January 2002

Effect of motor vehicle emissions on respiratory health in an urban area

David L Buckeridge  
*University of Toronto, Canada*

Richard Glazier  
*University of Toronto, Canada*

Bart J Harvey  
*University of Toronto, Canada*

Michael Escobar  
*University of Toronto, Canada*

Carl Amrhein  
*Aga Khan University, carl.amrhein@aku.edu*

*See next page for additional authors*

Follow this and additional works at: [https://ecommons.aku.edu/provost_office](https://ecommons.aku.edu/provost_office)

Part of the Urban Studies and Planning Commons

**Recommended Citation**

Buckeridge, D., Glazier, R., Harvey, B., Escobar, M., Amrhein, C., Frank, J. (2002). Effect of motor vehicle emissions on respiratory health in an urban area. *Environmental Health Perspectives, 110*(3), 293-300.

Available at: [https://ecommons.aku.edu/provost_office/5](https://ecommons.aku.edu/provost_office/5)
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$_{1.5}$ 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] http://ehpnet1.niehs.nih.gov/docs/2002/110p293-300buckeridge/abstract.html

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 admission 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
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.

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                            | 433.0-1,493.9 | 430         | 642        | 33                    |
| Bronchitis                        | 466.0-1, 490 | 127         | 139        | 9                     |
| COPD                              | 491.0-2, 491.8-9, 492, 493 | 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.

$$E = \sum_{n} \frac{\text{Value}(B_n) \times \text{Area}(B_n \text{ in } EA)}{\text{Area}(EA)}$$

where $B_m$ is the $m$th 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$^2$), $\text{Area}(B_n \text{ in } EA_i)$ is the area of $B_n$ that falls in $EA_i$, $\text{Area}( EA_i)$ is the total area of $EA_i$, and $\text{Area}( EA_i)$ is the area of $EA_i$ in $B_n$.

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).

$$I = \frac{1}{n} \sum_{i,j} w_{ij} (y_i - \bar{y})(y_j - \bar{y})}{\sum_{i,j} w_{ij}},$$

where $y_i$ and $y_j$ are the values of the variable for two units, $\bar{y}$ is the mean value of the variable, and $w_{ij}$ is a weight that measures the spatial relationship between units $i$ and $j$. The weight $w_{ij}$ is usually chosen to reflect the spatial proximity of units, with higher weights for closer units.

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).

$$EA = \sum_{m} \text{Value}(B_m) \times \text{Area}(B_m \text{ in } EA \text{ in } B_m) \times \text{Area}(EA)$$

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).

$$I = \frac{1}{n} \sum_{i,j} w_{ij} (y_i - \bar{y})(y_j - \bar{y})}{\sum_{i,j} w_{ij}},$$

where $y_i$ and $y_j$ are the values of the variable for two units, $\bar{y}$ is the mean value of the variable, and $w_{ij}$ is a weight that measures the spatial relationship between units $i$ and $j$. The weight $w_{ij}$ is usually chosen to reflect the spatial proximity of units, with higher weights for closer units.
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 \((40)\). We calculated global Moran’s \( I \) and its variance using SAS \((43)\). 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 point momentum correlation coefficient. Informally, \(+1\) indicates strong positive spatial autocorrelation (i.e., clustering of similar values), \(-1\) 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 \((55)\). 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 admission 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 the respiratory chapter, and 7.8 (95% CI, 9.6–8.8) for the genitorinary 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 genitorinary 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

| Procedure applied to data | Number of records remaining in diagnostic set after procedure | Respiratory subset | Respiratory chapter | Genitorinary chapter | Total* |
|---------------------------|-------------------------------------------------------------|---------------------|---------------------|---------------------|-------|
| 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 (81.8) | 3,688 (83.0) | 2,849 (85.2) | 6,310 (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 | 2,654 (1.6) | 3,481 (1.4) | 2,764 (1.0) | 6,225 (1.2) |
| Remove records without valid health number | 2,322 (5.4) | 3,318 (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) | 2,408 (9.9) | 5,052 (15.6) |

*Total is of the respiratory and the genitorinary chapters; the respiratory subset records are included in the respiratory chapter.
exposure to PM 2.5 does not have a significant effect 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 PM2.5 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 PM2.5 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 PM2.5 exposure on hospitalization for all respiratory conditions, and by the lack of a similar effect of exposure on hospitalization for non-respiratory (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 PM2.5 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 spatio-temporal activity patterns, indoor air quality, or meteorologic conditions. We attempted to minimize the impact of activity patterns by averaging 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 | Exposure PM$_{2.5}$ | SES (university graduation) |
|--------------------|---------------------------------------|---------------------|-----------------------------|
| Respiratory subset | 0.949 (0.001)                         | 0.222 (0.001)       | -0.226 (0.001)              |
| Respiratory chapter| 0.740 (0.001)                         | 0.206 (0.001)       | 0.030 (0.625)               |
| Genitourinary chapter| 0.784 (0.001)                       | 0.189 (0.001)       | 0.189 (0.001)               |
| PM$_{2.5}$         | 0.189 (0.001)                         | -0.099 (0.101)      | -0.184 (0.022)              |
| University graduation| -0.099 (0.101)                    | 1                   | 0.030 (0.625)               |

$^a$Correlation coefficients are shown in the matrix with $p$-values given in parentheses.
variables are not standardized in the same manner (75). A repeat analysis with standar-
dized variables (i.e., age, sex) as covariates suggests no bias from our approach to stan-
dardization (data not shown). Nevertheless, it is likely that we were not able to fully con-
trol for the effect of all confounders, espe-
cially SES, which varies considerably through-
out the study area (Figure 3). Other potential confounders that we were not able

to measure include duration of residence, comorbid-ity, 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 PM2.5 and other pollutants in vehicle emissions
(e.g., NO2), 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 mis-
classification 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), account-

ing for overdispersion in the regression

model (53) and examining regression resid-

uals for evidence of spatial autocorrelation

(49). There was no global spatial autocor-

relation of the regression residuals and only a

small region of significant local spatial auto-
correlation. The contribution of spatial

correlation 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 PM2.5 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.

REFERENCES AND NOTES

1. Dockery DW, Pope CAI, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG, Speizer FE. An association between air

pollution and mortality in six U.S. cities. N Engl J Med 329:1793–1799 (1993).

2. Pope CAI, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, Heath CW. Particulate air pollution as a

predisector of mortality in a prospective study of U.S. adults. Am J Respir Crit Care Med 151:689–674 (1995).

3. Pope A, Dockery D. Epidemiology of chronic health effects: cross-sectional studies. In: Particles in Our Air—

Concentrations and Health Effects (Wison R, Spengler J, eds). Boston: Harvard University Press, 1996:149–167.

4. Speizer JM, Dominici F, Curriero FC, Course J, Zeger SL. Fine particulate air pollution and mortality in 20 U.S.
cities, 1987–1994. N Engl J Med 343:1742–1749 (2000).

5. Westerholm R, Karl-Erik E. Exhaust emissions from light-
duty vehicles: chemical composition, impact of exhaust after treatment, and fuel parameters. Environ Health Perspect 102(suppl4):113–123 (1994).

6. Bascom R, Brromberg P, Costa D, Delvin R, Dockery D, Frampton M, Lambert W, Samet J, Speizer F, Utell M. Health

effects of outdoor air pollution. Am J Respir Crit Care Med 153:53–50 (1996).

7. Chow JC, Watson JO, Frazier CA, Egami RT, Goddard A, Ralph C. Spatial and temporal source contributions to

PM10 and PM2.5 in Reno, NV. In: PM-10: Implementation of Standards: Transactions: An APCHA/EPA International

Specialty Conference, San Francisco, CA (Mathal CV, Stonefield DH, eds). Pittsburgh: APCHA, 1986:438–457.

8. Japar SM. Motor vehicles and particle air pollution: an overview. In: Particulate Matter: Health and Regulatory

Issues VIP-49. Proceedings of an International Specialty

Conference, 4-6 April 1996, Pittsburgh, PA. Pittsburgh Air

and Waste Management Association: 1995:579–589.

9. Brook JR, Dann TF, Burnett RT. The relationship among

TSP, PM10, and inorganic constituents of atmos-

pheric particulate matter at multiple Canadian locations.

J Air Waste Manag Assoc 47:2–19 (1997).

10. Burton RM, Suh HH, Koutrakis P. Spatial variation in par-

ticulate concentrations within metropolitan Philadelphia.

Environ Sci Technol 30:460–467 (1996).

11. Eerens HC, Sliggers CJ. The CAR model: the Dutch

method to determine city street air quality. Atmos

Environ 27B:389–399 (1993).

12. Nitte H, Sato T, Nakai: S, Maeda K, Aoki S, Dno M. Respiratory health associated with exposure to automotive

exhaust. I. Results of cross-sectional studies in 1979, 1982, and 1983. Arch Environ Health 48:53–58 (1993).

13. Kundi N, Kaiser R, Medina S, Studnicka M, Chouel G, Filliger P, Herry M, Horak F, Juybournieu-Texier V, Quenel P, et al. Public-health impact of outdoor and traf

fic-related air pollution: a European assessment. Lancet 356:801–805 (2000).

14. Bruneckre B, Janssen NA, de Hartog J, Harssema H, Knape M, van Vliet P. Air pollution from truck traffic and lung function in children living near motorways. Epidemiology 8:289–303 (1997).

15. Buckeridge D, Godzry F, Ferguson K, Schrenk M, Skinner J, Tamm T, Amrenhe C. A study of the relationship between vehicle emissions and respiratory health in an urban area. Geor Envir Monit Model 2:17–36 (1998).

16. Edwards J, Walters S, Griffrs F. Hospital admissions for

asthma in preschool children: relationship to major roads in

Birmingham, UK. Arch Environ Health 49:223–227 (1994).

17. English P, Neutra R, Scafl R, Sullivan M, Walle L, Zhu L. Examining associations between childhood asthma and traffic flow using a geographic information system. Environ Health Perspect 107:761–767 (1999).

18. Livingstone AE, Shaddick G, Elliott P. Do people living near inner city main roads have more asthma needing treat-

ment? Case-control study. Br Med J 312:676–677 (1996).

19. Gosterlere A, Driyer M, Lebet E, Bruneckre B. Chronic respiratory symptoms in children living along streets with high traffic density. Occup Environ Med 53:241–247 (1996).

20. van Vliet P, Knape M, de Hartog J, Janssen N, Harssema H, Bruneckre B. Motor vehicle exhaust and chronic res-

piratory symptoms in children living near freeways. Environ Res 74:122–132 (1997).

21. Venn A, Lewis S, Cooper M, Hubbard R, Hill I, Boddy R, Bell M, Britton J. Local road traffic activity and the prevalence, severity, and persistence of wheeze in school children: combined cross sectional and longitudinal

study. Occup Environ Med 57:152–158 (2000).

22. Waidron D, Potelige B, Dod J. Asthma and the motor-

ways—one district’s experience. J Public Health Med 17:85–89 (1995).

23. Weiland S, Munt S, Ruckmann K, Ael U. Self-reported wheezing and allergic rhinitis in children and traffic density

on street of residence. Am Epidemiol 24:243–247 (1994).

24. Wijst M, Reitmar P, Dold S, Wulf A, Nicolai T, von Loeffelholz-Colberg E, von Mutius E. Road traffic and adverse effects on respiratory health in children. Br Med J 307:596–600 (1993).

25. Canadian Institute for Health Information. Ottawa, ON:Canadian Institute for Health Information. Available at:

http://www.cihi.ca/ [cited 30 January 2002].

26. WHO. Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death. 9th Revision. Geneva:World Health Organization, 1975.

27. Weiss LS, Mustard CA, Nicol JP, McLauren DF, Melanek DJ, Young TK, Cohen MM. Registries and administrative
data: organization and accuracy. Med Care 31:201–212 (1993).
28. Burnett RT, Dales R, Krewski D, Vincent R, Dann T, Brook JR. Associations between ambient particulate sulfate and admissions to Ontario hospitals for cardiac and respiratory disease. Am J Epidemiol 142:15–22 (1995).
29. Burnett RT, Cakmak S, Brook JR, Krewski D. The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. Environ Health Perspect 105:614–620 (1997).
30. Gordan ME, Mee S, Okiyak H, Xue J, Spengler J. Particulate air pollution and respiratory disease in Anchorage, Alaska. In: Particulate Matter, Health and Regulatory Issues VIP-49. Proceedings of an International Specialty Conference, 4–6 April 1995, Pittsburgh, PA. Pittsburgh:Air and Waste Management Association, 1995;143–166.
31. Stieb DM, Beveridge RC, Brook JR, Burnett RT, Anis AH, Dales RE. The Saint John particle health effects study measuring health effects, health costs and quality of life impacts using enhanced administrative data: design and preliminary results. In: Particulate Matter: Health and Regulatory Issues. Proceedings of an International Specialty Conference, Transactions: an APICA/EPA International Specialty Conference, Pittsburgh, PA, 4–6 April 1995. Pittsburgh:Air and Waste Management Association, 1995;131–142.
32. Statistics Canada. Postal Code Conversion File (PCCF) Codebook. Ottawa:Government of Canada, 1994.
33. Wilkins R. Use of postal codes and addresses in the analysis of health data. Health Rep 5:157–177 (1993).
34. Griffith DA. Spatial Regression Analysis on the PC: Spatial data—distributional considerations. Am J Epidemiol 139:747–760 (1994).
35. Haining R. Designing spatial data analysis modules for geographic information systems: their use in environmental epidemiological research. Environ Health Perspect 105:598–605 (1997).
36. Walter SD. Assessing spatial patterns in disease rates. Stat Med 12:1885–1894 (1993).
37. Walter SD. The analysis of regional patterns in health data—the power to detect environmental effects. Am J Epidemiol 136:724–759 (1992).
38. Walter SD. Assessing spatial patterns in disease rates. Stat Med 12:1885–1894 (1993).
39. Office of Mobile Sources. Highway Vehicle Particulate Modelling Software (PARTS). Ann Arbor, MI: U.S. Environmental Protection Agency, 1995.
40. Rayfield D, Langhurst JWS, Conlan DE, Watson AFR, Hewison T. Procedures for the estimation of vehicle emissions in an urban environment. In: Urban Transport and the Environment for the 21st Century. Southampton, UK: National Mechanics Publications, 1995;207–214.
41. Wrobel A, Rokita E, Maenhaut W. Transport of traffic-related aerosols in urban areas. Sci Total Environ 257:199–211 (2000).
42. Holscher N, Hoffer R, Niemann H-J, Brilon W, Romberg A. Methods for Small-Area Studies (Elliott P, Cuzick J, eds). New York:John Wiley & Sons, 1991;101–130.
43. Jolley DB, Jarman B, Elliott P. Socio-economic confounding. In: Geographical and Environmental Epidemiology: Methods for Small-Area Studies (Elliott P, Cuzick J, English D, Stern R, eds). Oxford:Oxford University Press, 1992;115–124.
44. Griffith DA. Spatial Regression Analysis on the PC. Spatial Statistics Using SAS. Washington, DC:Association of American Geographers, 1993.
45. Avin A. The analysis of regional patterns in health data—distributional considerations. Am J Epidemiol 136:730–741 (1992).
46. Walter SD. The analysis of regional patterns in health data—the power to detect environmental effects. Am J Epidemiol 136:724–759 (1992).
47. Walter SD. Assessing spatial patterns in disease rates. Stat Med 12:1885–1894 (1993).
48. Bailey T, Gatrell A. Interactive Spatial Data Analysis. New York:Longman Scientific & Technical, 1995.
49. Anselin L. Local indicators of spatial autocorrelation—LISA. Geog Anal 27:93–115 (1995).
50. Savada M. RookCase: An Excel 97/2000 Visual Basic (VB) add-in for exploring global and local spatial autocorrelation. Bull Ecol Soc Am 80:231–234 (1999).
51. Gets A, Ord JK. Local spatial statistics: an overview. In: Spatial Analysis: Modelling in a GIS Environment (Langethy P, Batty M, eds). New York:John Wiley & Sons, 1996;261–282.
52. Dean CB. Ondispersion. In: Encyclopedia of Biostatistics (Armitage P, Colton T, eds). New York:John Wiley & Sons, 1998;3226–3327.
53. McCullagh P, Nelder JA. Generalized Linear Models. London:Chapman & Hall, 1989.
54. Clayton D, Kaldor J. Empirical Bayes estimates of age-standardized relative risks for use in disease mapping. Biometrics 43:871–881 (1987).
55. Myers RH. Classical and Modern Regression with Applications. Boston:PIW-Kent, 1990.
56. Vine MF, Degnan D, Manchette C. Geographic information systems: their use in environmental epidemiological research. Environ Health Perspect 105:598–605 (1997).
57. Bailey T, Gatrell A. Interactive Spatial Data Analysis. Oxford:Oxford University Press, 1993.
58. Keil U, Weiland S, Duhme H, Chambless L. The international study of asthma and allergies in childhood (ISAAC): objectives and methods; results from German ISAAC centres concerning traffic density and wheezing and allergic rhinitis. Toxicol Lett 86:99–103 (1996).
59. Hill A. The environment and disease: association or causation? Proc R Soc Med 58:295–300 (1965).
60. Rothman K, Greenland S. Causation and causal inference. In: Modern Epidemiology (Rothman K, Greenland S, eds). Philadelphia:Lippincott-Raven, 1998:7–28.
61. Committee on Environmental Justice. Toward Environmental and Occupational Justice: Research, Education, and Winning Policy Needs. Washington, DC:Institute of Medicine, 1999.
62. Twigg L. Health bases geographical information systems: their potential examined in the light of existing data sources. Soc Sci Med 30:143–155 (1990).
63. Moschandreas D. Exposure to pollutants and daily time budgets of people. Bull NY Acad Med 57:845–859 (1981).
64. Alt E, Suh HH, Allen G, Koutrakis P. Characterization of indoor particles: a study conducted in the metropolitan Boston area. Environ Health Perspect 103:34–40 (1995).
65. Okiyak H, Xue J, Spengler J, Wallace L, Pellizzari E, Jenkins P. Personal exposure to airborne particles and metals: results from the particle team study in Riverside, California. J Expo Anal Environ Epidemiol 6:57–78 (1996).
66. Kono H, Is S. A micro-scale dispersion model for motor vehicle exhaust in urban areas—OMG volume-source model. Atmos Environ 30:245–256 (1996).
67. Fisher BEA. Atmospheric dispersion and emission modelling. In: Highway Pollution (Hamilton RS, Harrison RM, eds). New York:Elsevier, 1991;91–130.
68. Glen GW, Zelenka MP, Graham RC. Relating meteorological variables and trends in vehicle emissions to monthly urban carbon monoxide concentrations. Atmos Environ 30:425–432 (1996).
69. Oglesby L, Rokita T, Kruft P, Boudet C, Krueze H, Den MJ, Kunzli N. Personal exposure assessment studies may suffer from exposure-relevant selection bias. J Expo Anal Environ Epidemiol 10:251–266 (2000).
70. Greenland S, Robins J. Invited commentary: ecological studies—biases, misconceptions and counterexamples. Am J Epidemiol 139:747–760 (1994).
71. Margenstern H. Ecologic studies. In: Modern Epidemiology (Rothman K, Greenland S, eds). Philadelphia:Lippincott-Raven, 1998;459–480.
72. Schwartz S. The fallacy of the ecological fallacy: the potential misuse of a concept and the consequences. Am J Public Health 84:819–824 (1994).
73. Rosenbaum P, Rubin D. Difficulties with regression analyses of age-adjusted rates. Biometrics 40:437–443 (1984).
74. Brenner H, Savitz D, Jockel K, Greenland S. Effects of nondifferential exposure misclassification in ecologic studies. Am J Epidemiol 135:85–92 (1992).
75. Mausner M. Linkage failures in ecologic studies. World Health Stat Q 40:79–84 (1995).
76. Campbell SM, Diehr P. Testing the null hypothesis in small area analysis. Health Serv Res 27:267–294 (1992).