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Original Contribution

Association of Long-term Exposure to Community Noise and Traffic-related Air Pollution With Coronary Heart Disease Mortality

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In metropolitan areas, road traffic is a major contributor to ambient air pollution and the dominant source of community noise. The authors investigated the independent and joint influences of community noise and traffic-related air pollution on risk of coronary heart disease (CHD) mortality in a population-based cohort study with a 5-year exposure period (January 1994–December 1998) and a 4-year follow-up period (January 1999–December 2002). Individuals who were 45–85 years of age and resided in metropolitan Vancouver, Canada, during the exposure period and did not have known CHD at baseline were included ($n = 445{,}868$). Individual exposures to community noise and traffic-related air pollutants, including black carbon, particulate matter less than or equal to 2.5 μm in aerodynamic diameter, nitrogen dioxide, and nitric oxide, were estimated at each person's residence using a noise prediction model and land-use regression models, respectively. CHD deaths were identified from the provincial death registration database. After adjustment for potential confounders, including traffic-related air pollutants or noise, elevations in noise and black carbon equal to the interquartile ranges were associated with 6% (95% confidence interval: 1, 11) and 4% (95% confidence interval: 1, 8) increases, respectively, in CHD mortality. Subjects in the highest noise decile had a 22% (95% confidence interval: 4, 43) increase in CHD mortality compared with persons in the lowest decile. These findings suggest that there are independent effects of traffic-related noise and air pollution on CHD mortality.

air pollution; cohort studies; coronary heart disease; environmental exposure; mortality; transportation noise; vehicle emissions

Abbreviations: CHD, coronary heart disease; CI, confidence interval; L_{den} dB(A), annual day-evening-night A-weighted equivalent continuous noise level; MI, myocardial infarction; $PM_{2.5}$, particulate matter less than or equal to 2.5 μm in aerodynamic diameter; SES, socioeconomic status.

Epidemiologic evidence has demonstrated that air pollution is associated with increased cardiovascular disease (especially coronary heart disease (CHD) morbidity and mortality) (1). Meanwhile, accumulating evidence has suggested that community noise from road and air traffic is associated with an increased risk of CHD, especially myocardial infarction (MI) (2–5). In metropolitan areas, road traffic is a major contributor to ambient air pollution and the dominant source of community noise (6–9). Persons exposed to higher levels of air pollution might also be exposed to excessive traffic noise (6, 7, 10). Therefore, it is possible that the observed associations between air pollution and adverse cardiovascular outcomes could be confounded by community noise and vice versa (6, 7). Furthermore, these coexistent environmental pollutants might interact with each other in association with coronary mortality (6).

In a previous study in metropolitan Vancouver, Canada (11), we found that living close to a major road was associated with a 29% (95% confidence interval (CI): 18, 41) increase in the risk of death from CHD. We further examined the associations between death from coronary disease and 4 major traffic-related air pollutants, including black carbon, particulate matter less than or equal to 2.5 μm in aerodynamic diameter ($PM_{2.5}$), nitrogen dioxide, and nitric oxide, and found that black carbon, an indicator of traffic-related fine particulate air pollution, was associated with a 6% (95% CI: 3, 9) increase in the risk of death from CHD. No robust associations were found with $PM_{2.5}$, nitrogen dioxide, or nitric oxide (12).

These findings suggested that exposure to traffic-related air pollutants cannot fully explain the higher risk of death from CHD associated with residential proximity to road traffic; traffic noise might also play a role in the observed association. We therefore investigated the relations between long-term exposure to community noise and CHD mortality, as well as the joint influences of community noise and traffic-related air pollution (black carbon) on the risk of CHD mortality.

MATERIALS AND METHODS

Study design

In British Columbia, Canada, the mandatory health insurance program provides health care coverage for nearly all residents (13). We used linked administrative health insurance databases to assemble this population-based cohort (11, 12, 14). The present study included a 5-year exposure period (January 1994–December 1998) and a 4-year follow-up period (January 1999–December 2002). All metropolitan Vancouver residents who had registered with the provincial health insurance plan, had resided in the study region during the 5-year exposure period, were 45–85 years of age, and had no previous diagnosis of CHD at baseline (January 1999) were included in the cohort.

During the 5-year exposure period, individual exposures to community noise and traffic-related air pollutants were estimated at each person's residence (residential postal code) using noise prediction and land-use regression models, respectively. During the 4-year follow-up period, instances of CHD death were identified from the provincial death registration database. The associations of CHD mortality with noise and black carbon were examined using the Cox proportional hazards regression model. This study was approved by the institutional review board of The University of British Columbia (Behavioural Research Ethics Board certificate #H08-00185).

Noise exposure assessment

We used the noise prediction software CadnaA (Datakustik, Greifenberg, Germany) to estimate annual average community noise levels at each person's residence (residential postal code) in 2003. The method has been described in detail elsewhere (15). Briefly, noise exposure was based on transportation-related information, including road traffic data (e.g., speed limits, traffic volume, fleet composition, and road width), railway data (e.g., type of train, velocity, and frequency), and building heights and footprints. Aircraft noise data were obtained from aircraft noise exposure forecasts produced by Vancouver International Airport Authority. On the basis of these data, the annual day-evening-night A-weighted equivalent continuous noise level was calculated for each area covered by a 6-digit postal code. This metric (L_{den} dB(A), hereafter referred to as dB(A)) integrated noise levels during the day, evening, and night, with a 5-dB(A) weighting added to evening noise and a 10-dB(A) weighting added to night noise to reflect increased sensitivity of residents to noise during these periods (16, 17). Railways were a minor contributor to overall community noise in this region, and thus we did not separately assess railway noise exposure.

Air pollution exposure assessment

We used high-spatial-resolution land-use regression models to estimate participants' residential exposures to traffic-related air pollutants, including black carbon, $PM_{2.5}$, nitrogen dioxide, and nitric oxide, in 2003 (18–20). Furthermore, these estimates were combined with air quality-monitoring data to calculate monthly concentrations and average concentrations during the 5-year exposure period for each pollutant in each postal code area (11, 12).

Assignment of exposure data

Because our exposure assessment did not cover the whole study region, a small proportion of study subjects for whom we were missing data were excluded. Some subjects had partially missing data because of changes in residences (moving from or to areas outside the exposure assessment domain); persons missing data for more than a total of 15 months or in more than 3 consecutive months during the exposure period were also excluded. For subjects who changed their residences, we calculated equivalent noise levels and average air pollution levels during the exposure period. The noise and air pollution data were assigned to study subjects based on their 6-digit residential postal codes. In urban areas, a residential postal code typically represents a high-rise building or one side of a city block; in rural areas, it may represent a larger area. Metropolitan Vancouver is highly urbanized; the vast majority of the postal codes represent a small geographic area. On average, a residential postal code includes about 35 persons.

Case definitions

The study outcome was death from CHD during the 4-year follow-up period, defined as having *International Classification of Diseases* codes 410–414 and 429.2 (Ninth Revision) and I20–I25 (Tenth Revision) listed as the cause of death in the death registration database (see Web Table 1 (available at http://aje.oxfordjournals.org/) for the codes for other cardiovascular diseases). Subjects who were hospitalized with CHD as the principal diagnosis (the diagnosis most responsible for a hospital admission) or primary diagnosis (the diagnosis that had a substantial influence on hospital length of stay) before baseline (based on data from 1991 to 1998) were regarded as having previously diagnosed CHD and were excluded from the analyses.

Preexisting comorbid conditions

Diabetes (21), chronic obstructive pulmonary disease (22, 23), and hypertensive heart disease (21) are independent risk factors for CHD. Additionally, these chronic diseases and CHD share common behavioral risk factors. As in our previous analyses (11, 12), we used these preexisting comorbid conditions as proxy variables for common behavioral cardiovascular risk factors (24). To sufficiently identify subjects with preexisting comorbid conditions, we used all diagnoses in a hospitalization record (up to 16 diagnoses); one hospitalization record with the diagnosis of any of these chronic diseases during 1991–1998 was defined as the presence of preexisting comorbid conditions.

Neighborhood socioeconomic status

Because individual socioeconomic status (SES) data were not available, we used neighborhood SES to estimate individual SES (25, 26). Study subjects were assigned neighborhood-income quintiles from the 2001 Statistics Canada Census based on their residential postal codes. The method for calculation of neighborhood income quintiles has been described previously (11).

Statistical analysis

The baseline characteristics of the study subjects across deciles of noise levels were compared using a chi-squared test for categorical variables, 1-way analysis of variance for continuous variables, and Tukey's post hoc analysis for pair-wise comparisons of continuous variables. Correlations between pollutants were examined using Spearman's rank correlation analysis.

The Cox proportional hazards regression model was used to determine the associations between noise or air pollution and CHD mortality. Age, sex, preexisting comorbid conditions, and neighborhood SES were included as covariates, and air pollution and noise variables were added to the final models. Person-years of observation were calculated from baseline to the date of CHD death or the end of follow-up. For persons who died from other diseases or moved out of the province, person-years were calculated from baseline to the date of death or the last known date in the province.

We first treated noise levels as a continuous variable to calculate the relative risks of CHD mortality associated with a 10-dB(A) elevation in noise levels. We then treated noise levels as a categorical variable to examine exposure-response relations by dividing study subjects into deciles based on noise levels; relative risks of death from coronary disease were calculated for each decile, with decile 1 (lowest) being the reference category. Because there was no substantial difference in effect estimates across deciles 2–9 (Web Figure 1), the results were presented in 4 exposure groups: decile 1, deciles 2–5, deciles 6–9, and decile 10. All statistical tests were 2-sided and were performed using SAS, version 9.2 (SAS Institute, Inc., Cary, North Carolina).

RESULTS

A total of 466,727 subjects who met the inclusion criteria were included at baseline. Of these subjects, we excluded 20,859 (4.5%) subjects for whom data on air pollution or noise were missing, which left 445,868 subjects for the analyses. During the follow-up period, 33,448 (7.5%) subjects were lost to follow-up; the reasons included moving out of the province (3.9%) and dying from other diseases (3.6%).

Overall, the annual average noise level was 63.4 dB(A) (interquartile range: 59.8–66.4). Noise levels were not strongly correlated with traffic-related air pollutant concentrations; traffic-related air pollutants were weakly correlated with each other, with the exception of nitrogen dioxide and nitric oxide (Table 1). Compared with persons exposed to lower noise levels (decile 1), subjects in decile 10 were more likely to have preexisting comorbid conditions and lower neighborhood SES (Table 2).

During the follow-up period, 3,095 subjects died of CHD (mortality rate = 1.83 per 1,000 person-years). Residential noise exposure was associated with CHD mortality: A 10-dB(A) elevation in noise levels was associated with a 26% (95% CI: 17, 35) increase in the risk of CHD mortality. Adjustment for age, sex, preexisting comorbid conditions, and neighborhood SES halved the effect estimate, whereas further adjusting for $PM_{2.5}$ and nitrogen dioxide concentrations had little influence; additional adjustment for back carbon levels had a greater influence on the effect estimate, but a 10-dB(A) elevation in noise levels was still associated with a 9% (95% CI: 1, 18) increase in the risk of death from CHD (Table 3). Web Table 2 presents results based on a 5-dB(A) increase in noise exposure. For other cardiovascular diseases, there were nonsignificant increases in mortality rates associated with noise exposure (Web Table 3). The associations between black carbon and cardiovascular disease mortality are presented in Web Table 4.

When study subjects were categorized into noise deciles, subjects in deciles 2–5 and deciles 6–9 had little increase in the risk of death from CHD compared with persons in decile 1, whereas persons in decile 10 had a 22% (95% CI: 4, 43) increase in coronary mortality risk after adjustment for all covariates, including traffic-related air pollutants. These results suggested that there was no linear exposure-response relation between noise and coronary mortality ($P = 0.174$ for test of linear trend across decile groups in the fully adjusted model) (Table 3).

Both noise and black carbon exposure were independently associated with death from CHD (Figure 1); elevations equal to the interquartile range in noise (6.6 dB(A)) and black carbon (0.97×10^{-5}/m) were associated with 6% (95% CI: 1, 11) and 4% (95% CI: 1, 8) increases in coronary mortality, respectively. We did not find any positive interaction between noise and black carbon on risk of coronary mortality when they were assessed on either additive (Table 4) or multiplicative ($P = 0.980$ for the interaction term in the fully adjusted model) scales.

Stratified analysis showed that in the fully adjusted models, the risk of death from CHD associated with noise exposure was greater for women, persons 65 years of age or older, and persons with higher neighborhood SES. However, there was considerable overlap in the 95% confidence intervals of these subgroups (Table 5).

For persons exposed to aircraft noise ($n = 294{,}783$), the annual average noise level was 32 dB(A). Aircraft noise was less correlated with traffic-related air pollutants than was road traffic noise (Web Table 5). There was no significant increase in the risk of death from CHD associated with exposure to aircraft noise (Web Table 6).

DISCUSSION

In the present large population-based cohort study, we found that a 10-dB(A) elevation in residential noise levels was associated with a 9% increase in CHD mortality after adjustment for various covariates, including traffic-related air pollutants. There was no discernible linear exposure-response relation; persons in the highest noise decile (>70 dB(A)) had a 22% increase in the risk of death from CHD compared with

Table 1. Annual Average Noise Levels, 5-Year Average Traffic-related Air Pollutant Concentrations, and Correlation Coefficients, Metropolitan Vancouver, Canada, 1994–2002

Pollutant	Mean (SD)	Median (Interquartile Range)	Range	Correlation Coefficient*				
				Noise	Black Carbon	$PM_{2.5}$	Nitrogen Dioxide	Nitric Oxide
Noise, L_{den} dB(A)	63.4 (5.0)	62.4 (59.8–66.4)	33.0–90.0	1.00				
Black carbon, 10^{-5}/m	1.50 (1.10)[a]	1.02 (0.83–1.80)	0.0–4.98	0.44	1.00			
$PM_{2.5}$, $\mu g/m^3$	4.10 (1.64)	4.04 (3.22–4.81)	0.0–10.24	0.14	0.13	1.00		
Nitrogen dioxide, $\mu g/m^3$	32.3 (8.1)	30.8 (26.7–35.2)	15.3–57.5	0.33	0.39	0.47	1.00	
Nitric oxide, $\mu g/m^3$	32.2 (12.0)	29.5 (24.3–37.6)	8.8–126.0	0.39	0.43	0.43	0.66	1.00

Abbreviations: L_{den} dB(A), annual day-evening-night A-weighted equivalent continuous noise level; $PM_{2.5}$, particulate matter less than or equal to 2.5 μm in aerodynamic diameter; SD, standard deviation.

* $P < 0.001$ for each correlation coefficient.

[a] Equivalent to 1.20 (0.88) $\mu g/m^3$ elemental carbon (10^{-5}/m black carbon $\approx$ 0.8 $\mu g/m^3$ elemental carbon).

persons in the lowest noise decile (≤58 dB(A)). Also in the fully adjusted model, an increase in the concentration of black carbon equal to the interquartile range (0.97×10^{-5}/m; equivalent to approximately 0.8 $\mu g/m^3$ elemental carbon) was associated with a 4% increase in the risk of death from CHD. We did not detect any interaction between noise and black carbon with relation to coronary mortality. In persons exposed to aircraft noise, we did not find a significant increase in the risk of death from CHD compared with unexposed subjects.

Findings from previous studies have been inconsistent, but most studies have shown positive associations between community noise and coronary events. In a 9-year Dutch cohort study, Beelen et al. (27) reported that cardiovascular mortality increased 17% (95% CI: −6, 45) for persons exposed to higher levels of road traffic noise (>65 dB(A) vs. ≤50 dB(A)) and 11% (95% CI: −5, 28) in response to a 10-$\mu g/m^3$ increase in black smoke concentrations (about 1.1 $\mu g/m^3$ elemental carbon) (28); however, there was no discernible increase in CHD mortality rates (27). In a case-control study in Stockholm County, Selander et al. (3) found that exposure to higher levels of road traffic noise (≥50 dB(A) vs. <50 dB(A)) was associated with a 12% (95% CI: −5, 33) increase in MI risk after adjustment for nitrogen dioxide and other cardiovascular risk factors. After further excluding persons with hearing loss or other sources of noise exposure, the risk of MI increased by 38% (95% CI: 11, 71) (3). In a 5-year Swiss cohort study of 4.6 million subjects and 65 airports, Huss et al. (4) found that people exposed to higher levels of aircraft noise (≥60 dB(A) vs. <45 dB(A)) had a 30% (95% CI: −4, 76) increase in MI mortality after adjusting

Table 2. Baseline Characteristics of Study Subjects by Decile of Noise Level, Metropolitan Vancouver, Canada, 1994–2002*

Characteristic	Decile of Noise Levels, L_{den} dB(A)							
	1 (≤58)		2–5 (59–62)		6–9 (63–70)		10 (>70)	
	%	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%	Mean (SD)
Male sex	46.0		46.6		45.9		45.8	
Age, years		59.3 (10.8)		59.0 (10.6)		59.4 (10.7)		60.0 (10.9)
Comorbid condition								
Diabetes	2.1		2.1		2.4		2.9	
Chronic obstructive pulmonary disease	1.5		1.3		1.5		1.8	
Hypertensive heart disease	4.3		4.0		4.3		4.8	
Any of the above	6.6		6.3		6.8		7.9	
Income quintile[a]								
1	11.8		14.8		21.4		28.3	
2	13.4		18.5		20.0		23.2	
3	17.3		20.3		19.4		17.4	
4	25.3		21.9		18.7		15.3	
5	32.3		24.5		20.4		15.9	

Abbreviations: L_{den} dB(A), annual day-evening-night A-weighted equivalent continuous noise level; SD, standard deviation.

* $P \leq 0.001$ for all comparisons between groups.

[a] Quintile 1 represents the lowest neighborhood income quintile and quintile 5 represents the highest.

Table 3. Relative Risk of Death From Coronary Heart Disease Associated With an Elevation in A-Weighted Equivalent Continuous Noise Level of 10 Decibels or by Decile of Noise Level, Metropolitan Vancouver, Canada, 1994–2002

Model	10-dB(A) Elevation		Decile of Noise Levels, L_{den} dB(A)						
			1 (≤58)	2–5 (59–62)		6–9 (63–70)		10 (>70)	
	RR	95% CI	Referent	RR	95% CI	RR	95% CI	RR	95% CI
1: Unadjusted	1.26	1.17, 1.35	1.00	1.01	0.89, 1.15	1.09	0.96, 1.24	1.49	1.28, 1.73
2: Model 1 + sex and age	1.18	1.10, 1.26	1.00	1.06	0.93, 1.20	1.09	0.96, 1.24	1.39	1.20, 1.61
3: Model 2 + comorbidity and SES	1.13	1.06, 1.21	1.00	1.05	0.92, 1.19	1.06	0.93, 1.20	1.30	1.12, 1.51
4: Model 3 + $PM_{2.5}$	1.13	1.06, 1.21	1.00	1.04	0.91, 1.19	1.05	0.92, 1.20	1.29	1.11, 1.50
5: Model 4 + nitrogen dioxide	1.12	1.05, 1.21	1.00	1.05	0.92, 1.20	1.05	0.92, 1.20	1.28	1.10, 1.50
6: Model 5 + black carbon	1.09	1.01, 1.18	1.00	1.04	0.91, 1.19	1.02	0.89, 1.17	1.22	1.04, 1.43

Abbreviations: CI, confidence interval; L_{den} dB(A), annual day-evening-night A-weighted equivalent continuous noise level; $PM_{2.5}$, particulate matter less than or equal to 2.5 μm in aerodynamic diameter; RR, relative risk; SES, neighborhood socioeconomic status.

for particulate matter less than or equal to 10 μm in aerodynamic diameter, residential proximity to major roads, and other covariates; when the analysis was restricted to persons who had lived in their residences for at least 15 years, the risk of death from MI increased by 48% (95% CI: 1, 118) (4). In some previous studies, investigators did not adjust for coexistent traffic-related air pollution but reported positive associations between noise exposure and CHD risk (2, 29). As for other cardiovascular outcomes, our effect estimates for hemorrhagic stroke and congestive heart failure were largely comparable to those described in prior studies (27, 30). As discussed previously (12), some studies have also reported associations between black carbon and risk of death from CHD, but most of these studies did not consider coexistent community noise (12).

Potential biologic mechanisms for the observed associations have been proposed. Psychological stress has been demonstrated to be an independent risk factor for cardiovascular disease (31–33). Chronic exposure to community noise might cause annoyance, speech interference, sleep disturbance, and psychological stress (15, 16, 34); therefore, chronic noise exposure may serve as a potent environmental stressor that might activate the sympathetic nervous and endocrine systems to release stress hormones, such as noradrenaline and cortisol (5, 35–38). Stress hormones bind with beta-adrenergic receptors in the heart and blood vessels (39), leading to increased myocardial contractility, flow velocity, coronary artery constriction, vulnerable plaque rupture, thrombosis, and subsequent myocardial ischemia or MI (39–42). As discussed previously (12), there is also convincing pathophysiological evidence, such as pulmonary and systemic oxidative stress and inflammation, to support the associations between black carbon and CHD mortality (1).

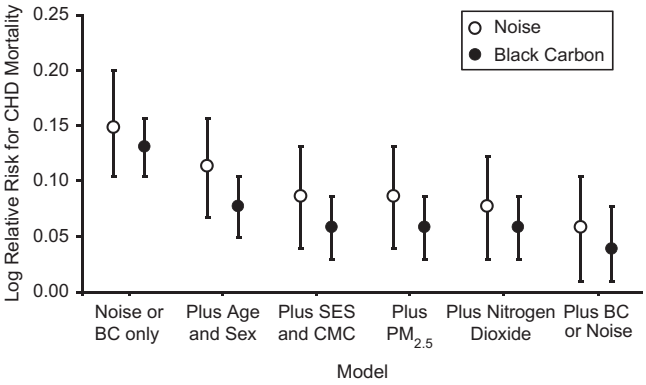

Figure 1. Log relative risks for coronary heart disease (CHD) mortality associated with an elevation equal to the interquartile range in noise levels (A-weighted equivalent continuous noise level of 6.6 decibels) or black carbon (BC) concentrations (0.97×10^{-5}/m), metropolitan Vancouver, 1994–2002. Models were successively adjusted for age and sex; neighborhood socioeconomic status (SES) and comorbid conditions (CMC); particulate matter less than or equal to 2.5 μm in aerodynamic diameter ($PM_{2.5}$) concentrations; and nitrogen dioxide concentrations. The final model for black carbon was further adjusted for noise, and the final model for noise was further adjusted for black carbon. Bars, 95% confidence interval.

In the present study, correlations between modeled community noise and air pollution ranged from 0.14 (for $PM_{2.5}$) to 0.44 (for black carbon), which is within the range of correlations reported in previous studies (6, 7, 10). In practice, some road traffic factors, such as speed, volume, and operating conditions, might differentially affect the emission levels of noise and combustion-derived traffic-related air pollution (43–45). For example, for vehicle speeds less than 30 km/hour, noise levels are lower, but air pollution emissions are relatively higher; however, for vehicle speeds over 40 km/hour, noise levels, mainly from tire-road interaction, rapidly increase, whereas air pollution emissions are relatively lower (43, 44). Furthermore, traffic volume is more strongly related to air pollution than to noise levels. For example, when traffic volume doubles, traffic noise levels increase 3 dB(A) (43). In addition, some environmental factors, such as road pavement materials, noise barriers, and surrounding buildings, have little influence on air pollution emissions but may strongly affect road traffic noise levels, especially when vehicle speeds are over 40 km/hour (43). Finally, meteorological factors, such as wind direction and speed, may strongly affect traffic-related air pollution but have a smaller influence on traffic noise levels (7). Rain and wet road surfaces may increase road traffic noise but may decrease ambient air pollution. All of these factors might partly explain the low-to-moderate correlations between noise and traffic-related air pollution in this study region.

Previous findings about sex differences in the associations between community noise and coronary mortality were

Table 4. Relative Risk of Death From Coronary Heart Disease by Decile of Noise Level and Quartile of Black Carbon Concentration, Metropolitan Vancouver, Canada, 1994–2002[a]

Quartile of Black Carbon Concentration	Decile of Noise Levels, L_{den} dB(A)							
	1 (≤58)		2–5 (59–62)		6–9 (63–70)		10 (>70)	
	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
1: 0–0.83 × 10^{-5}/m	1.00	Referent	1.11	0.88, 1.40	1.12	0.86, 1.45	1.29	0.79, 2.12
2: 0.84–1.02 × 10^{-5}/m	1.05	0.78, 1.41	1.09	0.87, 1.37	1.06	0.84, 1.34	1.24	0.88, 1.74
3: 1.03–1.80 × 10^{-5}/m	1.09	0.79, 1.49	1.23	0.97, 1.55	1.18	0.94, 1.49	1.48	1.09, 2.02
4: 1.81–4.98 × 10^{-5}/m	1.50	1.04, 2.17	1.21	0.93, 1.56	1.23	0.98, 1.54	1.45	1.14, 1.85

Abbreviation: CI, confidence interval; L_{den} dB(A), annual day-evening-night A-weighted equivalent continuous noise level; RR, relative risk.
[a] Adjusted for age, sex, preexisting comorbid conditions, neighborhood income quintiles, and concentrations of copollutants, including nitrogen dioxide and particulate matter less than or equal to 2.5 μm in aerodynamic diameter.

inconsistent. In some studies, men were found to be more vulnerable to noise exposure than were women (2), whereas in other studies, there were no sex differences (3, 27). We found that men and women had similar risks; however, after adjustment for traffic-related air pollutants, including $PM_{2.5}$, nitrogen dioxide, and black carbon, women had a 7% nonsignificant excess risk of coronary mortality compared with men. This observation is supported by findings that women had greater levels of salivary cortisol than did men in response to noise exposure (36, 46).

The present study had some limitations that should be considered. First, exposure assessment was conducted using the residential postal codes of the study subjects to estimate exposure at their residences. This method cannot precisely reflect actual individual exposure (47, 48) because many environmental factors, such as street canyons (49), wind direction (7), noise barriers, and specific housing characteristics (50), as well as individual factors like noise sensitivity (51), time spent at home (47, 52), living room/bedroom orientation (5, 51), and occupational noise exposure (52, 53), might substantially affect actual individual exposure levels. Nevertheless, it is likely that these factors might affect study subjects equally and thus cause nondifferential exposure misclassification, leading to underestimations of the true risk of coronary mortality (48).

Second, this cohort was constructed using administrative health insurance databases. Some individual cardiovascular risk factors were not available, and thus we could not control for them in the data analysis. We adjusted for the following preexisting comorbid conditions: diabetes, chronic obstructive pulmonary disease, and hypertensive heart disease. Because these comorbid conditions and CHD share common behavioral risk factors, the adjustment was able to reduce the influences of unmeasured risk factors and these conditions themselves on the effect estimates to some extent (24); on the other hand, because these conditions might serve as intermediate variables for the associations of coronary mortality with noise and air pollution, the adjustment might cause underestimation of the true adverse effects (5, 54). Furthermore, as discussed previously (11, 12),

Table 5. Relative Risk of Coronary Heart Disease Mortality Associated With an Elevation in A-Weighted Equivalent Continuous Noise Level of 10 Decibels, Stratified by Each Covariate, Metropolitan Vancouver, Canada, 1994–2002

Characteristic	%	Model[a]					
		1		2		3	
		RR	95% CI	RR	95% CI	RR	95% CI
Sex							
Male	46.2	1.28	1.17, 1.39	1.15	1.05, 1.26	1.07	0.97, 1.18
Female	53.8	1.24	1.11, 1.39	1.11	0.99, 1.23	1.12	0.99, 1.27
Age, years							
<65	68.0	1.21	1.03, 1.43	1.07	0.91, 1.27	1.03	0.85, 1.25
≥65	32.0	1.18	1.10, 1.28	1.14	1.06, 1.23	1.09	1.00, 1.19
Comorbid conditions							
No	93.3	1.22	1.13, 1.33	1.11	1.02, 1.20	1.07	0.98, 1.18
Yes	6.7	1.20	1.06, 1.35	1.18	1.05, 1.33	1.10	0.96, 1.26
Income quintiles							
High (4–5)	43.1	1.32	1.17, 1.49	1.19	1.05, 1.34	1.12	0.97, 1.29
Low (1–3)	56.9	1.14	1.05, 1.24	1.12	1.03, 1.22	1.06	0.97, 1.17

[a] Model 1: bivariable analysis; model 2: adjusted for age, sex, preexisting comorbid conditions, and neighborhood socioeconomic status; model 3: model 2 further adjusted for concentrations of traffic-related air pollutants, including particulate matter less than or equal to 2.5 μm in aerodynamic diameter, nitrogen dioxide, and black carbon.

although cigarette smoking is the single most important risk factor for coronary mortality (55), it does not substantially affect the associations between fine particulate air pollution and CHD (56, 57). Similarly, recent studies have also shown that cigarette smoking does not substantially affect the associations between community noise and coronary events (3, 27).

Third, low individual SES is a risk factor for CHD (58), and persons with low SES are more likely to be exposed to community noise and traffic-related air pollution (59). Individual SES is thus a possible confounder for the observed associations. As individual SES were not available in this study (11, 12), we used neighborhood income quintiles as a surrogate for individual SES. There is some evidence that this approach is valid for control of individual SES (25, 26). In addition, in a subgroup analysis of the subjects ($n = 1{,}194$) who participated in the Canadian Community Health Survey (2000–2001), neighborhood income quintiles were strongly associated with individual annual household income, educational level, marital status, and daily fruit and vegetable intakes (all $P < 0.001$) (Web Table 7).

Finally, A-weighted equivalent sound pressure level based on the equal energy principle over a specific time period has been widely used in community noise exposure assessment (2–4, 16, 27, 29). This method may be appropriate for continuous noise, such as road traffic noise, but cannot reflect actual disturbance caused by aircraft noise, which is composed of a small number of high-level discrete noise events (16). This may partly explain the null association between aircraft noise and coronary mortality in our study.

In conclusion, in the present population-based cohort study, a 10-dB(A) elevation in residential noise levels was associated with a 9% increase in the risk of death from CHD. There was no discernible linear exposure-response relation; subjects in the highest noise decile (>70 dB(A)) had a 22% increase in CHD mortality compared with persons in the lowest decile (≤58 dB(A)). An elevation in black carbon concentrations equal to the interquartile range (0.97×10^{-5}/m) was associated with a 4% increase in the risk of CHD mortality. We did not find any interactions between noise and black carbon levels with respect to coronary mortality. These findings suggest that both community noise and traffic-related fine particulate air pollution indicated by black carbon concentration may be partly responsible for the observed associations between exposure to road traffic and adverse cardiovascular outcomes.

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REFERENCES

1. Brook RD, Rajagopalan S, Pope CA III, et al. Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. *Circulation*. 2010;121(21):2331–2378.
2. Babisch W, Beule B, Schust M, et al. Traffic noise and risk of myocardial infarction. *Epidemiology*. 2005;16(1):33–40.
3. Selander J, Nilsson ME, Bluhm G, et al. Long-term exposure to road traffic noise and myocardial infarction. *Epidemiology*. 2009;20(2):272–279.
4. Huss A, Spoerri A, Egger M, et al. Aircraft noise, air pollution, and mortality from myocardial infarction. Swiss National Cohort Study Group. *Epidemiology*. 2010;21(6):829–836.
5. Babisch W. Cardiovascular effects of noise. *Noise Health*. 2011; 13(52):201–204.
6. Davies HW, Vlaanderen JJ, Henderson SB, et al. Correlation between co-exposures to noise and air pollution from traffic sources. *Occup Environ Med*. 2009;66(5):347–350.
7. Allen RW, Davies H, Cohen MA, et al. The spatial relationship between traffic-generated air pollution and noise in 2 US cities. *Environ Res*. 2009;109(3):334–342.
8. Brauer M, Hoek G, van Vliet P, et al. Estimating long-term average particulate air pollution concentrations: application of traffic indicators and geographic information systems. *Epidemiology*. 2003;14(2):228–239.
9. Zhu Y, Hinds WC, Kim S, et al. Concentration and size distribution of ultrafine particles near a major highway. *J Air Waste Manag Assoc*. 2002;52(9):1032–1042.
10. Foraster M, Deltell A, Basagaña X, et al. Local determinants of road traffic noise levels versus determinants of air pollution levels in a Mediterranean city. *Environ Res*. 2011;111(1): 177–183.
11. Gan WQ, Tamburic L, Davies HW, et al. Changes in residential proximity to road traffic and the risk of death from coronary heart disease. *Epidemiology*. 2010;21(5):642–649.
12. Gan WQ, Koehoorn M, Davies HW, et al. Long-term exposure to traffic-related air pollution and the risk of coronary heart disease hospitalization and mortality. *Environ Health Perspect*. 2011;119(4):501–507.
13. British Columbia Ministry of Health Services. *Medical Services Plan of B.C*. Victoria, Canada: British Columbia Ministry of Health Services; 2010. (http://www.health.gov.bc.ca/msp/index.html). (Accessed July 1, 2011).
14. Chamberlayne R, Green B, Barer ML, et al. Creating a population-based linked health database: a new resource for health services research. *Can J Public Health*. 1998;89(4): 270–273.
15. Gan WQ, McLean K, Brauer M, et al. Modeling population exposure to community noise and air pollution in a large metropolitan area. *Environ Res*. In press.

16. Berglund B, Lindvall T, Schwela DH, eds. *Guidelines for Community Noise*. Geneva, Switzerland: World Health Organization; 1999. (http://www.bvsde.paho.org/bvsci/i/fulltext/noise/noise.pdf). (Accessed July 1, 2011).
17. World Health Organization. *Burden of Disease From Environmental Noise: Quantification of Healthy Life Years Lost in Europe*. Geneva, Switzerland: World Health Organization; 2011. (http://docs.wind-watch.org/WHO-burden-of-disease-from-environmental-noise-2011.pdf). (Accessed July 1, 2011).
18. Henderson SB, Beckerman B, Jerrett M, et al. Application of land use regression to estimate long-term concentrations of traffic-related nitrogen oxides and fine particulate matter. *Environ Sci Technol*. 2007;41(7):2422–2428.
19. Larson T, Henderson SB, Brauer M. Mobile monitoring of particle light absorption coefficient in an urban area as a basis for land use regression. *Environ Sci Technol*. 2009;43(13):4672–4678.
20. Brauer M, Lencar C, Tamburic L, et al. A cohort study of traffic-related air pollution impacts on birth outcomes. *Environ Health Perspect*. 2008;116(5):680–686.
21. Pearson TA, Blair SN, Daniels SR, et al. AHA guidelines for primary prevention of cardiovascular disease and stroke: 2002 update: consensus panel guide to comprehensive risk reduction for adult patients without coronary or other atherosclerotic vascular diseases. *Circulation*. 2002;106(3):388–391.
22. Hole DJ, Watt GC, Davey-Smith G, et al. Impaired lung function and mortality risk in men and women: findings from the Renfrew and Paisley Prospective Population Study. *BMJ*. 1996;313(7059):711–715.
23. Sin DD, Man SF. Chronic obstructive pulmonary disease as a risk factor for cardiovascular morbidity and mortality. *Proc Am Thorac Soc*. 2005;2(1):8–11.
24. Pope CA III, Ezzati M, Dockery DW. Fine-particulate air pollution and life expectancy in the United States. *N Engl J Med*. 2009;360(4):376–386.
25. Domínguez-Berjón F, Borrell C, Rodríguez-Sanz M, et al. The usefulness of area-based socioeconomic measures to monitor social inequalities in health in Southern Europe. *Eur J Public Health*. 2006;16(1):54–61.
26. Krieger N. Overcoming the absence of socioeconomic data in medical records: validation and application of a census-based methodology. *Am J Public Health*. 1992;82(5):703–710.
27. Beelen R, Hoek G, Houthuijs D, et al. The joint association of air pollution and noise from road traffic with cardiovascular mortality in a cohort study. *Occup Environ Med*. 2009;66(4):243–250.
28. Janssen NA, Hoek G, Simic-Lawson M, et al. Black carbon as an additional indicator of the adverse health effects of airborne particles compared to PM_{10} and $PM_{2.5}$. *Environ Health Perspect*. 2011;119(12):1691–1699.
29. Babisch W, Ising H, Gallacher JE, et al. Traffic noise and cardiovascular risk: the Caerphilly and Speedwell Studies, third phase—10-year follow up. *Arch Environ Health*. 1999;54(3):210–216.
30. Sørensen M, Hvidberg M, Andersen ZJ, et al. Road traffic noise and stroke: a prospective cohort study. *Eur Heart J*. 2011;32(6):737–744.
31. Rozanski A, Blumenthal JA, Kaplan J. Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation*. 1999;99(16):2192–2217.
32. Rosengren A, Hawken S, Ounpuu S, et al. Association of psychosocial risk factors with risk of acute myocardial infarction in 11119 cases and 13648 controls from 52 countries (the INTERHEART Study): case-control study. *Lancet*. 2004;364(9438):953–962.
33. von Känel R, Mills PJ, Fainman C, et al. Effects of psychological stress and psychiatric disorders on blood coagulation and fibrinolysis: a biobehavioral pathway to coronary artery disease? *Psychosom Med*. 2001;63(4):531–544.
34. World Health Organization. *Night Noise Guidelines for Europe*. Geneva, Switzerland: World Health Organization; 2009. (http://www.euro.who.int/__data/assets/pdf_file/0017/43316/E92845.pdf). (Accessed July 1, 2011).
35. Babisch W, Fromme H, Beyer A, et al. Increased catecholamine levels in urine in subjects exposed to road traffic noise: the role of stress hormones in noise research. *Environ Int*. 2001;26(7-8):475–481.
36. Selander J, Bluhm G, Theorell T, et al. Saliva cortisol and exposure to aircraft noise in six European countries. HYENA Consortium. *Environ Health Perspect*. 2009;117(11):1713–1717.
37. Remme WJ. The sympathetic nervous system and ischaemic heart disease. *Eur Heart J*. 1998;19(suppl F):F62–F71.
38. Babisch W. Stress hormones in the research on cardiovascular effects of noise. *Noise Health*. 2003;5(18):1–11.
39. Frishman WH. Beta-adrenergic blockers. *Circulation*. 2003;107(18):e117–e119.
40. Maseri A, Davies G, Hackett D, et al. Coronary artery spasm and vasoconstriction: the case for a distinction. *Circulation*. 1990;81(6):1983–1991.
41. Tofler GH, Muller JE. Triggering of acute cardiovascular disease and potential preventive strategies. *Circulation*. 2006;114(17):1863–1872.
42. Libby P, Theroux P. Pathophysiology of coronary artery disease. *Circulation*. 2005;111(25):3481–3488.
43. Samuel S. Road traffic noise. In: Fwa TF, ed. *The Handbook of Highway Engineering*. 5. Boca Raton, FL: Taylor & Francis; 2006:15–32.
44. Highways Agency. *The Role of the Highways Agency in Local Air Quality Management, 2003*. London, United Kingdom: Highways Agency; 2003. (http://www.highways.gov.uk/knowledge/1801.aspx). (Accessed July 1, 2011).
45. Tong HY, Hung WT, Cheung CS. On-road motor vehicle emissions and fuel consumption in urban driving conditions. *J Air Waste Manag Assoc*. 2000;50(4):543–554.
46. Paris JJ, Franco C, Sodano R, et al. Sex differences in salivary cortisol in response to acute stressors among healthy participants, in recreational or pathological gamblers, and in those with posttraumatic stress disorder. *Horm Behav*. 2010;57(1):35–45.
47. Nethery E, Leckie SE, Teschke K, et al. From measures to models: an evaluation of air pollution exposure assessment for epidemiological studies of pregnant women. *Occup Environ Med*. 2008;65(9):579–586.
48. Van Roosbroeck S, Li R, Hoek G, et al. Traffic-related outdoor air pollution and respiratory symptoms in children: the impact of adjustment for exposure measurement error. *Epidemiology*. 2008;19(3):409–416.
49. Xie SD, Zhang YH, Li Q, et al. Spatial distribution of traffic-related pollutant concentrations in street canyons. *Atmos Environ*. 2003;37(23):3213–3224.
50. Hystad PU, Setton EM, Allen RW, et al. Modeling residential fine particulate matter infiltration for exposure assessment. *J Expo Sci Environ Epidemiol*. 2009;19(6):570–579.
51. Paunović K, Jakovljević B, Belojević G. Predictors of noise annoyance in noisy and quiet urban streets. *Sci Total Environ*. 2009;407(12):3707–3711.
52. Gan WQ, Davies HW, Demers PA. Exposure to occupational noise and cardiovascular disease in the United States: the National Health and Nutrition Examination Survey 1999–2004. *Occup Environ Med*. 2011;68(3):183–190.

53. Davies HW, Teschke K, Kennedy SM, et al. Occupational exposure to noise and mortality from acute myocardial infarction. *Epidemiology.* 2005;16(1):25–32.
54. Schisterman EF, Cole SR, Platt RW. Overadjustment bias and unnecessary adjustment in epidemiologic studies. *Epidemiology.* 2009;20(4):488–495.
55. Ockene IS, Miller NH. Cigarette smoking, cardiovascular disease, and stroke: a statement for healthcare professionals from the American Heart Association. *Circulation*. 1997;96(9): 3243–3247.
56. Pope CA III, Burnett RT, Thurston GD, et al. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. *Circulation*. 2004;109(1): 71–77.
57. Hoffmann B, Moebus S, Möhlenkamp S, et al. Residential exposure to traffic is associated with coronary atherosclerosis. Heinz Nixdorf Recall Study Investigative Group. *Circulation*. 2007;116(5):489–496.
58. Kaplan GA, Keil JE. Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation*. 1993;88(4 pt 1): 1973–1998.
59. Gunier RB, Hertz A, Von Behren J, et al. Traffic density in California: socioeconomic and ethnic differences among potentially exposed children. *J Expo Anal Environ Epidemiol*. 2003;13(3):240–246.