Outdoor Air Pollution and Incidence of Ischemic and Hemorrhagic Stroke
A Small-Area Level Ecological Study

Ravi Maheswaran, MD; Tim Pearson, MSc; Nigel C. Smeeton, MSc; Sean D. Beevers, PhD; Michael J. Campbell, PhD; Charles D. Wolfe, MD

Background and Purpose—Evidence linking outdoor air pollution and incidence of stroke is limited. We examined effects of outdoor air pollution on the incidence of ischemic and hemorrhagic stroke at the population level focusing on middle-aged and older people.

Methods—We used a small-area level ecological study design and a stroke register set up to capture all incident cases of first-ever stroke occurring in a defined geographical area in south London (948 census output areas) where road traffic contributes to spatial variation in air pollution. Population-weighted averages were calculated for output areas using outdoor nitrogen dioxide and PM$_{10}$ concentrations modeled at a 20-m resolution.

Results—There were 1832 ischemic and 348 hemorrhagic strokes in 1995 to 2004 occurring among a resident population of 267,839. Mean (SD) concentration was 25.1 (1.2) μg/m$^3$ (range, 23.3–36.4 μg/m$^3$) for PM$_{10}$ and 41.4 (3.0) μg/m$^3$ (range, 35.4–68.0 μg/m$^3$) for nitrogen dioxide. For ischemic stroke, adjusted rate ratios per 10-μg/m$^3$ increase, for all ages, 40 to 64 and 65 to 79 years, respectively, were 1.22 (0.77–1.93), 1.12 (0.55–2.28), and 1.86 (1.10–3.13) for PM$_{10}$ and 1.11 (0.93–1.32), 1.13 (0.86–1.50), and 1.23 (0.99–1.53) for nitrogen dioxide. For hemorrhagic stroke, the corresponding rate ratios were 0.52 (0.20–1.37), 0.78 (0.17–3.51), and 0.51 (0.12–2.22) for PM$_{10}$ and 0.86 (0.60–1.24), 1.12 (0.66–1.90), and 0.78 (0.44–1.39) for nitrogen dioxide.

Conclusions—Although there was no significant association between outdoor air pollutants and ischemic stroke incidence for all ages combined, there was a suggestion of increased risk among people aged 65 to 79 years. There was no evidence of increased incidence in hemorrhagic stroke. (Stroke. 2012;43:22-27.)

Key Words: air pollution ■ incidence ■ stroke

Stroke is a major cause of mortality and morbidity worldwide. It accounts for 9% of all deaths and is the sixth largest cause of reduced disability-adjusted life-years. Several studies have examined the acute effects of outdoor air pollution on stroke using daily time series and case–crossover study designs. Although a number of these studies have examined mortality and others have used hospital admissions as a proxy for incident cases, a few have used population case registers, which aim to capture all incident cases. A number of these acute effects studies have reported associations between outdoor air pollutants and ischemic stroke, whereas others have also reported associations with hemorrhagic stroke.

In contrast, although there is an increasing number of studies examining the effects of chronic exposure to outdoor air pollution on cardiovascular mortality using cohort study designs, few have specifically examined stroke mortality. Studies examining mortality, however, are unable to distinguish between the effects of an exposure on incidence of a disease and the effects of the exposure on survival after development of the disease and we have recently found that outdoor air pollution has a substantial adverse effect on survival after stroke. To date, only 1 cohort study has examined the effects of outdoor air pollution on incidence of stroke and an increase in incidence associated with increasing levels of particulate matter <2.5 μm in diameter (PM$_{2.5}$) was observed. However, the effects on stroke subtypes were not examined.

Although cohort studies may be the design of choice for examining incidence of disease, they can be very costly to carry out. A useful alternative is the small-area level ecological study design. Small-area level studies overcome a number of the recognized limitations of traditional ecological studies because populations tend to be more homogenous in...
terms of their sociodemographic characteristics and exposures in smaller geographical areas. In addition, small-area level studies are able to capture fine-grain spatial variation in ecological exposure levels. This is particularly relevant when examining outdoor air pollution effects because road traffic is a major contributor to outdoor pollution levels and traffic-related pollution levels vary substantially within short distances from roads. We have previously used small-area level studies to examine the associations between air pollution exposure and stroke admissions and mortality.\textsuperscript{15,16}

In this article, we report the results of a small-area level ecological study we carried out to examine the effects of outdoor air pollution on stroke incidence at the population level.\textsuperscript{17} We used data from the South London Stoke Register, which was set up to capture all incident cases of first-ever stroke occurring among the resident population in a defined geographical area.\textsuperscript{18} We examined the effects on ischemic and hemorrhagic stroke separately. We also examined the effects in middle-aged and older people separately because previous studies suggest that older people are more susceptible to the adverse effects of air pollution, and we examined the effects on fatal and nonfatal stroke because there is a suggestion that the effect is stronger for fatal stroke.\textsuperscript{14,19}

\textbf{Methods}

We used census output areas as the units of analysis. The census output area is the smallest geographical unit at which population counts by 5-year age band and sex from the 2001 UK census are available. The Stroke Register started in 1995 and used multiple active surveillance methods. Hospital and community notification sources included accident and emergency records, hospital staff, brain imaging requests, death certificates, coroners’ records, general practitioners, community nurses and therapists, bereavement officers, social services, hospital-based stroke registries, general practice computer records, and notification by patients or relatives. Complete data capture varied from 80% to 88%.\textsuperscript{20,21} The Stroke Register area boundary expanded 2-fold in November 2004. Because of the change in boundaries, we restricted the study area for our analysis to census output areas whose constituent postcode centroids were all within the study area throughout the study period from 1995 to 2004. The study had approval from the ethics committee of Guy’s and St Thomas’ Hospital Trust, King’s College Hospital.

\textbf{Exposure Assessment}

We used modeled PM\textsubscript{10} and nitrogen dioxide (NO\textsubscript{2}) concentrations as measures of exposure to outdoor air pollution. Previous research has shown that when PM\textsubscript{10} or NO\textsubscript{2} was controlled for, the effects of other pollutants such as carbon monoxide, sulfur dioxide, and ozone became mostly nonsignificant.\textsuperscript{9} The modeled data had been produced for Greater London and were available at a 20 m\texttimes20-m grid point resolution for 2002.\textsuperscript{22} The model took into account a range of pollution sources and emissions, including large and small regulated industrial processes, boiler plants, domestic and commercial combustion sources, agriculture, rail, ships, airports, and pollution carried into the area by prevailing winds. We have previously described validation of the model against measured outdoor annual air pollution values for 2002.\textsuperscript{13} Correlations were $r=0.90$ for PM\textsubscript{10} and $r=0.91$ for NO\textsubscript{2}.

To derive exposure estimates for output areas, we used a weighted average procedure. First, we assigned to all residential postcode centroids the pollution concentration at the grid point nearest to the centroid with average values taken for equidistant points. We then calculated the average value for all the postcode centroids contained within a census output area weighting the average by the 2001 census total population count for each postcode. There were on average 5 postcodes per output area in the study.

We used the Income Domain of the Index of Multiple Deprivation as a measure of socioeconomic deprivation at the small-area level.\textsuperscript{23} This is a standard index used by government agencies in England and Wales and was available at the lower superoutput area level. Each lower superoutput area contained approximately 5 output areas in our study region and we assigned the lower superoutput area score to all output areas within the lower superoutput area.

\textbf{Statistical Analysis}

We calculated the expected number of strokes for each output area, standardizing for age (using 5-year age bands) and sex. We used Poisson regression for the analysis. Air pollutant concentrations and the Index of Multiple Deprivation income scores were entered as continuous variables with the logarithm of expected counts as the offset. We found no evidence of spatial autocorrelation in stroke incidence using Winbugs\textsuperscript{24} and therefore proceeded to carry out the analyses using Poisson regression within Stata Version 9 using robust SEs to take account of any overdispersion.

We examined ischemic and hemorrhagic strokes separately as well as all strokes combined for completeness. We chose a priori to focus on the 40- to 64- and 65- to 79-year age bands because there is evidence of underenumeration of younger and very old people in UK censuses, especially in the London area.\textsuperscript{25} We defined fatal stroke as death occurring within 2 days of onset of stroke. We felt that using longer periods to define fatal stroke would result in attributing adverse effects of air pollution on survival after stroke, which we found previously, to effects on incidence of stroke.\textsuperscript{13} The effects of PM\textsubscript{10} and NO\textsubscript{2} were examined in separate analyses. Results are expressed as rate ratios with 95% CIs.

\textbf{Results}

A summary of the data used in the study is shown in Table 1. There were 2610 incident, first-ever stroke cases in 1995 to 2004 in the study area occurring among a population of 267 839 giving a crude incidence rate of 97 per 100 000 person-years. Of these, 1832 were ischemic strokes and 348 were hemorrhagic strokes (primary intracerebral hemorrhage). The remainder was caused by subarachnoid hemorrhage or was unclassified. There were 948 output areas in the study area giving an average population of 283 people per output area. The mean (SD) PM\textsubscript{10} concentration in the study output areas was 25.1 (1.2) $\mu$g/m\textsuperscript{3} with a range of 23.3 to 36.4 $\mu$g/m\textsuperscript{3}. The mean (SD) concentration for NO\textsubscript{2} was 41.4 (3.0) $\mu$g/m\textsuperscript{3} with a range of 35.4 to 68.0 $\mu$g/m\textsuperscript{3} (1 part per billion, 1.91 $\mu$g/m\textsuperscript{3}).

The distribution of pollutants across output areas in the study area is shown in the Figure. Pollutant concentrations tended to be higher in areas with a denser network of roads, reflecting the fact that the spatial distribution of both pollutants in the study area was influenced by traffic related pollution.

Table 2 shows incidence rate ratios (relative risks) for ischemic and hemorrhagic stroke before and after additional adjustment for area-level socioeconomic deprivation. Adjustment for deprivation made little difference to the rate ratios. There were no significant correlations between pollutants and deprivation ($r=-0.05, P=0.1$ for PM\textsubscript{10}; $r=-0.01, P=0.74$ for NO\textsubscript{2}).

With regard to PM\textsubscript{10}, the adjusted rate ratio associated with a 10-$\mu$g/m\textsuperscript{3} increase in PM\textsubscript{10} concentration was 1.22 (0.77–
(1.93) for ischemic stroke for all ages combined. In the age bands less likely to be influenced by inaccuracies in denominator population counts, the rate ratios were 1.12 (0.55–2.28) in the 45- to 64-year age band and a statistically significant 1.86 (1.10–3.13) in the 65- to 79-year age band. However, the age-by-pollutant interaction comparing these 2 age bands was nonsignificant (P<0.27). There was no evidence of association in the 80+ year age band in which the rate ratio was 0.50 (0.19–1.32). For hemorrhagic stroke, there appeared to be no statistically significant associations with a rate ratio of 0.52 (0.20–1.37) for all ages combined and rate ratios of 0.78 (0.17–3.51) and 0.51 (0.12–2.22) in the 40- to 64- and 65- to 79-year age bands, respectively.

With regard to NO2, the rate ratio for ischemic stroke associated with a 10-μg/m³ increase in NO2 concentration was 1.11 (0.93–1.32) for all ages combined. It was 1.13 (0.86–1.50) in the 40- to 64-year age band but of borderline statistical significance at 1.23 (0.99–1.53) in the 65- to 79-year age band. However, there was no evidence of association in the 80+ year age band in which the rate ratio was 0.83 (0.58–1.20). For hemorrhagic stroke, there were no statistically significant associations with a rate ratio of 0.86 (0.60–1.24) for all ages combined and rate ratios of 1.12 (0.66–1.90) and 0.78 (0.44–1.39) in the 40- to 64- and 65- to 79-year age bands, respectively.

With regard to fatal stroke (deaths occurring <2 days of stroke onset), there were 41 of 1832 (2.2%) for ischemic stroke, 64 of 348 (18.4%) for hemorrhagic stroke, and 179 of 2610 (6.9%) for all strokes combined. Table 3 shows rate ratios associated with 10-μg/m³ increases in PM10 and NO2 concentrations for fatal and nonfatal stroke. No significant associations were observed.

Weighted pollution concentrations were calculated using total population counts at postcode level as the weights because counts were not available by age. However, there were no significant correlations between the percentage of the population aged <40 years and PM10 (r=0.003, P=0.94) and NO2 (r=−0.03, P=0.34) concentrations at the output area level.

**Discussion**

With regard to the incidence of ischemic stroke, although overall there was no significant evidence of association with outdoor air pollution concentrations, we observed an incon-
Table 2. Stroke Incidence Rate Ratios (95% CIs) Before and After Adjustment for Socioeconomic Deprivation, Associated With a 10-μg/m³ Increase in Outdoor PM₁₀ and NO₂ Concentrations, South London Stroke Register Study Area, 1995–2004

<table>
<thead>
<tr>
<th>Stroke Subtype</th>
<th>Before Adjustment for Deprivation</th>
<th>After Adjustment for Deprivation</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM₁₀ Ischemic stroke, age, y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;40</td>
<td>3.01 (0.45–20.11)</td>
<td>3.95 (0.63–24.76)</td>
</tr>
<tr>
<td>40–64</td>
<td>1.03 (0.51–2.08)</td>
<td>1.12 (0.55–2.28)</td>
</tr>
<tr>
<td>65–79</td>
<td>1.73 (1.02–2.93)</td>
<td>1.86 (1.10–3.13)</td>
</tr>
<tr>
<td>≥80</td>
<td>0.52 (0.20–1.35)</td>
<td>0.50 (0.19–1.32)</td>
</tr>
<tr>
<td>All ages</td>
<td>1.14 (0.72–1.81)</td>
<td>1.22 (0.77–1.93)</td>
</tr>
<tr>
<td>Hemorrhagic stroke, age, y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;40</td>
<td>0.01 (0.00–2.72)</td>
<td>0.01 (0.00–2.66)</td>
</tr>
<tr>
<td>40–64</td>
<td>0.69 (0.16–3.00)</td>
<td>0.78 (0.17–3.51)</td>
</tr>
<tr>
<td>65–79</td>
<td>0.43 (0.10–1.84)</td>
<td>0.51 (0.12–2.22)</td>
</tr>
<tr>
<td>≥80</td>
<td>0.24 (0.03–2.04)</td>
<td>0.35 (0.04–3.31)</td>
</tr>
<tr>
<td>All ages</td>
<td>0.44 (0.17–1.13)</td>
<td>0.52 (0.20–1.37)</td>
</tr>
<tr>
<td>All strokes, age, y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;40</td>
<td>0.51 (0.09–2.96)</td>
<td>0.60 (0.10–3.60)</td>
</tr>
<tr>
<td>40–64</td>
<td>1.08 (0.61–1.94)</td>
<td>1.20 (0.66–2.19)</td>
</tr>
<tr>
<td>65–79</td>
<td>1.40 (0.86–2.26)</td>
<td>1.52 (0.95–2.46)</td>
</tr>
<tr>
<td>≥80</td>
<td>0.51 (0.20–1.27)</td>
<td>0.52 (0.20–1.32)</td>
</tr>
<tr>
<td>All ages</td>
<td>1.00 (0.66–1.50)</td>
<td>1.09 (0.72–1.65)</td>
</tr>
<tr>
<td>NO₂ Ischemic stroke, age, y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;40</td>
<td>1.58 (0.72–3.48)</td>
<td>1.73 (0.77–3.88)</td>
</tr>
<tr>
<td>40–64</td>
<td>1.12 (0.85–1.46)</td>
<td>1.13 (0.86–1.50)</td>
</tr>
<tr>
<td>65–79</td>
<td>1.21 (0.97–1.50)</td>
<td>1.23 (0.99–1.53)</td>
</tr>
<tr>
<td>≥80</td>
<td>0.84 (0.59–1.20)</td>
<td>0.83 (0.58–1.20)</td>
</tr>
<tr>
<td>All ages</td>
<td>1.09 (0.91–1.30)</td>
<td>1.11 (0.93–1.32)</td>
</tr>
<tr>
<td>Hemorrhagic stroke, age, y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;40</td>
<td>0.30 (0.05–1.71)</td>
<td>0.31 (0.05–1.72)</td>
</tr>
<tr>
<td>40–64</td>
<td>1.09 (0.65–1.81)</td>
<td>1.12 (0.66–1.90)</td>
</tr>
<tr>
<td>65–79</td>
<td>0.75 (0.43–1.31)</td>
<td>0.78 (0.44–1.39)</td>
</tr>
<tr>
<td>≥80</td>
<td>0.62 (0.29–1.32)</td>
<td>0.69 (0.31–1.54)</td>
</tr>
<tr>
<td>All ages</td>
<td>0.83 (0.58–1.17)</td>
<td>0.86 (0.60–1.24)</td>
</tr>
<tr>
<td>All strokes, age, y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;40</td>
<td>0.88 (0.49–1.58)</td>
<td>0.91 (0.49–1.68)</td>
</tr>
<tr>
<td>40–64</td>
<td>1.16 (0.92–1.45)</td>
<td>1.18 (0.94–1.50)</td>
</tr>
<tr>
<td>65–79</td>
<td>1.12 (0.92–1.37)</td>
<td>1.15 (0.94–1.40)</td>
</tr>
<tr>
<td>≥80</td>
<td>0.82 (0.59–1.15)</td>
<td>0.83 (0.59–1.17)</td>
</tr>
<tr>
<td>All ages</td>
<td>1.05 (0.89–1.23)</td>
<td>1.07 (0.91–1.26)</td>
</tr>
</tbody>
</table>

All rate ratios adjusted for age (5-year bands) and sex. NO₂ indicates nitrogen dioxide; CI, confidence interval.

Table 3. Stroke Incidence Rate Ratios (95% CIs) for Fatal and Nonfatal Stroke Associated With 10-μg/m³ Increases in Outdoor PM₁₀ and NO₂ Concentrations, South London Stroke Register Study Area, 1995–2004

<table>
<thead>
<tr>
<th>Stroke Subtype</th>
<th>Rate Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM₁₀ Ischemic stroke</td>
<td>1.86 (0.25–13.63)</td>
</tr>
<tr>
<td>Hemorrhagic stroke</td>
<td>0.05 (0.00–1.18)</td>
</tr>
<tr>
<td>All strokes</td>
<td>0.66 (0.18–2.48)</td>
</tr>
<tr>
<td>NO₂ Ischemic stroke</td>
<td>1.17 (0.51–2.70)</td>
</tr>
<tr>
<td>Hemorrhagic stroke</td>
<td>0.47 (0.16–1.35)</td>
</tr>
<tr>
<td>All strokes</td>
<td>0.93 (0.57–1.50)</td>
</tr>
</tbody>
</table>

Fatal defined as dead within 2 d of stroke onset. NO₂ indicates nitrogen dioxide; CI, confidence interval.

Persistent association varying by age with a statistically significant rate ratio of 1.86 (1.10–3.13) per 10-μg/m³ increase in PM₁₀ concentration and a borderline significant rate ratio of 1.23 (0.99–1.53) per 10-μg/m³ increase in NO₂ concentration in the 65- to 79-year age band. However, in the 80+ year age band, there was no statistical evidence of association, but the estimates in this age band may have been affected by inaccuracies in the census denominator population counts at the small-area level. We found no evidence of higher incidence in hemorrhagic stroke associated with living in more polluted areas. We also found no evidence that air pollution was more likely to be associated with fatal rather than nonfatal stroke. Adjustment for socioeconomic deprivation at the small-area level made little difference to the associations observed.

Miller et al found a relative risk of 2.08 (1.25–3.48) for stroke incidence associated with a 10-μg/m³ increase in PM₂.₅ concentration when they examined within-city variations in air pollution concentrations using a cohort study design. Although they did not differentiate between ischemic and hemorrhagic stroke, it is likely that the majority of their cases were of ischemic origin. However, it should be noted that 2 cohort studies examining stroke mortality and a case–control study examining hospital admissions for ischemic stroke did not find any significant associations with outdoor air pollution concentrations. A further cohort study examining stroke mortality found no evidence of association with outdoor background pollution concentrations but did observe a significant association with road traffic exposure.

In an ecological study carried out in Edmonton, Canada, Johnson et al found no significant associations between a range of air pollutants and ischemic or hemorrhagic stroke after adjustment for area-level deprivation. The geographical areas available for their analysis were relatively large with 1600 to 54 000 households in their areal units in contrast to our study with an average population of 283 people per output area. They had a limited number of pollution monitoring stations available (6 stations for PM₂.₅ and 9 for NO₂) for interpolating exposure estimates to a large geographical area (approximately 900 km²) containing a population of >1
million people, whereas we estimated exposure using a pollution model at a very fine spatial scale (20-m grid resolution). Our incident cases were obtained from a register, which aimed to capture all cases occurring within a defined geographical area, which would have included cases in which death occurred before patients could be admitted and cases that did not attend the hospital, whereas their source was all patients attending emergency departments. With regard to deprivation, the effects of pollutants remained relatively unchanged after adjustment in our study in contrast to the results observed by Johnson et al.28 Possible explanations include the relatively limited range in deprivation across our study area and the lack of correlation between pollutants and deprivation. In addition, we have previously observed a diminishing effect of deprivation on stroke risk with increasing age.29

The ecological correlation study design we used would capture both acute and chronic exposure effects of air pollutants on ischemic stroke incidence and could not distinguish between them.30 This is because concentrations of pollutants, which are high on average, may be due to consistently high daily pollution levels, widely varying daily pollution levels with numerous spikes, or some combination of the two. Potential pathways range from short-term increases in pollutant levels triggering an acute stroke by precipitating thrombus formation through to chronic exposure causing progression of atherosclerosis. Particulate matter-induced injury to lung tissue can trigger an inflammation-related cascade.31 Air pollution exposure has been reported to be associated with increased plasma viscosity and shorter prothrombin time.32,33 Animal studies have shown that chronic exposure to particulates promotes progression of atherosclerosis.34–36 Epidemiological studies also support the link between chronic particulate exposure and atherosclerosis, as indicated by increased carotid artery intima-media thickness and increased coronary artery calcification.37–39

There is some experimental evidence suggesting that exposure to particulate air pollution raises blood pressure.40 There is little to suggest that NO2 itself is a causative agent for thrombosis, but it is generally regarded as a proxy for traffic-related pollution.

The higher risk of ischemic stroke we observed in the 65- to 79-year age group is consistent with previous research on the adverse effects of air pollution on cardiovascular disease. A combined analysis of acute exposure studies carried out in numerous European and North American cities found that higher proportions of older people in study populations were associated with increased PM10 risk estimates.19 These results suggest that older people may be more susceptible to adverse effects of pollution and a number of potential explanations have been proposed, including impaired homeostasis, altered immune response, and concurrent cardiopulmonary diseases.41 Another potential explanation may be that because atheroma formation is a chronic process and older people are less likely to move house frequently, those living in polluted areas are more likely to have been exposed to activated atherosclerotic processes for longer periods of time.

PM10 in our study area came from a number of sources, including external sources. However, spatial variation in the study area was influenced by local sources, of which road traffic was an important contributor as we have shown previously.13 Traffic was also the main contributor to spatial variation in NO2 concentrations within the study area.

A number of potential limitations to our study need to be considered. The association between air pollutants and ischemic stroke was only seen in a subgroup of the population and needs to be interpreted with caution. Because this was an ecological study, the possibility of ecological bias, that is the situation in which the association seen at the area level is different from that which exists at the individual level, cannot be ruled out. However, we used very small geographical units, which might be expected to reduce ecological bias. Exposure misclassification is possible because we only took residential exposure into consideration. We could only adjust for a limited number of potential confounders and the possibility that the association might be explained by other unmeasured confounders cannot be ruled out. The South London Stroke Register was specifically established to examine stroke incidence at a population level and all cases were confirmed by the study team. However, subtype could not be established for a proportion of cases. Case capture was incomplete, potentially introducing further error. Population denominator counts were a further source of error and census underenumeration is known to have been greater among younger people and to a lesser extent among very old people.25 The Office for National Statistics had adjusted population estimates to take into account estimated undercounts. Although these estimates are likely to be reliable at a large-area level such as the whole South London Stroke Register study area, it would be difficult to adjust accurately for spatial variation in undercounts at a very small-area level such as the output area level we used in this analysis. However, examining age groups limited to the 40- to 79-year age category would have minimized the impact of underenumeration. In addition, the limitations might be expected to have affected rates of ischemic and hemorrhagic stroke to similar extents and a strength of our study is the comparative analysis of these stroke subtypes.

In summary, although we found no statistically significant association between outdoor air pollutants and ischemic stroke incidence in our population-based study for all ages combined, there was a suggestion of increased risk among people aged 65 to 79 years. Further studies are needed to examine if ischemic stroke risk associated with outdoor air pollution is more pronounced among older people.

Acknowledgments
This work uses Crow data supplied by Ordnance Survey.

Sources of Funding
We would like to thank the Colt Foundation, which supported this study through a research grant. C.D.W. acknowledges financial support from the Department of Health via the National Institute for Health Research (NIHR) Comprehensive Biomedical Research Centre award to Guy’s and St Thomas’ National Health Service (NHS) Foundation Trust in partnership with King’s College London. The views expressed in this article do not necessarily reflect the views of the funding bodies.
Disclosures

All authors were investigators or collaborators on the following grant: Colt Foundation; amount: \( \geq 10,000 \) (significant). In addition, C.D.W. is an investigator on the following grant: Department of Health through the National Institute for Health Research Comprehensive Biomedical Research Centre award to Guy’s and St Thomas’ National Health Service Foundation Trust in partnership with King’s College London; amount: \( \geq 10,000 \) (significant).

References

Outdoor Air Pollution and Incidence of Ischemic and Hemorrhagic Stroke: A Small-Area Level Ecological Study
Ravi Maheswaran, Tim Pearson, Nigel C. Smeeton, Sean D. Beevers, Michael J. Campbell and Charles D. Wolfe

Stroke. 2012;43:22-27; originally published online October 27, 2011;
doi: 10.1161/STROKEAHA.110.610238

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2011 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

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/43/1/22

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/