Original Contributions

Ecological Analysis of Long-Term Exposure to Ambient Air Pollution and the Incidence of Stroke in Edmonton, Alberta, Canada

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Background and Purpose—Long-term air pollution effects on stroke incidence have not been examined extensively. We investigated the associations between ambient pollution and the incidence of stroke, as well as stroke subtypes, in a northern Canadian city surrounded by energy-sector pollution sources.

Methods—Stroke data from an administrative database from 2003 through 2007 were used to estimate annual incidence rates within small geographic regions within Edmonton, Canada. Air pollution levels for each region were estimated from continuous fixed-site monitoring stations in and around Edmonton. We fit models estimating stroke risk in relation to pollution levels; risks were adjusted for age, sex, income, social deprivation, and other factors.

Results—Between 2003 and 2007, the average 5-year concentration of NO₂ and CO was positively associated with the incidence of stroke, particularly for hemorrhagic and nonhemorrhagic stroke subtypes (NO₂: hemorrhagic stroke relative risk \(1.46; 95\%\) CI, 1.19–1.80; nonhemorrhagic stroke relative risk \(1.36; 95\%\) CI, 1.19–1.56). However, these estimates of risk diminished after controlling for the ecological measures of income and deprivation. Adjustment for ecologically derived indices of smoking, hypertension, and body mass index did not alter the estimates of risk in any meaningful way.

Conclusions—Although long-term NO₂ and CO levels were positively associated with a higher incidence of stroke in the entire study area, the risk estimates were strongly attenuated by household income levels. Further research that incorporates individual-level risk factor data would improve our understanding of the relation of longer-term exposures to ambient air pollution and stroke outcomes. (Stroke. 2010;41:1319-1325.)

Key Words: air pollution ■ risk factors ■ stroke ■ epidemiology ■ ecological study

Research into the effects of ambient air pollution on stroke events is an active field of investigation in environmental epidemiology. Associations between short-term, episodic peaks in air pollution and stroke events have been demonstrated in several studies, although heterogeneity of the effect across pollutants has been noted.¹ It has been suggested that the exposure window used in the analyses of short-term effects of pollution on cardiovascular outcomes may reflect the triggering of stroke but may not adequately capture relevant pathophysiologic processes that precede the onset of stroke.⁴ In contrast, the cumulative effects of chronic exposure to air pollution, even at subacute levels, measured in long-term studies may provide information about risk factors for the earlier and middle stages of the disease process. Moreover, these acute and chronic exposures may interact with important risk factors associated with stroke. Hypothetical models of long-term effects from exposure to ambient pollution may involve the promotion of the gradual development of atherosclerosis and subsequent plaque rupture, thrombosis, and emboli by initiating oxidative stress or an inflammatory response.⁴ Long-term exposure to particulate matter up to 2.5 \(\mu\)m in diameter (PM_{2.5}) among a cohort of healthy adults has been shown to increase carotid artery intima-media thickness, which is an indicator of subclinical atherosclerosis.⁵

One UK study considered the effects of longer-term measures of air pollution on stroke within small geographic areas.⁶ From data on emergency hospital admissions for stroke from a 5-year period, a land-use regression model to estimate ambient pollution levels, and survey data to estimate the prevalence of cigarette smoking, investigators found that nitrogen oxides and CO were associated with stroke; however, the association was strongly reduced once adjustments were made for social deprivation and smoking.

In this article, we examine the associations between annual average estimates of ambient air pollution and stroke inci-


dence within small geographic areas within the city of Edmonton, Alberta, Canada. This city is flanked by intensive gas and oil processing regions to its northeast and by coal processing 50 km to the southwest. Additionally, Edmonton meteorology is characterized by shallow atmospheric mixing height regimes that exist throughout the year, which limit the extent of vertical dispersion of air pollutants. The resulting thermal inversion prevents the release of pollutants from the troposphere. Unlike previously published intrarural analyses, smoothed estimates for ambient air pollutants based on monitoring data were generated for geographic areas in this study, associations were evaluated across distinct subtypes of stroke, and a dose-response model was assessed.

Methods

Study Area and Air Pollution Data

The metropolitan area of Edmonton had a population of 1 034 945 in 2006 and a land area that was ~900 km². For the purposes of this study, the metropolitan area of Edmonton was partitioned into 41 geographic units, delineated by the forward sortation areas (FSA). FSAs consist of the first 3 characters of a 6-character Canadian postal code, and the average number of households across the 41 FSAs included in the study ranged from 1600 to 54 000. Data used for air pollution estimates were collected at spatially independent continuous-monitoring stations within and around the study area, including 3 stations within the city of Edmonton. NO₂ (parts per billion [ppb]) was monitored at 9 stations, SO₂ (ppb) at 7, O₃ (ppb) at 6, PM₂.₅ (µg/m³) at 6, and CO (ppm) at 5. Hourly concentrations for these stations were obtained from the Alberta Ambient Air Data Management System data warehouse, which included data from Environment Canada’s National Air Pollution Surveillance network of fixed site monitoring stations. Using the daily averages from January 1, 2003, through December 31, 2007, we calculated air pollution exposure estimates for each FSA in a geographic information system (ArcView 9.0, ESRI, Redlands, Calif) as the inverse distance-weighted average at each FSA centroid. The inverse distance-weighted average is a function of the inverse of the square of distance to each monitoring station. For each FSA, long-term pollutant exposures during the study period were estimated by using the 5-year mean of annual median concentrations.

Data on total road length by road type and total area of FSAs were obtained from the CanMap Streetfiles 2005 (Desktop Mapping Technologies Inc, Markham, Ontario). Traffic density within each FSA was estimated as the sum of weighted road lengths for each road category. Traffic density was estimated by using the 5-year mean of annual median concentrations. The resulting index was a composite measure including individual pollutants, traffic density, SES variables, and stroke risk factors for the 41 FSAs. Poisson regression models were used to estimate risk ratios and 95% CIs, comparing the stroke incidence in FSAs with higher quintiles of pollution to those with the lowest quintile, while adjusting for effects due to age and sex. Deviation was used as the scale parameter to correct the standard error of the risk estimates for possible overdispersion. In addition, we adjusted our risk ratios to take into account the potential confounding effects of social deprivation, income, smoking, hypertension, overweight, and inactivity by adding each separately to models adjusted for age and sex. To test for linear dose-response effects, Poisson charts. Strokes were defined as those admissions with discharge diagnosis between ICD-10 classifications I60 through I68, or G45. Stroke subtypes were designated as follows: hemorrhagic, I60 through I62; nonhemorrhagic, I63 through I68; and transient ischemic attack, G45, excluding G45.4. Diagnostic criteria included confirmation by an internist or neurologist by history and cerebral imaging in the majority of cases. In the Capital Zone, computed tomography is standard procedure for all suspected stroke cases; magnetic resonance imaging or magnetic resonance angiography may also be required for confirmation.

Cases were grouped by residential FSA at the time of first stroke admission and classified by sex and age group (0 to 44, 45 to 64, ≥65 years). The outcome measure was the incidence rate of stroke admissions in each FSA in a 5-year period. The 2006 Canadian Census was used to estimate population denominator data for the age-sex strata. These census data originated at the dissemination, or area-level, and therefore required conversion to an FSA. Assignment of all 6-digit postal codes to dissemination areas in the study area was done in ArcView 9.0 (ESRI, Redlands, Calif). Postal codes were then merged with census data in SAS 9.1 (SAS Inc, Cary, NC), and this composite file was summarized at the 3-digit FSA level. For mapping purposes at the FSA level, annual incidence per 1000 was standardized by age, during 10-year age increments and the total study population age distribution in 2006 as a reference.

Socioeconomic Status and Stroke Risk Factor Data

Data on household income and individual-level data on recognized risk factors for stroke were acquired from the Canadian Community Health Survey 2003, cycle 2.1. This survey was conducted by Statistics Canada from January 2003 through December 2003 according to a multistage, random sampling method. The target population consisted of residents who were 12 years of age and older who lived in private residences within the provinces and territories of Canada. For each FSA, we used Canadian Community Health Survey data to estimate the percentage of residents with a household income that exceeded $40 000 and who were current smokers, hypertensive, overweight, and inactive. Canadian Community Health Survey data are maintained by Statistics Canada. Cycle 2.1 included responses from 2817 participants who resided in the study area and were representative of a population of 828 699.

In addition, we applied the Vancouver Area Neighborhood Deprivation Index model to 2006 Census socioeconomic status (SES) data for the Edmonton area to estimate the percentage of socially deprived individuals within the study FSAs. The Vancouver Area Neighborhood Deprivation Index model is based on 2001 Census and survey data gathered in Vancouver, British Columbia, which, like Edmonton, is a large urban center in western Canada. Details of the Vancouver Area Neighborhood Deprivation Index are presented elsewhere. In brief, the index is a composite measure including 7 SES variables (index weights in parentheses): average personal income (0.089), home ownership (0.089), single-parent family (0.143), no high school completion (0.250), university degree (0.179), employment ratio (0.036), and unemployment rate (0.214). Weights for each variable were derived from a survey among provincial medical officers of health on the relative importance of SES variables as measures of neighborhood deprivation.

Statistical Analysis

Statistical analyses were conducted with SPSS version 17.0 (SPSS Inc, Chicago, Ill). Pearson correlation coefficients were calculated among individual pollutants, traffic density, SES variables, and stroke risk factors for the 41 FSAs. Poisson regression models were used to estimate risk ratios and 95% CIs, comparing the stroke incidence in FSAs with higher quintiles of pollution to those with the lowest quintile, while adjusting for effects due to age and sex. Deviation was used as the scale parameter to correct the standard error of the risk estimates for possible overdispersion. In addition, we adjusted our risk ratios to take into account the potential confounding effects of social deprivation, income, smoking, hypertension, overweight, and inactivity by adding each separately to models adjusted for age and sex. To test for linear dose-response effects, Poisson
regression models were run, replacing quintile-level data with standardized means.

**Results**

**Pollutants**

Across the 41 FSAs, the mean (interquartile range) of 5-year mean pollutant concentrations were as follows: NO₂, 15.7 (2.2) ppb; O₃, 19.6 (1.5) ppb; CO, 0.3 (0.05) ppm; SO₂, 1.3 (0.1) ppb; and PM₉.₅, 5.0 (0.2) μg/m³. A very strong positive correlation was observed between 5-year mean concentrations of NO₂ and CO (ρ=0.95, P<0.001), and very strongly negative correlations existed between O₃ and NO₂ (ρ=−0.98, P<0.001) and between O₃ and CO (ρ=−0.95; P<0.001; Table 1). Although the FSAs with highest quintiles of NO₂ and CO were near the downtown core (Figure 1, Appendix), FSAs with the highest quintile of PM₉.₅ were along the north and east perimeter in newer residential neighborhoods. High SO₂ was reported along the southern border of the study area.

**SES Indicators and Stroke Risk Factors**

At the FSA level, the percentage of individuals with a household income >$40,000 had a strong negative correlation with the percentage of socially deprived individuals (ρ=−0.61, P<0.001) and was negatively correlated with the percentage of current smokers (ρ=−0.43, P=0.005; Table 2). Income was also associated with 5-year mean concentrations for NO₂, CO, O₃, SO₂, and PM₉.₅ (P<0.05). Hypertension was marginally associated with O₃ (ρ=0.31, P=0.05). Current smoking was not associated with any pollutant (P>0.05).

**Risk of All Strokes and Stroke Subtypes**

From 2003 through 2007, there were 7336 first admissions for stroke to Capital Zone EDs for patients who lived in the study area. The age-adjusted incidence of stroke was higher in spatially isolated FSAs within Edmonton, whereas a lower age-adjusted stroke incidence was found in rural FSAs (Figure 2, Appendix). Higher levels of NO₂, CO, and traffic density were associated with an increased incidence of all types of stroke, after adjustment for age and sex (fifth quintile of NO₂, relative risk [RR] =1.29; 95% CI, 1.17 to 1.43; fifth quintile of CO, RR=1.29; 95% CI, 1.16 to 1.43); however, further adjustment for income attenuated these effects (Table 3). After controlling for income, an increased risk of stroke was apparent only when the fourth quintile of traffic density was compared with the lowest quintile. The risks of stroke were relatively unchanged after adjustment for ecological measures of hypertension, smoking, overweight, and inactivity (data not shown; available on request). There was some indication of an inverse association between stroke and high levels of SO₂, even after controlling for income (fifth quintile, RR=0.84; 95% CI, 0.73 to 0.96). When we adjusted our risk estimates for stroke by using the social deprivation term, similar to income, our risk estimates were attenuated, albeit the change was less pronounced (data not shown; available on request). The standardized means of NO₂, CO, and PM₉.₅ were associated with stroke incidence, but similar to the quintile-level analyses, adjustment for income removed any apparent effect. Among all markers for air pollution, traffic density had the only statistically significant dose-response effect after adjustment for income (RR=1.05; 95% CI, 1.00 to 1.10).

Stratified analyses by stroke subtype revealed no associations between the incidence of stroke, and ambient pollution or traffic density in models that were adjusted for age, sex, and household income (Tables 4 through 6). Without adjustment for income, risk ratios for hemorrhagic and nonhemorrhagic stroke associated with NO₂ (hemorrhagic stroke fifth quintile RR=1.46; 95% CI, 1.19 to 1.80; nonhemorrhagic stroke fifth quintile RR=1.36; 95% CI, 1.19 to 1.56) and CO were higher than they were for transient ischemic attack (Tables 4 through 6). The negative association between SO₂ and incidence appeared to be stronger for nonhemorrhagic stroke (fifth quintile RR=0.63; 95% CI, 0.52 to 0.76) than for transient ischemic attack (fifth quintile RR=0.85; 95% CI, 0.70 to 1.03) or hemorrhagic stroke (fifth quintile RR=0.74; 95% CI, 0.54 to 0.99). Finally, the only evidence of a dose response for any stroke type was between traffic density and transient ischemic attack (RR=1.13; 95% CI, 1.05 to 1.21).

**Discussion**

Our results suggest that within small geographic areas, high levels of NO₂, CO, and PM₉.₅ may influence the incidence of all types of stroke; however, after controlling for income and deprivation, these effects are less pronounced. Risk ratios for stroke reported in a previous small-area analysis in the United Kingdom were similarly reduced after controlling for smoking and deprivation. Although the confounding effects of income and deprivation may be notable in our study, interpretation of the adjusted risk estimates should be prefaced with a caution. The reasons for the influence of this variable within the context of air pollution modeling at the ecological level are not entirely clear. Contrary to our results, a time-
Table 3. Risk Ratios for Air Pollution Effects on All Stroke Incidence, Edmonton, Alberta 2003–2007

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Level (Range)</th>
<th>Adjusted for Age and Sex</th>
<th>Adjusted for Age, Sex, and Household Income</th>
</tr>
</thead>
<tbody>
<tr>
<td>NO\textsubscript{2}, ppb</td>
<td>Q1 (10.1–14.0)</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>Q2 (14.0–15.2)</td>
<td>0.99 (0.88, 1.13)</td>
<td>0.95 (0.85, 1.08)</td>
</tr>
<tr>
<td></td>
<td>Q3 (15.2–15.8)</td>
<td>1.03 (0.93, 1.14)</td>
<td>1.03 (0.93, 1.13)</td>
</tr>
<tr>
<td></td>
<td>Q4 (15.8–16.7)</td>
<td>1.16 (1.04, 1.29)</td>
<td>1.06 (0.94, 1.18)</td>
</tr>
<tr>
<td></td>
<td>Q5 (16.7–20.3)</td>
<td>1.29 (1.16, 1.43)</td>
<td>1.10 (0.97, 1.24)</td>
</tr>
<tr>
<td>Standardized mean</td>
<td>1.08 (1.05, 1.12)</td>
<td>1.00 (0.96, 1.05)</td>
<td></td>
</tr>
<tr>
<td>O\textsubscript{3}, ppb</td>
<td>Q1 (16.4–19.0)</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>Q2 (19.0–19.7)</td>
<td>0.91 (0.83, 1.00)</td>
<td>0.98 (0.89, 1.08)</td>
</tr>
<tr>
<td></td>
<td>Q3 (19.7–20.0)</td>
<td>0.75 (0.68, 0.83)</td>
<td>0.88 (0.78, 1.00)</td>
</tr>
<tr>
<td></td>
<td>Q4 (20.0–20.7)</td>
<td>0.84 (0.76, 0.92)</td>
<td>0.95 (0.85, 1.06)</td>
</tr>
<tr>
<td></td>
<td>Q5 (20.7–22.6)</td>
<td>0.78 (0.70, 0.86)</td>
<td>0.91 (0.80, 1.03)</td>
</tr>
<tr>
<td>Standardized mean</td>
<td>0.91 (0.87, 0.94)</td>
<td>0.99 (0.95, 1.04)</td>
<td></td>
</tr>
<tr>
<td>CO, ppm</td>
<td>Q1 (0.26–0.30)</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>Q2 (0.30–0.32)</td>
<td>1.11 (0.99, 1.23)</td>
<td>1.09 (0.98, 1.21)</td>
</tr>
<tr>
<td></td>
<td>Q3 (0.32–0.33)</td>
<td>0.97 (0.88, 1.08)</td>
<td>0.97 (0.88, 1.07)</td>
</tr>
<tr>
<td></td>
<td>Q4 (0.33–0.35)</td>
<td>1.18 (1.05, 1.32)</td>
<td>1.08 (0.96, 1.21)</td>
</tr>
<tr>
<td></td>
<td>Q5 (0.35–0.4)</td>
<td>1.28 (1.16, 1.42)</td>
<td>1.10 (0.97, 1.24)</td>
</tr>
<tr>
<td>Standardized mean</td>
<td>1.13 (1.07, 1.19)</td>
<td>1.00 (0.94, 1.06)</td>
<td></td>
</tr>
<tr>
<td>SO\textsubscript{2}, ppm</td>
<td>Q1 (1.06–1.20)</td>
<td>1.00</td>
<td>1.00</td>
</tr>
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<td></td>
<td>Q2 (1.20–1.28)</td>
<td>0.97 (0.88, 1.07)</td>
<td>0.94 (0.85, 1.03)</td>
</tr>
<tr>
<td></td>
<td>Q3 (1.28–1.35)</td>
<td>0.85 (0.77, 0.94)</td>
<td>0.91 (0.83, 1.00)</td>
</tr>
<tr>
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<td>Q4 (1.35–1.37)</td>
<td>0.84 (0.76, 0.93)</td>
<td>0.89 (0.81, 0.98)</td>
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<td></td>
<td>Q5 (1.37–1.47)</td>
<td>0.72 (0.63, 0.83)</td>
<td>0.84 (0.73, 0.96)</td>
</tr>
<tr>
<td>Standardized mean</td>
<td>0.87 (0.83, 0.92)</td>
<td>0.93 (0.89, 0.98)</td>
<td></td>
</tr>
<tr>
<td>PM\textsubscript{2.5}, (\mu g/\text{m}^3)</td>
<td>Q1 (4.40–4.87)</td>
<td>1.00</td>
<td>1.00</td>
</tr>
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<td></td>
<td>Q2 (4.87–4.90)</td>
<td>0.85 (0.76, 0.94)</td>
<td>0.93 (0.84, 1.02)</td>
</tr>
<tr>
<td></td>
<td>Q3 (4.90–4.94)</td>
<td>1.04 (0.94, 1.15)</td>
<td>1.04 (0.94, 1.14)</td>
</tr>
<tr>
<td></td>
<td>Q4 (4.94–5.01)</td>
<td>0.99 (0.89, 1.10)</td>
<td>0.95 (0.86, 1.04)</td>
</tr>
<tr>
<td></td>
<td>Q5 (5.01–5.09)</td>
<td>0.98 (0.88, 1.10)</td>
<td>0.99 (0.90, 1.10)</td>
</tr>
<tr>
<td>Standardized mean</td>
<td>1.08 (1.02, 1.14)</td>
<td>1.01 (0.95, 1.06)</td>
<td></td>
</tr>
<tr>
<td>Traffic, vehicles/km-d</td>
<td>Q1 (5.2–13.2)</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>Q2 (13.2–26.9)</td>
<td>0.98 (0.86, 1.11)</td>
<td>0.95 (0.84, 1.07)</td>
</tr>
<tr>
<td></td>
<td>Q3 (26.9–43.6)</td>
<td>1.19 (1.06, 1.35)</td>
<td>1.10 (0.98, 1.25)</td>
</tr>
<tr>
<td></td>
<td>Q4 (43.6–53.0)</td>
<td>1.33 (1.19, 1.50)</td>
<td>1.17 (1.03, 1.32)</td>
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<tr>
<td></td>
<td>Q5 (53.0–102.4)</td>
<td>1.11 (0.99, 1.24)</td>
<td>1.06 (0.95, 1.19)</td>
</tr>
<tr>
<td>Standardized mean</td>
<td>1.08 (1.02, 1.14)</td>
<td>1.05 (1.00, 1.10)</td>
<td></td>
</tr>
</tbody>
</table>

Q indicates quintile.

*The standardized mean of a pollutant is the 5-year mean for an FSA divided by the interquartile range over all FSAs in the study area.

A series analysis of cardiac hospitalizations in Canada did not find that citywide income and education levels modified the effect of air pollution levels on hospitalization rates.\(^{14}\) However, income may contribute to the apparent associations between air pollution and stroke incidence at the small-area level over a long period of time. The proportion of repeat strokes in this database was unknown, but if many of the patients in the database were recurrent stroke patients, the influence of income might, in part, explain the confounding relation demonstrated here. Mohan et al\(^{15}\) estimated the cumulative risk of recurrence within 5 years among first-ever stroke survivors to be 16.2%. Those with an annual income <$40,000 might be less likely to be able to maintain compliance with rehabilitation therapy and preventive strategies (eg, proper diet and exercise regimens) and might have been more likely to live in highly polluted areas. Stroke
recurrence is more common among low-income stroke patients in Sweden, particularly among women. Social isolation and need for help at home, which could be associated with income, are predictive of secondary stroke.

Cohort research in the United States has demonstrated that long-term exposure to increased levels of PM$_{2.5}$ is associated with stroke incidence in women, but the upper-quintile concentrations reported were 5 times greater than the levels in Edmonton. Our findings suggest that, at the small-area level, fine particulate-matter pollution at levels between 4.4 and 5.1 µg/m$^3$ may not have a detectable effect on stroke incidence when comparing categories of pollution level. The levels of all pollutants measured in this study are substantially lower than levels reported in studies of the long-term effects of air pollution on cardiovascular/cerebrovascular morbidity and mortality from the United States, although the levels are similar to those reported in the UK study of small-area effects of air pollution on stroke. Furthermore, given that variability in exposures across Edmonton FSAs was not strong, effects were difficult to detect.

Traffic density is considered to be a valid surrogate measure for the assessment of air pollution effects on mortality; it broadly reflects other health-affecting factors, such as stress, noise, and pollutants apart from the typically...
monitored air pollution constituents. The increased stroke incidence associated with traffic density could also be explained by the tendency for populations more prone to stroke and with greater need for access to large health care centers to live in higher traffic areas than in remote areas.

Previous ecological studies of long-term air pollution effects on risk for stroke have not assessed risks for specific stroke subtypes. In contrast to the association with hemorrhagic and nonhemorrhagic stroke in this study, there was a lack of association with transient ischemic attack. Underreporting and delays in reporting are common for transient ischemic attack, and many transient neurologic cases present to non-ED settings, whereas patients with persistent symptoms (eg, stroke) are more likely to present to the ED or be sent there after primary care assessment. If underreporting is more prevalent in remote areas with low NO2 and CO as well as a lack of access to ED care, then the misclassification would have resulted in risk ratios biased away from the null. It is yet unclear whether this misclassification was present in our data.

There is little in the literature to support our finding that increased SO2 was associated with lower nonhemorrhagic stroke incidence. In Edmonton, higher levels of SO2 are found in less central FSAs; thus, the association may be spurious and confounded by the residential choices of a physically active, partly rural population and the need for accessibility to health care among the stroke-prone age groups. As in this study, Wellenius et al found that the effect that SO2 had on ischemic stroke incidence was greater than it was for hemorrhagic stroke. The fact that they observed a significant effect on ischemic strokes whereas we found none may be due to fact that the range of concentration in Edmonton was much lower. Another possible explanation for the difference in SO2 effects on risk of nonhemorrhagic and other subtypes of stroke could be due to a pathway including diabetes, which is a risk factor for nonhemorrhagic strokes but not other subtypes. Analysis of a nationwide Canadian health survey showed that, after adjusting for diabetes risk factors, urban Canadians were more likely to have self-reported diabetes than rural populations. It is possible then that rural populations near southern sections of Edmonton may have decreased risk of diabetes and nonhemorrhagic stroke and also happen to live in an environment with higher SO2 levels.

The reason for the lack of influence on risk estimates by variables that are recognized risk factors for stroke requires further discussion. This may be due in part to the small sample size of the Canadian Community Health Survey and to the lack of association between air pollutants and these variables among the study FSAs. Hypertension, smoking, overweight, and inactivity may predispose individuals to stroke, but the influence that they exert may be unassociated with the effect of air pollution on stroke in the long term and within small-area comparisons.

In our view, there are 4 main limitations of this study. The first is that inferences about individual health effects cannot be made from the results, which are derived from group-level analyses. The results are, however, potentially valuable to the extent to which effects of regional pollution can inform regional land use planning and the development of environmental health policies. Another limitation is that results are based on the assumption that, in the long term, the relative level of one’s personal exposure can be inferred from ambient measurements assigned to the centroid of one’s residential FSA. There were no data available from which location of work for working-age populations could be imputed. Regardless, our estimates should be quite valid given that Canadians, on average, spend over 67% of their time at home.

Also, there were no data available to permit the removal of recent immigrants to the study area from the file. Differential migration within the study area during the 5 years of interest could have biased the results. It is reasonable to assume that current addresses of inner-city populations, compared with suburban or rural populations, were more prone to change in a 5-year period, and this may result in misclassification of long-term exposure and a bias of chronic pollution effects. Related to this limitation is the inability to determine whether stroke events were the first for each patient. The constellation of risk factors for first-ever stroke, including the impact of environmental hazards, is likely different than for secondary stroke. Identification and exclusion of individuals who had their first stroke before 2003 is important because, in the absence of this, due to the possibility of migration, we cannot rule out the possibility that some patients, before their first stroke, were exposed to ambient air pollution that was very different from the level estimated for the FSA in which they lived when they entered the study database.

In summary, our study found modest associations between low levels of ambient air pollution and the incidence of stroke that diminished further on adjustment for sociodemographic characteristics available at a small-area level. Taken together, our findings underscore the critical need to adjust for SES variables when ambient air pollution is modeled on an intrarural scale. A better understanding of the role of long-term exposure to ambient air pollution in the development of stroke is possible through the use of prospective cohort studies that can incorporate individual-level data. Such studies should endeavor to collect detailed information on place of residence at the time of incident strokes, residential mobility, work place exposures, health care support, and other risk factors, such as hypertension and smoking status, that may predispose certain subpopulations to the effects of ambient air pollution.

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**Disclosures**

None.

**References**

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**Figure I.** Estimated NO₂ concentrations in Edmonton, Alberta, 2003–2007. Map represents the surface of inverse-distance weighted averages interpolated from the centroid values.

**Figure II.** Age-standardized stroke incidence in Edmonton, Alberta, from 2003 through 2007, by forward sortation area.
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