Traffic Air Pollution and Mortality Rate Advancement Periods

Murray M. Finkelstein[1,2], Michael Jerrett[2,3], and Malcolm R. Sears[4]

[1] Program in Occupational Health and Environmental Medicine, McMaster University, Hamilton, Ontario, Canada.
[2] Institute of Environment and Health, McMaster University, Hamilton, Ontario, Canada.
[3] School of Geography and Geology, Health Studies Program, McMaster University, Hamilton, Ontario, Canada.
[4] Department of Medicine, Faculty of Health Sciences, McMaster University, Hamilton, Ontario, Canada.

Chronic exposure to air pollution is associated with increased mortality rates. The impact of air pollution relative to other causes of death in a population is of public health importance and has not been well established. In this study, the rate advancement periods associated with traffic pollution exposures were estimated. Study subjects underwent pulmonary function testing at a clinic in Hamilton, Ontario, Canada, between 1985 and 1999. Cox regression was used to model mortality from all natural causes during 1992–2001 in relation to lung function, body mass index, a diagnosis of chronic pulmonary disease, chronic ischemic heart disease, or diabetes mellitus, household income, and residence within 50 m of a major urban road or within 100 m of a highway. Subjects living close to a major road had an increased risk of mortality (relative risk = 1.18, 95% confidence interval: 1.02, 1.38). The mortality rate advancement period associated with residence near a major road was 2.5 years (95% confidence interval: 0.2, 4.8). By comparison, the rate advancement periods attributable to chronic pulmonary disease, chronic ischemic heart disease, and diabetes were 3.4 years, 3.1 years, and 4.4 years, respectively.

air pollution; automobiles; mortality; urbanization; vehicles

There is increasing evidence that long-term exposure to the particulate and gaseous air pollution common to many metropolitan areas is an important risk factor for mortality. The strongest evidence comes from an updated study of mortality in approximately 550,000 adults who were enrolled in the American Cancer Society's Cancer Prevention Study II. During 16 years of follow-up, Pope et al. (1) found that fine particulate- and sulfur oxide-related pollution was associated with all-cause, lung cancer, and cardiopulmonary mortality. In a Dutch study, Hoek et al. (2) reported that proximity to major roads produced a larger association with cardiopulmonary mortality than did urban background concentrations of air pollution.

Although these studies and the earlier Six Cities Study (3) demonstrated a relation between air pollution and mortality rates, the impact of air pollution relative to other causes of death in a population has not been well established. This relation is of public health importance. Pope et al. (1) compared the mortality effect of fine particulate air pollution with that of cigarette smoking and obesity. They concluded that the risk associated with exposure to particulate air pollution was much smaller than the risk of cigarette smoking but was comparable to the estimated effect of being moderately overweight.

One measure of the impact of a risk factor on mortality is the "rate advancement period" developed by Brenner et al. (4). The rate advancement period estimates the impact of a risk factor on the *timing* of death; that is, the risk is phrased in terms of a premature mortality rate among exposed persons. In this study, we estimated the rate advancement period associated with intraurban traffic pollution exposures in a cohort of subjects in the city of Hamilton, Ontario, Canada.

Correspondence to Dr. Murray Finkelstein, Family Medicine Centre, Suite 413, Mt. Sinai Hospital, Toronto, Ontario, Canada M5G 1X5 (e-mail: murray.finkelstein@utoronto.ca).

MATERIALS AND METHODS

Study subjects and mortality follow-up

The study subjects were residents of the Hamilton metropolitan area who underwent pulmonary function testing between 1985 and 1999 at a Hamilton clinic. These included persons referred for specialist assessment and persons referred for spirometric testing only. Information available included the subject's Ontario Health Insurance Number and postal code, the subject's body mass index (weight (kg)/height (m)^2), and the results of pulmonary function tests, including forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV$_1$). Predicted FVC and FEV$_1$ were computed using the equations of Hankinson et al. (5). Smoking histories were not recorded.

The province of Ontario provides universal health insurance for all residents. A family-based Medicare number was replaced with a personal Health Insurance Number in 1992. This Health Insurance Number was used to link subjects to Ontario Health Insurance Plan physician billing files for 1992–1999.

Subjects entered mortality follow-up on January 1, 1992, or the date of their first clinic visit, whichever was later. To ensure that subjects without a visit after January 1, 1992, were still alive at the start of follow-up, we excluded subjects without an Ontario Health Insurance Plan transaction after 1991. Mortality was ascertained by linkage to the Ontario Mortality Registry for the period 1992–2001. Because there were only 27 deaths among younger subjects, this study was restricted to subjects aged 40 years or older. Subjects diagnosed with cancer, either before or within 45 days of the initial visit, were excluded. This left 5,228 subjects (950 total deaths, 923 nonaccidental) available for analysis. This study was approved by the Research Ethics Board at St. Joseph's Healthcare, Hamilton.

Underlying medical diagnoses

We used the Ontario Health Insurance Plan linkage to classify underlying respiratory disease status, utilizing diagnostic codes submitted to the Ontario Health Insurance Plan by specialist physicians. Diagnoses included chronic obstructive pulmonary disease (*International Classification of Diseases*, Ninth Revision (ICD-9), codes 491, 492, and 496: 2,035 subjects, 431 deaths), asthma (ICD-9 code 493: 604 subjects, 28 deaths), bronchiectasis (ICD-9 code 494: 141 subjects, 31 deaths), asbestosis (ICD-9 code 501: 39 subjects, four deaths), silicosis (ICD-9 code 502: 24 subjects, three deaths), and postinflammatory pulmonary fibrosis (ICD-9 code 515: 240 subjects, 75 deaths). Subjects with diagnoses of both asthma and chronic obstructive pulmonary disease were classified as having chronic obstructive pulmonary disease.

The Ontario Health Insurance Plan billing file and the Ontario hospital discharge database were also searched for diagnoses of diabetes mellitus (ICD-9 code 250) and chronic ischemic heart disease (ICD-9 codes 412–414). Subjects were classified as having these disorders if the diagnosis had been made in two or more claims submissions by a general practitioner, in one claim submission by a specialist, or in

any hospitalization. This algorithm has been validated for diagnosis of diabetes in Ontario (6).

Income and road traffic indicators

Postal codes were used to estimate household income on the scale of the enumeration area, the smallest unit in the Canadian census. They were also used to assign residential addresses to road traffic buffers. Canadian six-character postal codes are localized to small areas in urban regions, and the domain included may be as small as a single apartment building or city block. The ArcView 3.2 geographic information system (ESRI, Inc., Redlands, California) was used with the Statistics Canada 1999 postal code conversion file and the Census of 1996 to estimate mean household income in the subjects' neighborhoods by overlay of enumeration area data. The geographic information system was also used to map subjects' residences with respect to major sources of road traffic pollution. We used the same indicator of traffic-related pollution as Hoek et al. (2). Subjects' postal codes were mapped with respect to buffers of 50 m around major urban roads and 100 m from highways (road/highway buffers).

Modeling of mortality relative risk

The Cox proportional hazards model (7) was used to model the risk of mortality from all natural causes. We tested for deviations from the proportional hazards assumption by introducing interactions of time with all covariates. To account for possible geographic "clustering" of mortality, we grouped subjects among census tracts ($n = 120$ tracts; mean number of subjects per census tract = 59 (range, 1–224)), and we adjusted estimates of variance for clustering in census tracts. Calculations were performed with Stata statistical software (8).

Rate advancement periods

The effect of traffic-related pollution with respect to the time dimension of premature mortality was assessed using Brenner et al.'s (4) "rate advancement period," which estimates how much older a reference population would have to be to experience the same "attrition rate" as subjects residing within a road buffer. The rate advancement period is derived from a linear model of the form $\log(\text{hazard}) = b_1 \times \text{exposure} + b_2 \times \text{age} + \text{covariates}$. From this model, a point estimate of the rate advancement period is b_1/b_2 per unit increase in exposure. A formula for the variance is given in the paper by Brenner et al. (4).

Additional deaths per year

Rates of mortality from all natural causes are dependent on age. To compute the "additional deaths per year" attributable to traffic pollutant exposure, we added the rate advancement period to chronologic age and performed interpolation in Ontario life tables. We subtracted the mortality rate at a given reference age from the rate at the "rate-advanced" age to obtain the number of additional all-natural-cause deaths

Am J Epidemiol 2004;160:173–177

TABLE 1.   Characteristics of subjects exposed to traffic air pollution and control subjects in relation to distance of residence from major roads and highways, Hamilton, Ontario, Canada, 1992–2001

| Variable | Control subjects (>100 m from a highway *and* >50 m from a major road) | | | | Exposed subjects (within a road/highway buffer (<50 m from a major road or <100 m from a highway)) | | | |
| | (*n* = 4,280) | | | | (*n* = 948) | | | |
	No.	%	Median	IQR*	No.	%	Median	IQR
Male gender	1,905	45			401	42		
Age (years)			60.4	49.8–70.0			63.6	51.7–72.4
Annual household income (thousands of Canadian dollars)			48.3	36.5–61.1			39.1	30.7–52.2
Forced vital capacity (% predicted)			84	70–97			81	67–94
Forced expiratory volume in 1 second (% predicted)			79	55–96			73	52–91
Body mass index†			27.8	24.4–31.6			27.6	24.4–31.6
Diagnosed with asthma	471	11			85	9		
Diagnosed with chronic pulmonary disease (except asthma)	1,753	41			432	46		
Diagnosed with chronic ischemic heart disease	1,372	32			318	34		
Diagnosed with diabetes mellitus	695	16			145	15		
Nonaccidental death	706				217			

* IQR, interquartile range (25th–75th percentile).
† Weight (kg)/height (m)^2.

per 1,000 subjects per year, adjusted for clinical and disease variables.

RESULTS

Study subjects and pollutants

The distribution of subjects by road buffer residence classification is shown in table 1, in conjunction with mean values for clinical variables. Approximately half of the subjects in this cohort of patients from a lung function laboratory did not have a chronic respiratory disease diagnosed by a specialist.

Mortality in relation to clinical variables, disease status, household income, and residence near major roads

Table 2 shows the results from Cox models for all-natural-cause mortality in relation to physiologic and disease variables, income, and residence within a traffic pollution buffer. To explore risk modification by respiratory disease status, we conducted analyses separately for subjects with and without diagnoses of chronic pulmonary disease. The proportional hazards hypothesis was not rejected for any of the models.

As has been observed in other studies, mortality rates were lower among subjects with higher FVC and FEV_1 (percent predicted) (9); there was a quadratic relation between mortality rates and body mass index (10); and mortality rates were lower in households with higher incomes (11). Subjects

residing within traffic pollution buffers had elevated mortality rates regardless of whether they had been diagnosed with chronic pulmonary disease (excluding asthma). A test for effect modification by chronic pulmonary disease did not give a significant result ($p = 0.77$). The final column of table 2 shows that after adjustment for clinical variables and chronic disease diagnoses, the relative risk for all-natural-cause mortality among subjects residing within a road buffer was 1.18 (95 percent confidence interval (CI): 1.02, 1.38).

Rate advancement periods

We used the formulae in the paper by Brenner et al. (4) to compute the rate advancement periods and their variances. The formula for the rate advancement period requires a correct statement of the dependency between log(hazard) and age. The hypothesis of linearity between log(hazard) and age was not rejected after examination of the martingale residuals (7). Table 3 shows the mortality rate advancement period attributable to residence within a traffic pollution buffer. Rate advancement periods attributable to diagnoses of chronic disease are presented for comparison.

The rate advancement period associated with traffic pollution was 2.5 years (95 percent CI: 0.2, 4.8). By way of comparison, the rate advancement periods attributable to chronic pulmonary diseases other than asthma, to chronic ischemic heart disease, and to diabetes were 3.4 years, 3.1 years, and 4.4 years, respectively. Thus, the "aging effect"

TABLE 2. Relative risk of mortality from all natural causes (*n* = 923) during follow-up in relation to clinical measures, chronic disease diagnoses, household income, and pollutant exposures, Hamilton, Ontario, Canada, 1992–2001

Variable	Diagnosed with CPD*		Not diagnosed with CPD		All subjects	
	RR*	95% CI*	RR	95% CI	RR	95% CI
Female gender	0.62	0.52, 0.74	0.28	0.20, 0.41	0.52	0.44, 0.61
Age (per year above 40)	1.06	1.05, 1.08	1.07	1.06, 1.08	1.07	1.06, 1.08
Forced vital capacity (% predicted—per 1%)	0.987	0.980, 0.993	0.991	0.981, 1.001	0.986	0.980, 0.992
Forced expiratory volume in 1 second (% predicted—per 1%)	0.993	0.987, 0.999	0.985	0.977, 0.995	0.993	0.988, 0.998
Body mass index† (per unit)	0.93	0.91, 0.95	0.95	0.92, 0.99	0.94	0.92, 0.95
Body mass index squared	1.003	1.001, 1.004	1.001	0.999, 1.003	1.002	1.001, 1.003
CPD					1.26	1.05, 1.49
Asthma			0.68	0.48, 0.96	0.67	0.48, 0.94
Chronic ischemic heart disease	1.12	0.97, 1.30	1.55	1.20, 2.01	1.23	1.06, 1.42
Diabetes mellitus	1.36	1.13, 1.62	1.32	0.97, 1.78	1.34	1.13, 1.59
Log(household income)	0.88	0.72, 1.07	0.74	0.48, 1.14	0.84	0.69, 1.03
Residence within a road/highway buffer	1.18	0.98, 1.41	1.27	0.92, 1.75	1.18	1.02, 1.38

* CPD, chronic pulmonary disease (excluding asthma); RR, relative risk; CI, confidence interval.
† Weight (kg)/height (m)^2.

attributable to traffic pollution was only slightly lower than the aging effect associated with these chronic diseases.

By interpolation in Ontario life tables, we estimated the number of deaths attributable to an "aging effect" of 2.5 years. The estimates were 0.4 additional deaths/1,000/year among subjects aged 40 years, 1.6 deaths/1,000/year among subjects aged 50 years, 4.4 deaths/1,000/year among subjects aged 60 years, and 10.9 deaths/1,000/year among subjects aged 70 years.

DISCUSSION

We have confirmed Hoek et al.'s observation (2) that rates of mortality from all natural causes vary in association with

TABLE 3. Rate advancement period for mortality from all natural causes in relation to residence close to a major road and common chronic diseases, Hamilton, Ontario, Canada, 1992–2001

Risk factor	RAP*,† (years)	95% CI*
Residence within a road/highway buffer	2.5	0.2, 4.8
Diagnosis of chronic pulmonary disease (excluding asthma)	3.4	0.8, 6.0
Diagnosis of chronic ischemic heart disease	3.1	0.8, 5.4
Diagnosis of diabetes mellitus	4.4	1.8, 7.0

* RAP, rate advancement period; CI, confidence interval.
† The rate advancement period is the number of years older the comparison subjects would have to be in order to have the same attrition rates as subjects with the indicated risk factor exposure.

residential proximity to traffic pollution. The importance of pollution with respect to mortality and other health outcomes in a population is meaningful for cost-benefit analyses of interventions designed to lower pollution exposures. There is a consensus that these assessments should not rely on the results of time-series studies but rather should be based on long-term follow-up in cohort studies (12, 13). Our cohort comprised subjects who underwent lung function testing. We found that there was no risk modification by respiratory disease and that the relative risk associated with residential proximity to traffic was the same for subjects with and without physician-diagnosed chronic respiratory disease.

Use of the rate advancement period provides one view of the impact of increased mortality rates. Since interpretation of the rate advancement period is conditional on the absence of competing causes of death (4), we restricted our analysis to mortality from all natural causes and did not consider specific underlying causes, such as cardiopulmonary diseases, which are most causally linked to air pollutant exposures (1). The use of all-natural-cause mortality provides a global assessment of mortality impact. We found that the "aging effect" attributable to traffic pollution exposure was 2.5 years, which is similar to the rate advancement period attributable to common chronic diseases. We emphasize that the rate advancement period does not equate directly with "years of life lost" but rather indicates the advancement of mortality *rates*. The extent of life shortening will depend on the ages of the subjects.

A weakness of our study is that we have, as yet, no measurements of pollutant levels within the road buffers. In North American cities, pollution is generated by gasoline-powered cars and diesel-fueled buses and trucks. The

burning of fossil fuels creates primary pollutants, including carbon dioxide, carbon monoxide, oxides of nitrogen, sulfur dioxide, hydrocarbons, and airborne particulates. It has been estimated that particulate emission from diesel engines, per distance traveled, is over 100 times higher than that from gasoline engines fitted with catalytic converters (14). Dutch investigators (15) reported that daytime concentrations of particulate matter less than 10 μm in aerodynamic diameter were, on average, 30 percent higher near busy roads than at background locations, differing by 7–13 μg/m^3. Researchers in another Dutch study reported that indoor and outdoor particulate concentrations were higher at homes located on high-traffic-intensity streets than at homes on low-traffic streets (16). The mean indoor concentration of particulate matter less than 2.5 μm in aerodynamic diameter was 27 μg/m^3 in high-traffic homes and 12 μg/m^3 in low-traffic homes. In Roxbury, Massachusetts (17), concentrations of fine particulate matter and particle-bound polycyclic aromatic hydrocarbons were higher on roads reported to have heavy bus traffic. In the South Bronx (New York City), sidewalk measures of diesel exhaust particulates were correlated with the count of large trucks (18).

As in the other studies of air pollution cohorts, we did not have a measure of residential mobility during the follow-up period, so misclassification of traffic exposure was possible. Similar to several of the other air pollution cohorts, our cohort was not a random sample of the population. The cohort in the Six Cities Study (3) was a random sample of 8,111 White adults. Participants in the American Cancer Society study (1) were friends, neighbors, or acquaintances of American Cancer Society volunteers. In the Dutch study by Hoek et al. (2), only 36 percent of potential subjects completed the baseline questionnaire.

Our subjects underwent lung function testing at an academic clinic. Although only half of the subjects were diagnosed with respiratory disease, they presumably had respiratory symptoms such as cough and shortness of breath prior to referral. Although our cohort contained a greater proportion of persons with respiratory illness than is found in the general population, we believe that our results can be generalized because of the availability of clinical diagnoses. To our knowledge, this was the first cohort study with access to physicians' diagnoses of underlying disease status. Thus, we were able to adjust for diagnoses of chronic respiratory and pulmonary diseases and diabetes. We found no effect modification by underlying respiratory disease status; and our adjusted estimate of the all-natural-cause mortality risk associated with residential exposure to traffic was 1.18 (95 percent CI: 1.02, 1.38), similar to the Dutch estimate of 1.35 (95 percent CI: 0.93, 1.95) (2).

In conclusion, we found an association between residential proximity to traffic and mortality. The mortality rate advancement attributable to traffic pollution was similar to that associated with chronic respiratory and pulmonary diseases and diabetes. This suggests that decreasing pollutant exposures may have a substantial public health impact.

REFERENCES

1. Pope CA 3rd, Burnett RT, Thun MJ, et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 2002;287:1132–41.
2. Hoek G, Brunekreef B, Goldbohm S, et al. Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study. Lancet 2002;360:1203–9.
3. Dockery DW, Pope CA 3rd, Xu X, et al. An association between air pollution and mortality in six U.S. cities. N Engl J Med 1993;329:1753–9.
4. Brenner H, Gefeller O, Greenland S. Risk and rate advancement periods as measures of exposure impact on the occurrence of chronic diseases. Epidemiology 1993;4:229–36.
5. Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general U.S. population. Am J Respir Crit Care Med 1999;159:179–87.
6. Hux JE, Ivis F, Flintoft V, et al. Diabetes in Ontario: determination of prevalence and incidence using a validated administrative data algorithm. Diabetes Care 2002;25:512–16.
7. Hosmer DW, Lemeshow S. Applied survival analysis. New York, NY: John Wiley and Sons, Inc, 1999.
8. Stata Corporation. Stata, version 8.2. College Station, TX: Stata Corporation, 2003.
9. Neas LM, Schwartz J. Pulmonary function levels as predictors of mortality in a national sample of US adults. Am J Epidemiol 1998;147:1011–18.
10. Song YM, Sung J. Body mass index and mortality: a twelve-year prospective study in Korea. Epidemiology 2001;12:173–9.
11. Anderson RT, Sorlie P, Backlund E, et al. Mortality effects of community socioeconomic status. Epidemiology 1997;8:42–7.
12. Kunzli N, Medina S, Kaiser R, et al. Assessment of deaths attributable to air pollution: should we use risk estimates based on time series or on cohort studies? Am J Epidemiol 2001;153:1050–5.
13. McMichael AJ, Anderson HR, Brunekreef B, et al. Inappropriate use of daily mortality analyses to estimate longer-term mortality effects of air pollution. Int J Epidemiol 1998;27:450–3.
14. Sydbom A, Blomberg A, Parnia S, et al. Health effects of diesel exhaust emissions. Eur Respir J 2001;17:733–46.
15. Janssen NA, van Mansom DF, van der Jagt K, et al. Mass concentration and elemental composition of airborne particulate matter at street and background locations. Atmos Environ 1997;31:1185–93.
16. Fischer PH, Hoek G, van Reeuwijk H, et al. Traffic-related differences in outdoor and indoor concentrations of particles and volatile organic compounds in Amsterdam. Atmos Environ 2000;34:3713–22.
17. Levy JI, Houseman EA, Spengler JD, et al. Fine particulate matter and polycyclic aromatic hydrocarbon concentration patterns in Roxbury, Massachusetts: a community-based GIS analysis. Environ Health Perspect 2001;109:341–7.
18. Lena TS, Ochieng V, Carter M, et al. Elemental carbon and PM(2.5) levels in an urban community heavily impacted by truck traffic. Environ Health Perspect 2002;110:1009–15.