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Effect of Motor Vehicle Emissions on Respiratory Health in an Urban Area

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Motor vehicles emit particulate matter < 2.5 μm in diameter ($PM_{2.5}$), and as a result, $PM_{2.5}$ concentrations tend to be elevated near busy streets. Studies of the relationship between motor vehicle emissions and respiratory health are generally limited by difficulties in exposure assessment. We developed a refined exposure model and implemented it using a geographic information system to estimate the average daily census enumeration area (EA) exposure to $PM_{2.5}$. Southeast Toronto, the study area, includes 334 EAs and covers 16 km^2 of urban area. We used hospital admission diagnostic codes from 1990 to 1992 to measure respiratory and genitourinary conditions. We assessed the effect of EA exposure on hospital admissions using a Poisson mixed-effects model and examined the spatial distributions of variables. Exposure to $PM_{2.5}$ has a significant effect on admission rates for a subset of respiratory diagnoses (asthma, bronchitis, chronic obstructive pulmonary disease, pneumonia, upper respiratory tract infection), with a relative risk of 1.24 (95% confidence interval, 1.05–1.45) for a $\log_{10}$ increase in exposure. We noted a weaker effect of exposure on hospitalization for all respiratory conditions, and no effect on hospitalization for nonrespiratory conditions. *Key words*: geographic information system, respiratory health, spatial autocorrelation, vehicle emissions. *Environ Health Perspect* 110:293–300 (2002). [Online 14 February 2002]

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Time-series analyses suggest that chronic exposure to particulate matter < 2.5 μm in diameter ($PM_{2.5}$) has detrimental effects on respiratory health (*1–4*). Motor vehicles emit $PM_{2.5}$ along with a variety of other pollutants (*5,6*), and source apportionment studies in urban areas suggest that motor vehicles contribute from 25% to 35% of direct $PM_{2.5}$ emissions (*7,8*). It is therefore not surprising that $PM_{2.5}$ concentrations near busy roads can be 30% higher than background levels (*9*). However, the relatively higher exposure appears to be limited to an area quite close to streets, falling by approximately half within 10 m of a street (*9–12*). It is likely, therefore, that residence near busy streets results in increased exposure to $PM_{2.5}$ and, consequently, poorer respiratory health. The proportion of respiratory illness attributable to such exposure is potentially large, given the prevalence of the exposure (*13*).

Over the last decade, a number of epidemiologic studies have attempted to examine the relationship between exposure to motor vehicle emissions and respiratory health (*12,14–24*). These studies are methodologically diverse, using case–control, cross-sectional, and ecologic designs. A variety of health end points have been measured, and a wide range of exposure assessment methods employed. Most studies support a relationship between some measure of respiratory health and some type of modeled exposure. However, few studies find an association for all respiratory health measures studied, and exposure assessment generally limits evidence of association. As a proxy for exposure, studies tend to model either traffic volume on the nearest road or distance to the nearest road. In this study, we develop a single-pollutant exposure model that accounts for traffic emissions from all major streets and considers traffic volume, distance to residence, and vehicle type mix. We then implement this model with a geographic information system (GIS) to examine the relationship between exposure to $PM_{2.5}$ from motor vehicle emissions in an urban area and hospital admission rates for respiratory and other conditions.

Materials and Methods

We used an ecologic study design with the census enumeration area (EA) as the unit of analysis. Our aim was to examine the effect of exposure to motor vehicle emissions on respiratory hospitalization while controlling for socioeconomic status (SES). After an overview of the study area, we present detailed methods for measurement of health, assessment of exposure, and measurement of SES.

Southeast Toronto (SETO), the study area, encompasses 16 km^2 of urban area in Canada's largest city (Figure 1). In the 1991 census, SETO had a population of 121,875. The study area was divided into 334 EAs for the census, with a median EA population of 400. SETO borders the urban core of Toronto to the west, Lake Ontario to the south, and mixed commercial/residential areas to the north and east. The population and land use characteristics within SETO are diverse. The land use is predominantly residential, but pockets of commercial and industrial zoning also exist. Neighborhood SES within SETO ranges considerably between the most affluent neighborhood (Rosedale: median family income $123,920, 50.7% with university degree) and the least affluent neighborhood (Regent Park: median family income $18,214, 6.2% with university degree).

Measurement of health. We measured respiratory health from hospital admission diagnostic coding data for SETO residents of all ages who were admitted to a hospital in the Province of Ontario between 1990 and 1992. We calculated 3-year age- and sex-standardized hospitalization rates for a subset of respiratory diagnoses associated with exposure to $PM_{2.5}$ air pollution. As a comparison, we also calculated standardized hospitalization rates separately for all respiratory, and genitourinary admissions (i.e., conditions involving the genital or urinary systems).

We obtained hospital discharge data from the Hospital Medical Records Institute (HMRI) database. Shortly after acquisition of data for this study, HMRI was renamed the Canadian Institute for Health Information (*25*). HMRI collected Canadian hospital admissions data that were manually abstracted from patient charts and coded according to the *International Classification of Diseases*, Ninth Revision (ICD-9) (*26*). These data reflect physician-assigned diagnoses for inpatients, and the estimated agreement with reabstracted records is 95% for the primary diagnosis (*27*). Universal hospital insurance in Canada and complete participation of area hospitals in the HMRI database ensure that these data accurately reflect hospital admissions in the SETO population. Addresses in the HMRI data were acquired from the reporting hospitals, which routinely acquire or update addresses directly from patients at the time of admission. This address information, therefore, has high validity, although there is still the potential for error from sources such as data entry or patients reporting the address of a relative with whom they were staying before admission. The University

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of Toronto Human Subjects Review Committee approved the use of deidentified individual-level human health data for this study.

Using ICD-9 codes, we identified three diagnostic sets: respiratory subset, respiratory chapter, and genitourinary chapter (Table 1). Codes for the respiratory subset identify asthma, bronchitis, chronic obstructive pulmonary disease, pneumonia, and upper respiratory tract infections, all of which have been associated with $PM_{2.5}$ exposure in previous studies (*28–31*). We examined diagnoses other than those in the respiratory subset to assess the specificity of any association between respiratory health and exposure. As an example of nonrespiratory conditions, we selected genitourinary chapter admissions, which we believe are not associated with exposure to motor vehicle emissions. We selected records with a primary diagnostic code in the respiratory or genitourinary chapter over the years 1990–1992 from the HMRI database for the City of Toronto (respiratory subset records are contained within the respiratory chapter records).

The Postal Code Conversion File maintained by Statistics Canada (*32*) allowed matching of hospital admission records with six-digit postal codes to the most representative EA based on address range. We did not manually validate matches, but given manual validation performed by others in a similar context, we estimate the error rate at 3% (*32,33*). We limited matched records to EAs in SETO using EA numbers. Statistics Canada does not release detailed population figures for EAs with response rates ≤ 40% or populations < 40. This affected 32 of the 334 EAs in SETO, and we removed records in these EAs because the missing data precluded calculation of standardized rates. For quality assurance, we removed records without valid birth dates or health numbers. Finally, we limited records to the first hospital admission in the study period for each person in the data set.

For each EA, we calculated 3-year (1990 through 1992) indirectly standardized incident admission rates by diagnostic group. We calculated expected values from the age-sex–specific EA population counts from the 1991 census (*34*), and age-sex–specific admission rates for all of SETO.

Assessment of vehicle emissions and exposure to $PM_{2.5}$. We estimated emissions of $PM_{2.5}$ from traffic volume and vehicle type data for major streets in SETO. We then modeled EA exposures in average daily grams of $PM_{2.5}$ from emissions of $PM_{2.5}$ and EA street frontages using a GIS model that builds on previous work (*15*) and is described in detail elsewhere (*35*). The GIS model transfers emissions from a buffered street network to surrounding areas and estimates exposure for each study unit from the transferred emission value, the length of street frontage, and the proportion of the unit area that is close to a street. We performed geographic data operations with ARC/INFO software (version 7.1; Environmental Systems Research Institute, Redlands, CA), and statistical analyses with SAS software (version 8.00; SAS Corporation, Cary, NC).

Assessment of traffic count and development of street network. We acquired traffic count data from the Traffic Branch of Metropolitan Transportation and from Transportation Operations of the City of Toronto. Twenty-four hour counts were directly available, or could be converted from 8 hr counts, for 104.1 km of the 219.0 km network (47.8%). We converted eight-hour counts using a factor of 2.05 (*36*). These data describe traffic on all major streets between 1990 and 1992 and secondary streets with traffic volume > 5,000 vehicles per day between 1987 and 1994. Traffic counts were georeferenced to a digital Street Network File of Metro Toronto (*37*) by assigning a unique identifier to each network segment and the corresponding traffic count.

Modeling of $PM_{2.5}$ emissions. We obtained data on vehicle type distribution throughout the study area from two sources.

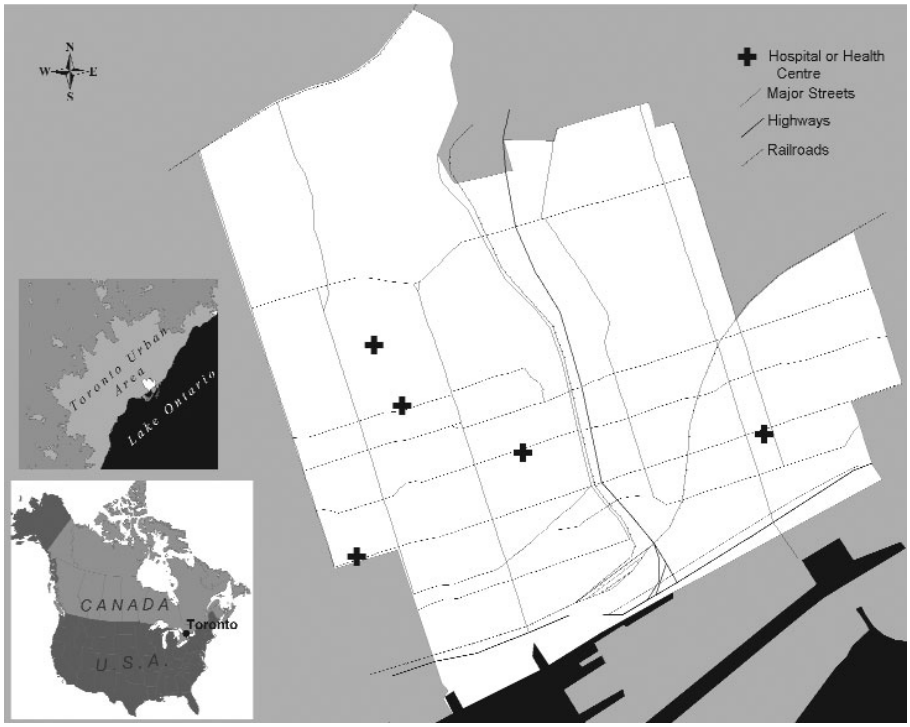

Figure 1. Location and characteristics of southeast Toronto (SETO).

Table 1. Diagnoses and associated ICD-9 codes used to abstract records for study.

Diagnostic set, specific diagnosis	ICD-9 codes	Individuals	Admissions	Repeat admissions (%)
Respiratory subset				
Asthma	493.0-1,493.9	430	642	33
Bronchitis	466.0-1, 490	127	139	9
COPD	491.0-2, 491.8-9, 492, 496	238	411	42
Pneumonia	480.0-2, 480.8-9, 481, 482.0-4, 482.8-9, 483, 485, 486, 514	709	834	15
URI	461.0-3, 461.8-9, 462, 464.4, 465.0,465.8-9, 472.0-2, 473.0-3, 473.8-9, 478.1-3, 478.7-9	275	296	7
Total		1,779	2,322	23
Respiratory chapter	460–519.9	2,646	3,316	20
Genitourinary chapter	580–629.9	2,406	2,669	10

Abbreviations: COPD, chronic obstructive pulmonary disease; URI, upper respiratory tract infection.

The first source was biennial manual counts of vehicle types performed by Metro Toronto Planning Department at 16 points in the study area. The average vehicle type distribution from this source over the years 1989, 1991, and 1993 provided an estimate of vehicle type distribution for 64.9% of modeled streets. We assigned the remaining 35.1% of streets the 1991 average vehicle type distribution in the Province of Ontario, obtained from the Ontario Ministry of Energy and the Environment (*38*). We did not perform sensitivity analyses to examine the impact of using Provincial vehicle type distribution, but the impact is likely minimal given the similarity between Provincial and Metro Toronto distributions. We calculated $PM_{2.5}$ emission factors for each vehicle type using the PART5 emission model (*39*). We then used vehicle type distribution, vehicle type emission factors, and traffic volumes to calculate the average daily mass of $PM_{2.5}$ emitted on each street segment (*40*).

Modeling of exposure to PM$_{2.5}$. We modeled EA exposure to $PM_{2.5}$ from motor vehicles by overlaying a modified street network on the EA boundaries and then proportionally transferring the street network emissions to the EAs based on street frontage and the proportion of the EA within 10 m of the street. Modification of the street network involved converting the street network to a series of polygons by creating a 10-m buffer polygon around each street segment. The buffers facilitated transfer of emissions to EAs near streets and allowed consideration of EA shape and size during exposure estimation. We selected a width of 10 m for the buffer because dispersion models and measurements suggest that curbside $PM_{2.5}$ concentrations decrease by approximately half within 10 m (*11,12,41*). Use of a single buffer size for all streets facilitates the calculation of exposure, and the 10-m buffer size accounts for the blocking effect of buildings on dispersion (*42*). Emission values in overlapping buffers were summed.

The overlay of the buffered street network on the EA boundaries produced a layer that contained 1,403 polygons, all labeled by the EA within which they fell, with 965 also labeled by the buffered street polygon within which they fell. We then calculated exposure values for each EA (g/24 hr) according to the following formula (graphically depicted in Figure 2):

$$EA_i = \sum_m^n \text{Value}(B_m) \times \frac{\text{Area}(B_m \text{ in } EA_i)}{\text{Area}(B_m)} \times \frac{\text{Area}(EA_i \text{ in } B_m)}{\text{Area}(EA_i)}, \quad [1]$$

where B_m is the mth of n buffer polygons that fall within EA_i, Value (B_m) is the total mass of emissions (in grams) in B_m, Area (B_m) is the total area of B_m (in m^2), Area (B_m in EA_i) is the area of B_m that falls in EA_i, Area (EA_i) is the total area of EA_i, and Area (EA_i in B_m) is the area of EA_i in B_m.

Value (B_m) and the first proportion in Equation 1 directly transfer the vehicle counts and PM emissions from the street network to the surrounding EAs on the basis of street frontage. Calculation of the direct transfer of emissions (i.e., without applying the weight in the last proportion of Equation 1) provided an opportunity to validate the method up to this point. The total emission of $PM_{2.5}$ from the street network was 549,170 g, whereas the total $PM_{2.5}$ exposure for the EA layer was 518,940 g (94.5%). A slightly lower value for the EA layer is attributable to the expected loss of emissions around the outer edge of the study area. The third and final element of the formula weight values transferred from the modified street network by the proportion of the EA area falling within 10 m of a street.

Measurement of SES. We obtained data describing SES of the EAs from the 1991 census (*34*). We constructed an SES index from census variables using a methodology previously employed for Canadian Census data (*43*). The index with the greatest explanatory power comprised variables describing educational attainment and family structure [see Buckeridge (*35*) for greater detail]. Besides examining the ability of an index to control for SES, we also considered a number of single variables describing dwelling characteristics, educational attainment, employment, income, mobility, family structure, and immigration.

Single variables describing EA income, unemployment, and education had greater explanatory power for hospital admissions than did other single variables or the SES index. Income and unemployment variables had a large number of missing values, so we used a measure of education in the final analysis to control for SES (*44*). Ultimately, we used the proportion of the population with a university degree as a measure of the SES of each EA. This variable offered the greatest explanatory power in isolation and had the least number of missing values, and sensitivity analyses revealed that neither addition nor substitution of other single SES variables meaningfully altered model fit or regression parameters.

Data analysis. Examination of spatial distributions involved mapping and calculation of global and local spatial autocorrelation. The literal meaning of spatial autocorrelation is self-correlation (autocorrelation) of observed values of a single attribute, according to the geographical (spatial) ordering of the values (*45*). Global autocorrelation statistics provide a single measure of spatial autocorrelation for an attribute in a region as a whole. Local spatial autocorrelation statistics provide a measure, for each unit in the region, of the unit's tendency to have an attribute value that is correlated with values in nearby areas. We examined local spatial autocorrelation for attributes that did not have significant global spatial autocorrelation.

To measure global spatial autocorrelation, we used the global Moran's I statistic (Equation 2) because it is robust in data structure, population structure, and size and has the power to detect clustering of the type likely to be seen in this study (*46–48*).

$$I = \frac{n}{\sum_i \sum_j w_{ij}} \times \frac{\sum_i \sum_j w_{ij}(y_i - \bar{y})(y_j - \bar{y})}{\sum_i (y_i - \bar{y})^2}, \quad [2]$$

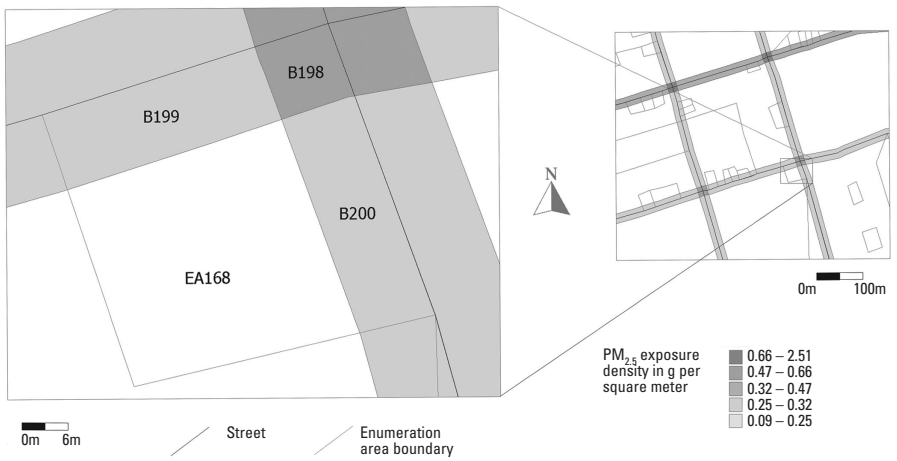

Figure 2. Graphical description of exposure assessment. The exposure for an EA (EA168; total area, 1,490 m^2; exposure, 32.8 g) is determined from the summation of total emissions in each overlaying buffer (B198, 215 g; B199, 994 g; B200, 2167 g), multiplied by the proportion of each buffer in the EA (B198, 0.24; B199, 0.07; B200, 0.04), weighted by the proportion of the EA occupied by each buffer (B198, 0.06; B199, 0.19; B200, 0.19).

where there are n EAs, the attribute value for EA i is y_i, and w_{ij} is the weight (or connectivity) for EAs i and j. We defined connectivity using a binary measure of adjacency (*46*). We calculated global Moran's I and its variance using SAS (*45*). We compared values of Moran's I against the expected value of $-1/(n-1)$ (*49*), and the interpretation is similar to that of the product moment correlation coefficient. Informally, +1 indicates strong positive spatial autocorrelation (i.e., clustering of similar values), 0 indicates random spatial ordering, and –1 indicates strong negative spatial autocorrelation (i.e., a checkerboard pattern).

We used local Moran's I to measure local spatial autocorrelation (*50*). Calculation of values and significance estimates used an Excel macro (*51*). This software required use of a distance weight matrix. We used a distance of 50 m because this gives a similar number of neighbors, and global Moran's I (which is equal to the sum of all possible local Moran's I values) as an adjacency measure (*52*).

We used custom programs to determine the adjacency matrix, validate the matrix structure (e.g., ensure symmetry), and assess the magnitude of the effect of missing EAs on spatial autocorrelation calculations. In addition, we manually selected a small number of areas from the matrix and verified the coding of neighbors.

Multivariate analysis involved the estimation of rank correlation among variables followed by the use of a Poisson mixed-effects regression model and spatial analysis of residuals. Poisson overdispersion was evident from the large residuals and poor goodness of fit after initial application of a fixed-effects Poisson model (*53*). Overdispersion probably results from violation of assumptions underlying the Poisson distribution—namely, a constant risk of hospital admissions and independence among admissions. We account for overdispersion because it can cause erroneously low standard error for regression parameters, and misleading inference (*53*). We used two approaches, adjustment of variance using the scale factor (*54*), and a Poisson mixed-effects model (*53*). We report findings for the mixed-effects model because results are similar with both approaches, and the mixed-effects model can be extended in future research. The mixed-effects model assumes that admissions are Poisson, conditional on fixed effects (i.e., exposure and SES) and a random error term. We assume the error term has a gamma distribution, which leads to a negative binomial distribution for the admission counts (*55*). We assessed the potential contribution of spatial autocorrelation to overdispersion by mapping and calculating Moran's I for the regression residuals (*45,56*).

To implement the regression model, we used the GENMOD procedure in SAS with a log link and a negative binomial error structure. The outcome variable was observed admission counts, and expected admissions were offset. The skewed distribution of exposure data suggested log or rank transformation of exposure data before regression modeling. Results were similar for both log and rank transformations, and we report results for exposure modeled as log $(x + 1)$. We modeled SES as a continuous covariate and assessed model goodness of fit by comparison of the model deviance against a chi-square distribution with the appropriate degrees of freedom and examination of regression residuals and influence measures (*57*). We also reanalyzed the data following deletion of influential and outlying observations.

Results

Table 2 shows results of procedures on hospital admissions data. Address matching to EA by postal code leaves 1.4% of all records unmatched because of postal codes that are invalid or outside of Ontario. This proportion is lower than results generally reported for address matching (*58*). Repeat admissions account for 15.6% of all admissions, but as Table 2 shows, readmissions are more common for respiratory than for genitourinary disorders. Table 1 shows the distribution of repeat admissions by respiratory disorder.

Univariate analysis. The vast majority of individuals in the study area were not admitted to the hospital during 1990–1992 (98.5% of the population for the respiratory subset). This resulted in relatively low incident admission counts for a number of EAs, with 72 EAs (24.5%) having no admissions and 110 EAs (36.4%) having between one and five admissions. The mean 3-year EA indirectly age- and sex-standardized admission rate per 1,000 is 5.4 [95% confidence interval (CI), 4.6–6.2] for the respiratory subset, 8.0 (95% CI, 7.0–9.0) for the respiratory chapter, and 7.8 (95% CI, 6.8–8.8) for the genitourinary chapter. Visual analysis of mapped rates identifies no clustering among EAs with similar values in any of the diagnostic sets. The calculated values of Moran's I confirm that there is no positive global spatial autocorrelation among values of the respiratory subset (Figure 3; Moran's $I = -0.005$, $p = 0.971$) or the respiratory chapter (Moran's $I = -0.045$, $p = 0.287$), although some mild global spatial autocorrelation appears to exist for the genitourinary chapter (Moran's $I = -0.081$, $p = 0.051$). Further examination of respiratory subset values revealed significant local spatial autocorrelation among a cluster of eight EAs in the southwest corner of the study area (Figure 4). The EAs in this cluster tend to have a higher respiratory subset admission rate (cluster average, 27.6 per 1,000; SETO average, 5.4 per 1,000) and a lower mean university completion rate (cluster average, 12.7 per 1,000; SETO average, 23.2 per 1,000) than the rest of SETO.

EAs exhibit considerable variation in modeled exposure to $PM_{2.5}$. The median exposure is 26.3 g/24 hr, but 63 EAs (20.9%) have an exposure of zero, and the distribution is skewed to the right by EAs with higher values (maximum, 1183.4 g/24 hr). Spatially, EAs with higher exposure tend to fall near busier streets (as indicated in Figure 3 by the vertical and horizontal swaths of higher exposure, which correspond to the location of busier streets), and this results in moderate positive spatial autocorrelation (Moran's $I = 0.308$, $p < 0.001$).

The proportion of the population with a university degree ranges from 1.2% to 62.5%. Values between these extremes are approximately normally distributed, with a mean of 23.2% (95% CI, 21.6–24.8). No large-scale spatial trend is evident in the

Table 2. Outcome of procedures applied to hospital admission data.

Procedure applied to data	Number of records remaining in diagnostic set after procedure (% decrease in records due to procedure)			
	Respiratory subset	Respiratory chapter	Genitourinary chapter	Total[a]
Acquire City of Toronto records from HMRI	14,344	21,945	19,377	41,322
Remove records not matching to Ontario EA	14,087 (1.8)	21,563 (1.7)	19,195 (0.9)	40,758 (1.4)
Remove records not in a SETO EA	2,596 (81.6)	3,668 (83.0)	2,849 (85.2)	6,517 (84.0)
Remove records in 32 suppressed EAs	2,495 (3.9)	3,529 (3.8)	2,781 (2.4)	6,310 (3.2)
Remove records without valid birth date	2,454 (1.6)	3,481 (1.4)	2,754 (1.0)	6,235 (1.2)
Remove records without valid health number	2,322 (5.4)	3,316 (4.7)	2,669 (3.1)	5,985 (4.0)
Remove records for repeat visits in study period	1,779 (23.4)	2,646 (20.2)	2,406 (9.9)	5,052 (15.6)

[a]Total is of the respiratory and the genitourinary chapters; the respiratory subset records are included in the respiratory chapter.

distribution of values, but local clustering of similar values is evident in several areas (Moran's $I = 0.352$, $p < 0.001$). Figure 3 shows clustering of high values in the northeast and northwest and clustering of low values in the southeast.

Multivariate analysis. The rank correlation results in Table 3 reveal that all health variables are moderately correlated with exposure to $PM_{2.5}$ and that SES is more strongly correlated with measures of respiratory than genitourinary admission. It is noteworthy that SES is not correlated with $PM_{2.5}$ exposure.

The regression results (Table 4) indicate that exposure to $PM_{2.5}$ has a significant effect on respiratory subset admission rates, before (model 1) and after (model 7) adjustment for SES. In the SES-adjusted model, the estimate of relative risk is 1.24 (95% CI, 1.05–1.45) for a $\log_{10}$ increase in exposure to $PM_{2.5}$. In this study, modeled $PM_{2.5}$ exposure ranges over three orders of magnitude. A slightly weaker and nonsignificant effect of $PM_{2.5}$ exposure is noted on all respiratory chapter conditions before (model 2) and after (model 8) adjustment for SES, with a relative risk of 1.17 (95% CI, 0.99–1.37) after SES adjustment.

The results also indicate that modeled exposure to $PM_{2.5}$ does not have a significant effect on genitourinary chapter admission rates (models 3 and 9). The relative risk after SES adjustment is 1.07 (95% CI, 0.92–1.25). SES has a significant effect on all outcomes (models 4–6), and control for SES tends to enhance the effect of $PM_{2.5}$ exposure on all outcomes studied.

Examination of model fit (deviance over degrees of freedom; Table 4) suggests that the models tend to fit the data well (*59*). An analysis conducted after deletion of seven poorly fitted and influential EAs produced a somewhat stronger effect of exposure on hospitalization rates. These EAs do not appear to demonstrate any spatial pattern, but the dominant type of housing in most is high-rise dwelling. We explored the contribution of a variable describing housing type and did not observe a significant contribution to model fit or impact on $PM_{2.5}$ effect.

There does not appear to be a large-scale spatial trend in the distribution of the likelihood residuals displayed in Figure 3. In addition, there is no global spatial autocorrelation of the residuals (global Moran's $I = -0.072$, $p = 0.919$). A cluster of significant local spatial autocorrelation exists in the same region where local spatial autocorrelation was noted in the respiratory subset rates (Figure 4).

Discussion

The results of this study identify an ecologic effect of modeled $PM_{2.5}$ exposure from motor vehicle emissions on the rate of hospitalization for selected respiratory diagnoses. The possibility that this is a causal association is supported by a weaker effect of $PM_{2.5}$ exposure on hospitalization for all respiratory conditions, and by the lack of a similar effect of exposure on hospitalization for nonrespiratory (i.e., genitourinary) conditions.

The strength of estimated effect in this study is similar to estimates from individual-level case–control (*16*) and cross-sectional studies (*12,19,23,24,60*) that note an association. Studies that do not find an association tend to use methods of exposure estimation that result in considerable misclassification (*18,21,22*), although this is not always so (*17*).

Our results suggest that exposure to $PM_{2.5}$ has a specific effect on certain respiratory conditions. The only published study to examine the specificity of the association between exposure and respiratory conditions reports an association between residential proximity to a major street and admission for all causes (*16*). Although this observed specificity of effect makes a causal association appear more likely (*61*), it is debatable how much weight should be given to specificity when assessing causality (*62*).

In general, our findings are noteworthy, but as with any study, the data and methods have both strengths and limitations. In the remainder of the discussion, we examine the strengths and limitations of our work under the broad categories of respiratory health, exposure assessment, and study design/analysis. By identifying limitations, we hope to clarify the problems encountered in addressing the research questions and highlight areas for future research.

Respiratory health. Incident hospital admissions as used in this study are a comprehensive measure in the population under study and have high validity. Lower respiratory diagnoses have been objectively assessed in only three other studies (*16–18*), with all other studies relying on self-reported symptoms. Despite their advantages, hospital admission rates are generally limited in that they probably give a conservative estimate of the health impact in comparison to prevalence estimates and ambulatory utilization or self-reported health status data. In addition, admission for some respiratory conditions, such as asthma, may be associated with suboptimal ambulatory care, which may in turn be associated with low SES. This could lead to a selection bias if individuals with low SES were more likely to live near busy streets. However, in our data there does not appear to be an association between SES and residential proximity to busy streets. This lack of association between area exposure to motor vehicle emissions and SES does not agree with much

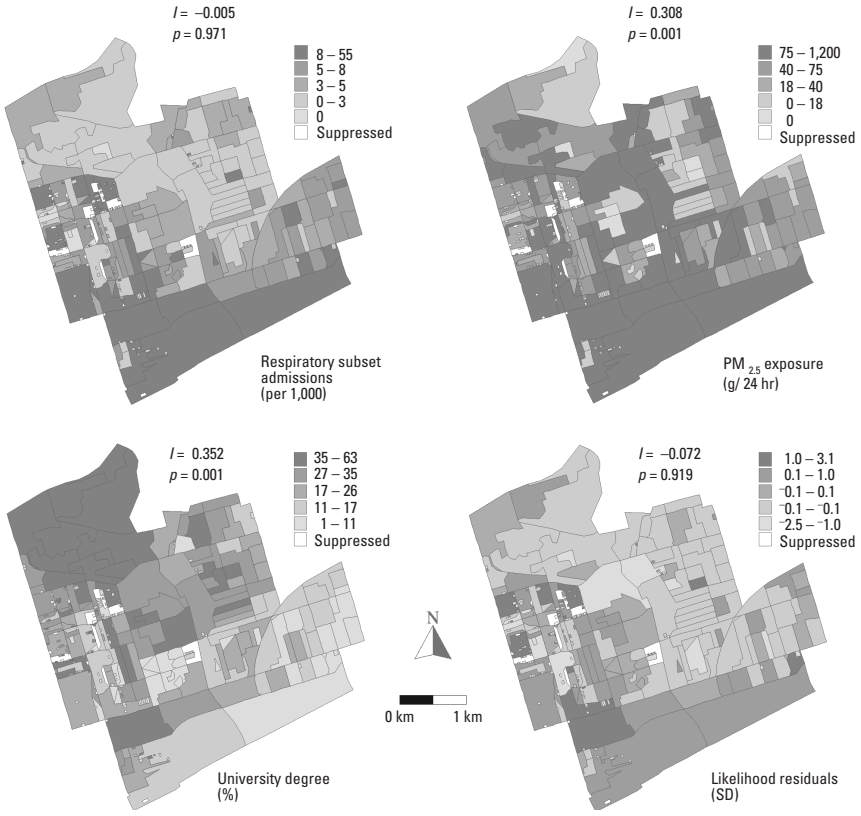

Figure 3. Spatial distributions and global spatial autocorrelation of regression analysis variables and residuals.

of the literature on environmental justice (*63*). This finding deserves further scrutiny. One possible explanation is the socioeconomic heterogeneity of the study area, which contains two college campuses and lacks the homogeneous areas of low SES that are seen in many other inner cities.

Exposure assessment. The exposure assessment model used in this study represents a refinement over previous studies in three important ways. First, the model accounts for emissions from all major streets. Except for one other study (*17*), all previous studies consider the contribution to exposure of only the one closest street. This could lead to an underestimation of exposure, especially in urban areas where busy streets are close together. Second, we model emission and dispersion of a single pollutant in an integrated manner to account for both traffic volume and distance from streets. One study models exposure in an integrated manner but uses a considerably more complex model that is not easily generalized to different settings (*19*). Other studies account for only the effect of emission (*21–24*) or dispersion (*12,18*) or account for both in an ad hoc manner (*14,16,17*). Incorporation of both emission and dispersion into a single measure should provide a more realistic estimate of exposure. Third, the use of a GIS automates the modeling process. This automation through a GIS can reduce error when compared to manual processes used in some studies (*12,16*) and allows for the integration of otherwise incompatible data sets (*64*).

Limitations of our exposure assessment model relate to data availability and the need for further validation. Data were not readily available to account for individual spatiotemporal activity patterns, indoor air quality, or meteorologic conditions. We attempted to minimize the impact of activity patterns by assessing average daily exposure at home, where individuals spend most of their time (*65*). However, this is a simplification that ignores potentially important and interacting factors such as temporal fluctuations in traffic flow (i.e., "rush hour") and the propensity for people to be away from their homes at certain times (e.g., at rush hour). Although we were unable to assess indoor air quality directly, we note that outdoor sources account for a considerable proportion of indoor $PM_{2.5}$ (*66*), with personal monitoring studies suggesting that outdoor sources account for 60% of total exposure on average (*67*). We addressed the lack of meteorologic data to some extent by studying exposure over an extended temporal period. Examination of urban emission dispersion models suggests that spatial dispersion patterns become decreasingly sensitive to meteorologic conditions as the time period under study increases (*68–70*). Nevertheless, more accurate modeling of the impact on exposure of temporal emission fluctuations is a subject requiring further investigation, possibly through the combined use of geographical and time-series methods. Other aspects of the model that should be subject to future research include the use of a single 10-m buffer around roads, modeling of exposure at intersections, and representation of physical and geographical characteristics such as buildings and valleys.

The exposure model has not been validated through spot measurement or personal monitoring because of our desire to demonstrate the general utility of our model before undertaking costly monitoring studies. In addition, exposure monitoring does not readily demonstrate the source of emissions and is susceptible to bias (*71*). Validation of our model through monitoring studies and/or replication of this study in another area are necessary future steps before further application of our model. Sensitivity analyses have been conducted around a number of model parameters, with the results described in detail elsewhere (*35*). In brief, these analyses suggest that exposure modeling is insensitive to the weight applied in transferring emissions from streets to study units, and that modeling of exposure to traffic volume produces results similar to those seen for $PM_{2.5}$ exposure.

Study design and analysis. We used an ecologic design for this study for two reasons. First, exposure, outcome, and associated policy issues are most naturally considered at the population or area level. Second, data on exposure and confounders are not readily available at the individual level. The potential biases in ecologic studies are, however, generally more numerous than those in individual-level studies and different in nature. Moreover, it is not possible to discern the magnitude or direction of these biases in the absence of individual-level data (*72,73*). Considerable caution must therefore be exercised in drawing individual-level inference from ecologic results. In the future, it would be informative to apply a further refined version of our model (e.g., one that employs multiple exposure zones to decrease exposure misclassification) in an individual-level study. Although the ecologic design limits individual-level inference, difficulties in cross-level inference are also encountered in individual-level studies (*74*).

The most likely sources of bias in this study are confounding and within-group misclassification. We attempt to control for some confounders through rate standardization, which may bias effect estimates if all

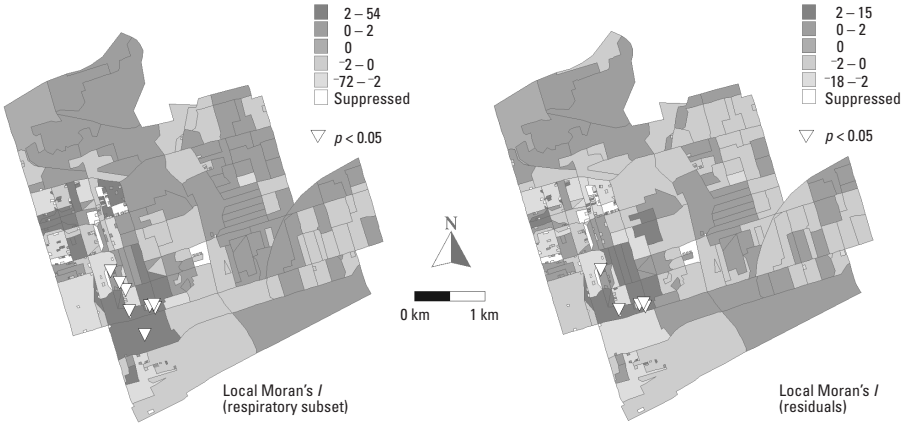

Figure 4. Local spatial autocorrelation of respiratory subset rates, and of regression residuals.

Table 3. Rank correlation results.[a]

Data	Standardized hospital admission rates: Respiratory subset	Respiratory chapter	Genitourinary chapter	Exposure $PM_{2.5}$	SES (university graduation)
Respiratory subset	1	0.949 (0.001)	0.740 (0.001)	0.222 (0.001)	–0.226 (0.001)
Respiratory chapter	0.949 (0.001)	1	0.784 (0.001)	0.206 (0.001)	–0.184 (0.002)
Genitourinary chapter	0.740 (0.001)	0.784 (0.001)	1	0.189 (0.001)	–0.099 (0.101)
$PM_{2.5}$	0.222 (0.001)	0.206 (0.001)	0.189 (0.001)	1	0.030 (0.625)
University graduation	–0.226 (0.001)	–0.184 (0.002)	–0.099 (0.101)	0.030 (0.625)	1

[a]Correlation coefficients are shown in the matrix with *p*-values given in parentheses.

Table 4. Regression analyses.

Model number, dependent variable	df	Fit[a]	Independent variable Name	χ^2	RR[b]	95% CI
Unadjusted models						
1. Respiratory subset	300	1.189	$PM_{2.5}$	3.95	1.18	1.00–1.39
2. Respiratory chapter	300	1.217	$PM_{2.5}$	1.89	1.12	0.95–1.31
3. Genitourinary chapter	300	1.228	$PM_{2.5}$	0.23	1.04	0.89–1.21
4. Respiratory subset	300	1.190	SES	14.26	0.84	0.77–0.92
5. Respiratory chapter	300	1.219	SES	11.74	0.86	0.79–0.94
6. Genitourinary chapter	300	1.229	SES	8.12	0.89	0.82–0.96
Models adjusted for SES						
7. Respiratory subset	299	1.195	$PM_{2.5}$	6.67	1.24	1.05–1.45
			SES	17.05	0.83	0.76–0.91
8. Respiratory chapter	299	1.225	$PM_{2.5}$	3.58	1.17	0.99–1.37
			SES	13.50	0.85	0.78–0.93
9. Genitourinary chapter	299	1.234	$PM_{2.5}$	0.80	1.07	0.92–1.25
			SES	8.73	0.88	0.81–0.96

Abbreviations: df, degrees of freedom; RR, relative risk. SES is measured by percentage of population with a university degree.
[a]Fit is the model deviance over degrees of freedom. [b]Relative risk is relative to a log base 10 increase for $PM_{2.5}$ and relative to a 10% increase in SES.

variables are not standardized in the same manner (*75*). A repeat analysis with standardized variables (i.e., age, sex) as covariates suggests no bias from our approach to standardization (data not shown). Nevertheless, it is likely that we were not able to fully control for the effect of all confounders, especially SES, which varies considerably throughout the study area (Figure 3). Other potential confounders that we were not able to measure include duration of residence, comorbidity, smoking, and exposure to other pollutants in vehicle emissions. Previous studies suggest that control for duration of residence has little influence on effect estimates (*12,19,24*), possibly because of an acute effect of exposure. We considered the use of consumer purchasing data to control for area-level smoking, but available data were of questionable validity. Given the similar dispersion characteristics of $PM_{2.5}$ and other pollutants in vehicle emissions (e.g., NO_2), some of the observed effect may be caused by exposure to other pollutants.

The assumption that all residents in a study unit receive the same exposure is likely not true and probably results in within-group misclassification. This misclassification is likely nondifferential with respect to outcome, but in an ecologic study nondifferential misclassification may bias effect estimates away from the null (*76*). We use the smallest possible study unit to minimize bias from this source (*77*). However, selection of a small geographic unit adversely affects the stability of rates for health events. We attempted to account for this impact by using 3-year rates and indirect standardization, but in selecting the size of the study unit there is an inherent trade-off between exposure misclassification and stability of rates.

From an analytic perspective, we attempted to minimize and characterize the impact of overdispersion by using incidence as opposed to prevalence rates (*78*), accounting for overdispersion in the regression model (*53*) and examining regression residuals for evidence of spatial autocorrelation (*45*). There was no global spatial autocorrelation of the regression residuals and only a small region of significant local spatial autocorrelation. The contribution of spatial autocorrelation to overdispersion therefore appears to be minor. This suggests that there is not a clear indication to fit a spatial autoregressive model (to explicitly account for spatial dependence), but such analysis could be a topic for future research (*49*).

In summary, using a refined exposure model, we demonstrate a significant effect of modeled area exposure to $PM_{2.5}$ from motor vehicle emissions on hospital admission rates for selected respiratory conditions. Although these results agree with those of many previous studies, caution should be exercised in drawing individual-level inference from these ecologic findings. Finally, we identified a number of avenues for further inquiry into exposure modeling and analysis of environmental exposure.

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