups, the proportion of undefined strokes decreased and of ischemic stroke increased. In black patients, a substantial decrease in patients with SAH was observed. In white patients, the prevalence of prior-to-stroke hypertension, atrial fibrillation, and smoking decreased; diabetes mellitus showed a borderline statistically significant increase (P for trend 0.0686). In black patients, a borderline statistically significant decrease in prevalence of prior-to-stroke hypertension was observed (P for trend 0.0586), more pronounced for female (P for trend 0.0386) compared to male (P for trend 0.4826); no other statistically significant trends or gender differences were detected.

Stroke Incidence

Annual stroke incidence per 100 000 inhabitants age-standardized to the European population was: for total stroke 148.7 (95% CI 125.8 to 174.6) in men and 108.2 (95% CI 88.8 to 130.6) in women; for IS 108.3 (95% CI 88.9 to 130.7) in men and 79.1 (95% CI 62.6 to 98.6) in women; for PICH 23.4 (95% CI 14.9 to 35.0) in men and 13.0 (95% CI 6.9 to 22.3) in women; for SAH 7.5 (95% CI 3.1 to 15.1) in men and 7.6 (95% CI 3.2 to 15.3) in women; for UND 9.5 (95% CI 4.4 to 17.7) in men and 8.5 (95% CI 3.8 to 16.4) in women.

Changes in European age-standardized incidence rates for stroke subtypes and ethnic groups are presented in Table 3. Overall, total stroke incidence decreased by 18% in men (IRR 2003 to 2004 compared to 1995 to 1996 0.82, 95% CI 0.69 to 0.97) and 24% in women (IRR 0.76, 95% CI 0.64 to 0.90). In whites, no major differences in IRR between men and women were observed; in blacks, a significant decline was observed only in women (IRR 0.48, 95% CI 0.31 to 0.75), mainly caused by a substantial decrease in incidence of PICH (IRR 0.23, 95% CI 0.06 to 0.86). In black men, total stroke incidence did not decline although a significant decrease of SAH was observed (IRR 0.12, 95% CI 0.02 to 0.59).

Prior-to-Stroke Vascular Risk Factors
Changes in demographic characteristics, stroke subtypes, and vascular risk factors over the 10-year study period are shown in Table 1. The proportion of patients of ethnic group other than Whites increased; no significant variations were observed for sex. Overall, the prevalence of hypertension, atrial fibrillation, and smoking decreased over the 10-year time period; prior-to-stroke diabetes mellitus increased slightly, although the increase did not reach statistical significance.

Ethnic Disparities in Prior-to-Stroke Risk Factors
Table 2 reports trends in demographic characteristics, stroke subtypes, and vascular risk factors stratified for white and black ethnic group. In white and black patients, no significant changes in sex distribution were observed. In both ethnic groups, the proportion of undefined strokes decreased and of ischemic stroke increased. In black patients, a substantial decrease in patients with SAH was observed. In white patients, the prevalence of prior-to-stroke hypertension, atrial fibrillation, and smoking decreased; diabetes mellitus showed a borderline statistically significant increase (P for trend 0.0686). In black patients, a borderline statistically significant decrease in prevalence of prior-to-stroke hypertension was observed (P for trend 0.0586), more pronounced for female (P for trend 0.0386) compared to male (P for trend 0.4826); no other statistically significant trends or gender differences were detected.

Stroke Incidence
Annual stroke incidence per 100 000 inhabitants age-standardized to the European population was: for total stroke 148.7 (95% CI 125.8 to 174.6) in men and 108.2 (95% CI 88.8 to 130.6) in women; for IS 108.3 (95% CI 88.9 to 130.7) in men and 79.1 (95% CI 62.6 to 98.6) in women; for PICH 23.4 (95% CI 14.9 to 35.0) in men and 13.0 (95% CI 6.9 to 22.3) in women; for SAH 7.5 (95% CI 3.1 to 15.1) in men and 7.6 (95% CI 3.2 to 15.3) in women; for UND 9.5 (95% CI 4.4 to 17.7) in men and 8.5 (95% CI 3.8 to 16.4) in women.

Changes in European age-standardized incidence rates for stroke subtypes and ethnic groups are presented in Table 3. Overall, total stroke incidence decreased by 18% in men (IRR 2003 to 2004 compared to 1995 to 1996 0.82, 95% CI 0.69 to 0.97) and 24% in women (IRR 0.76, 95% CI 0.64 to 0.90). In whites, no major differences in IRR between men and women were observed; in blacks, a significant decline was observed only in women (IRR 0.48, 95% CI 0.31 to 0.75), mainly caused by a substantial decrease in incidence of PICH (IRR 0.23, 95% CI 0.06 to 0.86). In black men, total stroke incidence did not decline although a significant decrease of SAH was observed (IRR 0.12, 95% CI 0.02 to 0.59).
Changes in Ethnic Disparities in Stroke Incidence

Table 2. Changes in Socio-Demographic Characteristics, Stroke Subtypes, and Vascular Risk Factors Over the 10-Year Study Period Stratified for Ethnic Group

|----------------------|-----------|-----------|-----------|-----------|-----------|------------
| n                    |           |           |           |           |           |            |
| Age                  |           |           |           |           |           |            |
| Median (IQR)         |           |           |           |           |           |            |
| <65 y                | 109 (21.04) | 53 (49.53) | 105 (20.59) | 50 (45.87) | 99 (27.35) | 52 (52.53) | 106 (27.11) | 60 (54.55) | 93 (26.96) | 40 (40.00) | 0.0021* 0.38*
| 65 to 74 y           | 149 (28.76) | 35 (32.71) | 129 (25.29) | 34 (31.19) | 83 (22.93) | 29 (29.29) | 95 (24.30) | 30 (30.91) | 73 (21.16) | 41 (41.00) |
| 75 to 84 y           | 168 (32.43) | 13 (12.15) | 184 (36.08) | 21 (19.27) | 117 (32.32) | 11 (11.11) | 116 (29.67) | 13 (11.82) | 122 (35.36) | 16 (16.00) |
| 85 y+                | 92 (17.76) | 6 (5.61) | 92 (18.04) | 4 (3.67) | 63 (17.40) | 7 (7.07) | 74 (18.93) | 3 (2.73) | 57 (16.52) | 3 (3.00) |
| Female sex, n (%)    | 284 (54.83) | 50 (46.73) | 262 (51.37) | 46 (42.20) | 179 (49.45) | 46 (46.48) | 202 (51.66) | 57 (51.82) | 180 (52.17) | 40 (40.00) | 0.80† 0.65†
| Stroke subtype, n (%) |           |           |           |           |           |            |
| Ischemic stroke      |           |           |           |           |           |            |
| PICH                 | 56 (10.81) | 19 (17.76) | 66 (12.94) | 19 (17.43) | 50 (13.81) | 18 (18.18) | 49 (12.53) | 24 (21.82) | 34 (8.86) | 16 (16.00) |
| SAH                  | 31 (6.06) | 14 (13.08) | 15 (2.94) | 8 (7.34) | 16 (4.42) | 9 (9.09) | 26 (6.65) | 8 (7.27) | 22 (6.38) | 3 (3.00) |
| UND                  | 64 (12.36) | 5 (4.59) | 62 (12.16) | 5 (4.59) | 19 (5.25) | 3 (3.03) | 24 (6.14) | 2 (1.82) | 13 (3.77) | 5 (5.00) |
| Vascular risk factors, n (%) |           |           |           |           |           |            |
| Hypertension         | 353 (70.60) | 86 (81.90) | 304 (65.38) | 73 (73.74) | 151 (50.00) | 61 (70.93) | 216 (56.54) | 75 (69.44) | 213 (64.35) | 70 (72.16) | 0.0017§ 0.0586§
| Diabetes mellitus    | 63 (12.57) | 30 (28.57) | 55 (11.58) | 39 (39.39) | 39 (12.70) | 30 (32.61) | 62 (16.36) | 21 (19.81) | 49 (15.12) | 32 (34.04) | 0.0686§ 0.57§
| Atrial fibrillation  | 118 (23.55) | 5 (4.76) | 120 (25.70) | 7 (7.37) | 52 (16.67) | 5 (4.98) | 65 (17.02) | 5 (4.63) | 65 (19.70) | 6 (6.32) | 0.0113§ 0.92§
| Smoking              | 196 (38.43) | 30 (28.85) | 185 (37.91) | 32 (31.07) | 139 (41.99) | 21 (23.86) | 110 (32.54) | 24 (24.00) | 106 (34.53) | 27 (29.67) | 0.0177§ 0.74§

IQR indicates interquartile range.

*Adjusted for sex; †adjusted for age; ‡adjusted for age and sex; §patients with missing values in the respective variable were excluded from the analysis.

Changes in Ethnic Disparities in Stroke Incidence

Total annual stroke incidence per 100 000 inhabitants age-standardized to the European population was for whites 136.7 (95% CI 114.8 to 161.7) in men compared to 96.5 (95% CI 78.2 to 117.8) in women and for blacks 173.0 (95% CI 148.2 to 200.8) in men compared to 124.5 (95% CI 103.6 to 148.4) in women. In the overall black-to-white age-adjusted IRR was 1.27 (95% CI 1.10 to 1.46) for men and 1.29 (95% CI 1.11 to 1.50) for women; black-to-white IRR was higher in PICH (IRR 1.87, 95% CI 1.36 to 2.56 for men and IRR 1.40, 95% CI 0.93 to 2.12 for women) compared to IS (IRR 1.21, 95% CI 1.02 to 1.44 for men and IRR 1.37, 95% CI 1.15 to 1.63 for women) and SAH (IRR 1.14, 95% CI 0.65 to 2.00 for men and IRR 1.00, 95% CI 0.56 to 1.80 for women). Over the 10-year time period the IRR between blacks and whites decreased from 1.43 (95% CI 1.13 to 1.82) to 1.18 (95% CI 0.93 to 1.49; Figure).

Discussion

Total stroke incidence decreased by 18% in men and 24% in women over the 10-year study period between 1995 and 2004. Reduction of stroke incidence was similar among white men and women; in blacks only a statistically significant decrease of total stroke incidence in black women was observed, mainly attributed to an about 80% decline in PICH rate. Ethnic group disparities in stroke incidence still exist, indicating higher attack rates in blacks; however, the black–white gap in stroke risk was slightly reducing at the end of the 10-year time period. The observed decline in stroke attack rates might be attributed to changes in prior-to-stroke risk factors. In white patients, a decrease in hypertension, smoking, and atrial fibrillation and a statistically nonsignificant increase in prevalence of diabetes mellitus was observed. No changes in the prevalence of the main prior-to-stroke risk factors could be detected in black patients over the study period except a trend toward lower prevalence of hypertension, especially in women.

A review of population-based registers reporting on trends in stroke incidence until the mid-1990s has recently been published; in most studies on time trends in stroke incidence a decrease in incidence to the mid/end of 1980s and an increase in the late 1980s/early 1990s could be observed. Only a few studies reported on trends in incidence since then. The OXVASC study was performed in a mainly white population (94%) of 90 542 individuals registered in 9 general practices in Oxfordshire, UK, and trends in stroke incidence were compared between 2 time points in 1981 to 1984 and 2002 to 2004. In agreement with our findings in the white SLSR population, a decrease in total stroke incidence was observed in men and women, and the trend for decline in PICH incidence was more pronounced compared to IS. In contrast to our findings for white men and women, stable incidence rates for total stroke were reported from the Dijon Stroke Register, comprising a mainly white population of...
150 138 inhabitants in Dijon, France (2004) between 1985 and 2004. In this register the mean age at stroke onset increased in men from 66 years to 71 years and in women from 68 years to 76 years during the 20-year time period. Thus, an increasing life expectancy in the source population might partially offset a potential decrease in stroke risk. Data on trends in stroke incidence in ethnic minorities are scarce. The Auckland Regional Community Stroke (ARCOS) study established a population-based stroke register at 3 time points (1981 to 1982, 1991 to 1992, and 2002 to 2003) in the multiethnic source population of about 940,000 people aged 15 years and above (2001) in Auckland, New Zealand (NZ), comprising 66% NZ/European and 34% other ethnic groups (Maori, Pacific people, and Asians). Total stroke incidence rates declined significantly in NZ/Europeans, similar to our white population. However, in contrast to our findings for the black women, total stroke incidence in ethnic minorities did not significantly decrease over time but a trend toward increasing rates in Maori and Pacific people was reported. The Greater Cincinnati/Northern Kentucky Stroke Study (GCNKSS) observed differences in stroke incidence within a biracial source population of 1.3 million in the United States between a 2-time point documentation in 1993 to 1994 and 1999. In hospitalized patients, annual age- and sex-adjusted total stroke incidence rates and incidence of main pathological subtypes remained stable in Blacks and Whites. In contrast to our findings, a slight increase in total stroke incidence was observed when considering out-of-hospital events, mainly attributed to higher incidence rates of ischemic strokes in Whites. Trends in incidence rates might be influenced by variations in case ascertainment. In addition to the retrospective ascertainment by ICD-9 codes, also a prospective case ascertainment technique was implemented in the GCNKSS in the second time point (screening of emergency departments for patients with stroke-like symptoms); these changes might lead to a more complete case ascertainment among out of hospital strokes and could explain a slight increase in stroke incidence.

The observed decline of stroke incidence in the white SLSR population might be caused by changes in major vascular risk factors. We saw a decline in prior-to-stroke hypertension, smoking, and atrial fibrillation and an increase in diabetes. This is in accordance with the findings from previous studies reporting on

### Table 3. Annual Stroke Incidence Rate per 100 000 Population Adjusted to the European Population

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Ethnic group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>157.37</td>
<td>119.68</td>
<td>169.91</td>
<td>116.29</td>
<td>139.30</td>
<td>0.82 (0.69–0.97)</td>
</tr>
<tr>
<td>Black</td>
<td>147.71</td>
<td>113.47</td>
<td>160.33</td>
<td>104.93</td>
<td>126.10</td>
<td>0.76 (0.62–0.93)</td>
</tr>
<tr>
<td>Other</td>
<td>176.53</td>
<td>74.10</td>
<td>173.69</td>
<td>78.99</td>
<td>186.90</td>
<td>1.13 (0.58–2.19)</td>
</tr>
<tr>
<td>Stroke subtype</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic stroke</td>
<td>115.23</td>
<td>81.09</td>
<td>120.14</td>
<td>83.49</td>
<td>103.46</td>
<td>0.89 (0.73–1.08)</td>
</tr>
<tr>
<td>PICH</td>
<td>20.42</td>
<td>15.23</td>
<td>30.38</td>
<td>17.47</td>
<td>26.31</td>
<td>0.74 (0.46–1.20)</td>
</tr>
<tr>
<td>SAH</td>
<td>7.69</td>
<td>10.32</td>
<td>7.28</td>
<td>4.41</td>
<td>11.29</td>
<td>0.66 (0.30–1.44)</td>
</tr>
<tr>
<td>UND</td>
<td>14.01</td>
<td>13.04</td>
<td>12.08</td>
<td>14.61</td>
<td>6.46</td>
<td>0.46 (0.23–0.93)</td>
</tr>
<tr>
<td>White ethnic group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic stroke</td>
<td>109.69</td>
<td>77.89</td>
<td>115.87</td>
<td>75.95</td>
<td>91.20</td>
<td>0.83 (0.66–1.05)</td>
</tr>
<tr>
<td>PICH</td>
<td>16.07</td>
<td>11.89</td>
<td>26.42</td>
<td>10.42</td>
<td>21.75</td>
<td>0.70 (0.37–1.33)</td>
</tr>
<tr>
<td>SAH</td>
<td>7.01</td>
<td>11.02</td>
<td>6.23</td>
<td>3.47</td>
<td>6.78</td>
<td>0.76 (0.30–1.38)</td>
</tr>
<tr>
<td>UND</td>
<td>14.97</td>
<td>12.68</td>
<td>11.77</td>
<td>15.14</td>
<td>6.34</td>
<td>0.28 (0.11–0.70)</td>
</tr>
<tr>
<td>Black ethnic group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic stroke</td>
<td>131.57</td>
<td>121.45</td>
<td>147.93</td>
<td>110.08</td>
<td>122.00</td>
<td>0.86 (0.52–1.43)</td>
</tr>
<tr>
<td>PICH</td>
<td>33.37</td>
<td>25.71</td>
<td>49.59</td>
<td>10.15</td>
<td>27.87</td>
<td>0.86 (0.37–1.99)</td>
</tr>
<tr>
<td>SAH</td>
<td>14.34</td>
<td>15.83</td>
<td>13.27</td>
<td>1.96</td>
<td>6.66</td>
<td>0.12 (0.02–0.59)</td>
</tr>
<tr>
<td>UND</td>
<td>6.31</td>
<td>15.19</td>
<td>9.88</td>
<td>9.63</td>
<td>12.63</td>
<td>1.52 (0.27–8.74)</td>
</tr>
</tbody>
</table>


![Figure](http://stroke.ahajournals.org/) Ten-year time trends in IRR and corresponding 95% CI of total stroke incidence between blacks and whites (whites served as reference group).
time trends in prior-to-stroke risk factors based on mainly white source populations, observing a decrease in prior-to-stroke hypertension and smoking\(^7\) and a trend toward an increase in diabetes mellitus.\(^6\) No data are available on time trends in prior-to-stroke risk factors in black populations. In contrast to the white population, levels of the main modifiable prior-to-stroke risk factors remained stable in blacks, except a borderline statistically significant decrease in the proportion of hypertension, mainly in black women. Hypertension is the main risk factor for different pathological stroke subtypes,\(^18\) and the association between elevated blood pressure and risk of coronary heart disease might be higher in black women.\(^19\) Thus, the statistically significant decline in stroke incidence observed in female blacks might be mainly attributed to the decrease in hypertension.

A higher incidence of total stroke was observed in blacks compared to whites, comparable to previous studies from the United States.\(^9,20,21\) The higher risk of blacks compared to Whites remained stable until 1999 to 2000, in accordance with the results of the GCNKSS reporting no substantial changes in ethnic disparities in stroke incidence between 1993 to 1994 and 1999.\(^9\) However, the black–white gap in stroke incidence was reducing since then. The observed ethnic group variance in stroke risk might be caused by differences in prevalence of main stroke risk factors.\(^22,23\) Overall, the prevalence of vascular risk factors was higher in black compared to white patients, except for atrial fibrillation. This finding is in agreement with the Behavioral Risk Factor Surveillance System (BRFSS) Survey, which identified substantial ethnic disparities in risk factors for heart disease and stroke in a representative sample of the U.S. civilian population aged 18 years or above in 2003.\(^24\) Similar to our results, the prevalence of reported multiple risk factors for heart disease and stroke among adults was higher among blacks compared to whites.\(^24\) Some of the observed differences in prevalence of risk factors prestroke might also be attributed to ethnic group differences in vascular biological processes, eg, black–white interactions in vascular reactivity.\(^25\)

Increased risk of stroke in blacks compared to whites might also represent a more nonspecific migration effect. Stroke incidence seems to be generally increased in ethnic minority populations, for example in Hispanics in the United States\(^21,26\) or in Maori/Pacific and Asian/other people in New Zealand.\(^27\) A previous comparison of stroke incidence in BC between Barbados and South London revealed that immigrant BC in the UK had an increased risk of total stroke compared to BC in their country of origin.\(^28\) A lower SES of migrants compared to natives might cause this “migration effect.” In the cross sectional Third National Health and Nutrition Survey (NHANES III) from the United States, black ethnicity was independently associated with risk of self-reported stroke; however, after adjustment for income as a proxy for SES, the increased risk in blacks was no longer significant.\(^29\) In the ARIC study, African-Americans had a higher prevalence of vascular risk factors corresponding to a 65% age- and sex-adjusted increased risk for cardiovascular disease (stroke and coronary heart disease combined).\(^30\) However, after adjustment for differences in vascular risk factors and educational level as proxy for SES, ethnicity was not longer an independent risk factor in this study.\(^30\) In addition, there

might be ethnic disparities in access to medical care and preventive treatment for ethnic minorities compared to indigenous groups.\(^31\) Because of the impact of ethnic group disparities on the health of the populations, it is claimed that the rate of disparities should emerge as a key measure of the quality for health care.\(^32\) However, more research is required identifying reasons for ethnic group disparities.\(^33\)

The validity of studies monitoring time trends in stroke incidence depends on stable case ascertainment methods.\(^3\) Detection methods were stable in the SLSR during the 10-year period, and standardized criteria for ensuring completeness of patient capturing were applied.\(^31\) Completeness of case ascertainment was estimated using indirect methods\(^33\) revealing a potential underdetection ranging from 25% to 16%. We used a capture-recapture model with a prespecified set of covariates; this contrasts the previously published model\(^13\) in which a stepwise choice of covariates was made. Stepwise choices can lead to models of differing complexity depending on the number of sources of notification that are used. In addition, models which base the choice of covariates on significant correlations fit the data better but tend not to be stable when new data accrue. However, the estimated completeness for 1995 to 1996 was similar in both models (84% with prespecified covariates and 88% with stepwise choice of covariates). The OXVASC study, based on persons registered in 9 family practices in Oxfordshire, suggested supplementary direct methods for assessing completeness of case ascertainment, including searches of anonymous primary care electronic patient records of the whole study population and a follow-up of a high risk subset of the study population.\(^34\) However, direct methods have not been shown to be superior to indirect estimates as no comparison between both methods has yet been performed. In addition, direct methods might be more time-consuming and resource-intense than indirect methods and, therefore, especially suitable for specific settings, as small study populations or studies with unrestricted access to electronic patient record systems.

Our study has strengths and limitations. Our source population in South London is a multiethnic population, enabling us to study changes in stroke incidence between different ethnic groups. However, the structure of the source population is rapidly changing over time, and some of the SLSR findings might be influenced by this phenomenon. Our definition of ethnic origin based on a valid variable, the self-definition of the 1991 Census question. However, we had no information on family history of migration in the current study, and risk factors might vary between first and second generation migrants. We estimated the completeness of our case ascertainment using indirect methods. However, estimates were provided only for total completeness; therefore, we could not exclude that completeness varied in specific subgroups. We had no information on delay from stroke onset to first brain imaging. Therefore, we cannot exclude that some of the observed differences in pathological stroke subtypes between black and white stroke patients might be attributed to differences in time to first CT or MRI scan.

The decreasing stroke incidence over a 10-year time period in whites can be linked with a decrease in most of the main risk factors in the population. However, the observed increase
in diabetes mellitus in whites, although only borderline statistically significant, might outweigh some achievements and needs further attention. Overall higher attack rates were found in blacks, although the black–white gap in stroke incidence was reducing slightly over time. More research is needed for a better understanding of reasons for black–white disparities, especially for the failing of transferring advances in risk factor reduction in whites to the black population.

Acknowledgments
We thank all the patients and their families and the health care professionals involved. Particular thanks to all the fieldworkers and the whole team who have collected data since 1995 for the South London Stroke Register.

Sources of Funding
The study was funded by the Northern & Yorkshire NHS R&D Programme in Cardiovascular Disease and Stroke, Guy’s and St Thomas’ Hospital Charity, Stanley Thomas Johnson Foundation, Department of Health, UK. The authors acknowledge financial support from the Department of Health via the National Institute for Health Research (NIHR) Biomedical Research Centre award to Guy’s & St Thomas’ NHS Foundation Trust in partnership with King’s College London.

Disclosures
A.P.G. has consultancy agreements with Pfizer Global R&D, Takeda Global R&D (Europe), Schwarz Biosciences, Solace Pharmaceuticals, Cytel, Novartis, and Organon. The remaining authors report no conflicts.

References
Peter U. Heuschmann, Andy P. Grieve, Andre Michael Toschke, Anthony G. Rudd and Charles D.A. Wolfe

Stroke. 2008;39:2204-2210; originally published online June 5, 2008; doi: 10.1161/STROKEAHA.107.507285

The online version of this article, along with updated information and services, is located on the World Wide Web at: http://stroke.ahajournals.org/content/39/8/2204

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

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

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