Long-term exposure to traffic noise and traffic-related air pollution and coronary heart disease mortality

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ABSTRACT

The authors investigated the independent and joint effects of urban noise and traffic-related air pollution on the risk of coronary heart disease (CHD) mortality. This population-based cohort study included a 5-year exposure period and a 4-year follow-up period. All residents aged 45-85 years who resided in metropolitan Vancouver during the exposure period and without known CHD at baseline were included (n = 445,868). Individual exposures to noise and traffic-related air pollutants (NO, NO2, black carbon, PM2.5) were estimated at subjects' residences using a detailed noise prediction model and land use regression models, respectively. CHD deaths were identified from provincial death registration database. After adjusting for covariates and coexistent traffic-related air pollutants or noise, an interquartile-range (IQR) increase in residential noise was associated with 6 % (95 % CI: 1-11 %) increase in CHD mortality (IQR increase in black carbon, 4%; 95%-CI: 1 - 8%). Subjects in the highest noise decile had a 22 % (95% CI: 4-43 %) increase in CHD mortality compared with those in the lowest decile. Co-exposure to higher levels of traffic noise and black carbon was associated with a substantially greater risk of coronary mortality; relative risk for CHD in top decile of noise and top quartile of black carbon was 1.45 (95 %-CI 1.14 -1.85). These results suggest independent effects of noise and traffic-related air pollution on CHD mortality.

INTRODUCTION

In recent decades, both air pollution and noise pollution have been associated with increased cardiovascular disease. In metropolitan areas, road traffic is a major contributor to ambient air pollution, and is the dominant source for community noise (Babisch 2008; Brook et al. 2010). Therefore, a concern in epidemiologic studies is that the observed associations between air pollution and adverse cardiovascular outcomes may be confounded by community noise, or vice versa; further, these coexistent environmental pollutants may interact with each other in modifying risks of coronary mortality.

In a previous study, we found that living close to major roads was associated with a 29 % (95% CI: 18-41 %) increase in CHD mortality (Gan et al. 2010). We found that black carbon, an indicator of traffic-related fine particulate air pollution, was associated with a 6 % (95% CI: 3-9 %) increase in coronary mortality, but no robust associations were found for $PM_{2.5}$, NO_2 or NO. These findings suggest that exposure to the traffic-related air pollutants alone cannot fully explain the excess risk of coronary mortality associated with residential proximity to road traffic; traffic noise might also play a role in the observed association.

In the present study, we investigated the relations between long-term exposure to community noise and CHD mortality as well as the independent and joint effects of community noise and air pollution (black carbon) on the risk of CHD mortality.

METHODS

We used linked administrative databases from the British Columbia (BC) health insurance system to assemble a population-based cohort. All Metro-Vancouver residents who met the following criteria were included in the cohort: (1) resided in the study region during the 5-year exposure period; (2) aged 45 to 85 years at baseline; and (3) without previous diagnosis of CHD.

This study included a 5-year exposure period (January 1994 to December 1998) and a 4-year follow-up period (January 1999 to December 2002). For the 5-year exposure period, individual exposures to community noise and traffic-related air pollutants were estimated at each person's residence using a noise prediction model and land use regression (LUR) models, respectively. For the 4-year follow-up period, CHD mortality information was retrieved from the provincial death registry. The associations of noise and black carbon with CHD mortality were examined using the Cox proportional hazards regression model.

We estimated annual average noise levels from road and light rail sources using CadnaA noise prediction model (Datakustik, Greifenberg, Germany). The main model inputs included: Traffic volumes, estimated using the 2003 transportation planning model EMME/2 (INRO Consultants, Montreal, Canada) used by Metro Vancouver transportation authority; fleet mix; road speed limits; traffic lights at intersections; road gradient (change in elevation along a given road); road surface (paved or loose surface); bridges (heights of the road segments above ground); buildings (height, footprint); and topography in the exposure assessment of road traffic noise. Railway noise exposure assessment was based on railway operation data including length of trains, velocity, percentage of disk brakes, and number of each type of train by day, evening, and night. For each municipality in Metro Vancouver, the data for each model input were available to a different degree and quality. Vancouver International Airport is Canada's second busiest airport and produces aircraft noise exposure forecast contours. We used their 2003 contours to estimate aircraft noise levels in the model area.

Annual average A-weighted equivalent continuous noise levels (L_{DEN} dBA) were calculated for a 10 × 10 m grid. The L_{DEN} metric integrates day noise levels (06:00 - 18:00 h), evening (18:00 - 22:00 h), and night (22:00 - 06:00 h), while reflecting increased sensitivity to noise in evening and night by adding a 5 dBA and 10 dBA weighting respectively. Based on the estimated noise levels, annual average total noise levels were calculated for each geographic area covered by a 6-digit postal code.

We used high-resolution land-use regression (LUR) models to estimate residential exposure to traffic-related air pollutants including black carbon, $PM_{2.5}$, NO_2, and NO in 2003 as described elsewhere. In brief, the concentrations of black carbon, $PM_{2.5}$, NO_2 and NO were measured in selected sampling sites (Henderson et al. 2007). Multiple linear regression techniques were used to estimate the quantitative relationships between measured air pollutant concentrations and the selected land use characteristics, and those most predictive variables were retained in the final models. Based

on the LUR models, a smooth spatial surface of predicted annual average concentrations for each air pollutant was generated in a GIS with a resolution of 10 m. Annual average concentrations of these air pollutants were assigned to each postal code in the study region.

The noise data and air pollution data were linked to study subjects' residential history through their 6-digit residential postal codes. In urban areas, a 6-digit postal code typically represents one side of a city block a single high-rise building.

The study outcome was coronary heart disease (CHD) deaths (ICD9 codes 410-414, 429.2 or ICD-10 codes I20-I25). Preexisting disease was defined as hospitalization before 1998 for diabetes, COPD or hypertension. Individual socioeconomic status (SES) data were not available; we used neighborhood-income quintiles from the 2001 Statistics Canada Census to estimate SES. The method for neighborhood-income quintiles calculation has been described in detail elsewhere (Gan et al. 2011).

Baseline characteristics between study subjects across deciles of noise levels were compared using a chi-square test for categorical variables, one-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 comorbidity, neighborhood SES were included as covariates; air pollutants or noise were added in the final models. Person-years of observation were calculated from baseline to the date of death or end of follow-up (for those who moved out of the province, the last known date in the province).

We first treated noise levels as a continuous variable to calculate relative risks (RRs) for CHD mortality associated with a 10 dBA elevation in noise levels. We then treated noise levels as a categorical variable to examine exposure-response relationships by dividing study subjects into deciles based on the noise levels; RRs of coronary mortality were calculated for each decile by using decile 1 (lowest) as the reference category. Because there was no substantial difference in effect estimates across decile 2-9, only results for decile 1, decile 2-5, decile 6-9, and decile 10 are presented.

All statistical analyses were performed using SAS 9.2 (SAS Institute Inc., Cary, NC, USA). All statistical tests were 2-sided.

RESULTS

For this cohort, the annual average noise level was 63.4 (interquartile range (IQR): 59.8 - 66.4) dBA (Table 1). Overall, noise levels were not strongly correlated with traffic-related air pollutant concentrations, the highest correlation was with black carbon ($r = 0.44$), the lowest correlation was with $PM_{2.5}$ ($r = 0.14$).

A total of 466,727 subjects met the inclusion criteria and were included at baseline. Among these, 13,992 (3.0 %) with missing air pollution data were excluded; 6,867 (1.5 %) with missing noise data were further excluded, which left 445,868 subjects for the analysis.

Table 1: Average noise levels and traffic-related air pollutant concentrations and correlation coefficients

Pollutant	Mean (SD)	Median	IQR	Range	Correlation Coefficient[a]				
					Noise	BC	$PM_{2.5}$	NO_2	NO
Noise, L_{den} dBA	63.4 (5.0)	62.4	59.8-66.4	33.0-90.0	1.00	--	--	--	--
BC, 10^{-5}/m	1.50 (1.10)[b]	1.02	0.83-1.80	0.0-4.98	0.44	1.00	--	--	--
$PM_{2.5}$, μg/m³	4.10 (1.64)	4.04	3.22-4.81	0.0-10.24	0.14	0.13	1.00	--	--
NO_2, μg/m³	32.3 (8.1)	30.8	26.7-35.2	15.3-57.5	0.33	0.39	0.47	1.00	--
NO, μg/m³	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: BC, black carbon; IQR, interquartile range; SD, standard deviation.
[a] $P < 0.001$ for each correlation coefficient.

Table 2 shows the characteristics of study subjects at baseline by deciles of noise levels. Compared with those exposed to lower noise levels (decile 1), subjects exposed to higher noise levels were more likely to have preexisting comorbidities including diabetes, COPD, and hypertensive heart disease, and to have lower neighborhood SES.

Table 2: Baseline characterization of study subjects (N=445,868) by noise exposure decile

Characteristic	Deciles of Noise Levels (L_{den} dBA)			
	Decile 1 (≤ 58)	Deciles 2-5 (59 - 62)	Deciles 6-9 (63 - 70)	Decile 10 (> 70)
Men, %	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)
Comorbidity, %				
Diabetes	2.1	2.1	2.4	2.9
COPD	1.5	1.3	1.5	1.8
Hypertensive heart disease	4.3	4.0	4.3	4.8
Any comorbidity	6.6	6.3	6.8	7.9
Income quintile, %[c]				
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

During the 4-year follow-up period 3,095 subjects died from CHD and the mortality rate was 1.83 per 1,000 person-years. Residential noise exposure was strongly associated with CHD mortality; a 10 dBA elevation in noise levels was associated with a 26 % (95% CI: 17-35 %) increase in the risk of CHD mortality. Adjusting for age, sex, preexisting comorbidity, and neighborhood SES halved the estimated relative risk, while adjustment for $PM_{2.5}$ and NO_2 had little influence on effect estimates. Adjusting for black carbon had a greater influence on the effect estimate, but a 10 dBA elevation in noise levels was still associated with a 9 % (95% CI: 1-18 %) increase in CHD mortality. For other cardiovascular diseases such as stroke, dysrhythmias, and congestive heart failure, there was no significant increase in mortality associated with a 10 dBA elevation in noise levels.

When study subjects were categorized into decile groups according to noise levels, compared with those in decile 1 with noise levels ≤58 dBA, subjects in decile 2-5 and decile 6-9 had little increase in coronary mortality, those in decile 10 exposed to

noise levels >70 dBA had a 22 % (95% CI: 4-43 %) increase in coronary mortality after adjusting for all covariates and traffic-related air pollutants, suggesting that there was no linear exposure-response relationship between noise and coronary mortality (p=0.174 for test of linear trend across decile groups in the fully adjusted model) (Table 3).

Effects of noise and black carbon on coronary mortality were additive (Table 3). No multiplicative effect (p=0.980 for the interaction term in the fully adjusted model) was observed.

Stratified analysis shows that coronary mortality associated with a 10 dBA elevation in noise was greater for female, those aged ≥65 years, with preexisting comorbidity, and with higher neighborhood SES. However, there was considerable overlap in the 95% CIs between these subgroups.

Table 3: Relative risks and 95% Confidence Interval for coronary heart disease mortality by deciles of noise levels and quartiles of black carbon concentrations[a]

Quartiles of Black Carbon (10^{-5}/m)	Deciles of Noise Levels (L_{den} dBA)							
	Decile 1 (≤ 58)		Decile 2-5 (59 – 62)		Decile 6-9 (63 – 70)		Decile 10 (> 70)	
	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
All*	1.00	Reference	1.04	0.91-1.19	1.02	0.89-1.17	1.22	1.04-1.43
Quartile 1 (0-0.83×)	1.00	Reference	1.11	0.88-1.40	1.12	0.86-1.45	1.29	0.79-2.12
Quartile 2 (0.84-1.02)	1.05	0.78, 1.41	1.09	0.87-1.37	1.06	0.84-1.34	1.24	0.88-1.74
Quartile 3 (1.03-1.80)	1.09	0.79, 1.49	1.23	0.97-1.55	1.18	0.94-1.49	1.48	1.09-2.02
Quartile 4 (1.81-4.98	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: L_{den} dBA, annual day-evening-night A-weighted equivalent continuous noise level.
[a] Adjusted for age, sex, preexisting comorbidity, neighborhood income quintiles, and co-pollutants including NO_2 and $PM_{2.5}$.*
* same as (a) but also adjusted for BC

DISCUSSION

This large population-based cohort study found that a 10 dBA elevation in residential noise levels (L_{den}) was associated with a 9 % increase in CHD mortality, after adjustment for the various covariates including traffic related air pollutants. There was no discernable linear exposure-response relationship, persons in the highest decile of noise levels (>70 dBA) had a 22 % increase in coronary mortality compared with those exposed ≤58 dBA.

A simple additive effect of the two exposures was noted (Selander et al. 2009). Also examined this but found while both exposure to noise and NO_2 showed independent elevation of risk, jointly no excess risk was observed (Selander et al. 2009).

These findings are largely consistent with prior studies of the combined effects of noise and traffic-related air pollution on CVD. All have supported a model in which

both noise and air pollution are independent risk factors for CVD. Beleen et al. (2009) reported cardiovascular mortality increased 17 % (95 % CI: 0.94 to 1.45) for those exposed > 65 Vs. ≤ 50 dBA) after adjusting for black smoke; Risk of heart failure was 1.9. Risks associated with exposure to black smoke was similarly insenstive to adjustment for noise. Unlike our study, there was no discernible increase in CHD mortality. Selander et al. (2009) found that road traffic noise (≥50 vs. <50 dBA) was associated with a 12 % (95% CI: 0.95-1.33) increase in the risk of MI after adjusting for NO_2 and other cardiovascular risk factors; after excluding those with hearing loss or with other sources of noise exposure, the excess risk of MI increased to 38 % (95 CI: 1.11-1.71). And in a 5-year Swiss National Cohort Study with 4.6 million subjects, Huss et al. (2010) found that people exposed to aircraft noise ≥60 vs. <45 dBA had a 30 % (95% CI: 0.96-1.76) increase in MI mortality after adjusting for particulate air pollution, residential proximity to major roads, and other covariates; when the analysis was restricted to those who lived in their residences for at least 15 years, the MI mortality increased by 48 % (95% CI: 1.01-2.18).

The idea of independent effects has been described by Allen & Adar (2011) who They point out that it is consitent with other strands of evidence: both mechanism have plausible biological mechanisms, and animal and experimental evidence exists that should be less influenced by confounding as would occupational studies.

This study shows that the correlations between modeled noise and air pollution levels range from 0.14 ($PM_{2.5}$) to 0.44 (black carbon), which is within the range of correlations reported in previous studies. In practice, some road traffic factors such as speed, volume, and operating conditions may differentially affect the emission levels of noise and traffic-related air pollution, which may partly explain the low-to-moderate correlations between noise and traffic-related air pollution in the study region.

Previous findings on gender differences in the risk of coronary mortality associated with noise exposure are not consistent. Some studies found men are more susceptible to noise exposure than women (Babisch et al. 2005) whereas other studies found no differences between men and women (Beelen et al. 2009; Selander et al. 2009). Our study shows that men and women had similar risk of coronary mortality associated with noise exposure; however after adjusting for traffic-related air pollutants including $PM_{2.5}$, NO_2 and black carbon, women had a 7 % excess risk of coronary mortality compared with men, although the difference was not statistically significant.

This study had limitations that should be considered. The exposure assessment was based on the residential postal codes of study subjects to estimate the exposure at their residences. This method cannot precisely reflect actual individual exposure (Nethery et al. 2008). Nevertheless, these factors are likely to cause non-differential exposure misclassification, leading to underestimations of true risk of coronary mortality associated with exposure to noise and traffic-related air pollution (van Roosbroeck et al. 2008).

Second, because the cohort was constructed using linked administrative health databases from the provincial health insurance system, few individual-level cardiovascular risk factors were available. We adjusted for preexisting comorbidity including diabetes, COPD, or hypertensive heart disease. Because these comorbidities and CHD share common behavioral risk factors, adjusting for these comorbidities to some extent was able to reduce the influence of some uncontrolled risk factors and these comorbidities themselves on the effect estimates. In addition, it does not substantially

confound the associations between fine particulate air pollution and CHD (Pope et al. 2004). Similarly, recent studies have also shown that cigarette smoking (either smoking status or daily smoking amount) did not substantially influence the associations between noise exposure and coronary events (Selander et al. 2009; Beelen et al. 2009).

Individual SES is a possible confounder for the observed association. As discussed before, individual SES was not available in this study, we used neighborhood income quintiles to approximately estimate individual SES. There is some evidence that this approach is valid for control of individual SES (Dominguez-Berjon et al. 2006; Krieger 1992). In addition, in a subgroup analysis of the study 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, education level, marital status, and daily fruit and vegetable intake (all $p<0.001$). Based on these results, we believe that including neighborhood income quintiles in the Cox model could effectively minimize the confounding effects of individual SES.

Finally, A-weighted equivalent sound pressure level based on the equal energy principle over some time period has been widely used in community noise measurement. This method may be appropriate for continuous noise such as road traffic noise, but it cannot reflect actual disturbance caused by a small number of high-level discrete noise events.

In conclusion, in this large population-based cohort study, we found that a 10 dBA elevation in residential noise levels (L_{den}) was associated with a 9 % increase in CHD mortality. There was no discernable linear exposure-response relationship, subjects in the highest noise decile (>70 dBA) had a 22 % increase in CHD mortality compared with those in the lowest decile (≤58 dBA). An IQR (0.97×10^{-5}/m) elevation in black carbon concentrations was associated with a 4 % increase in CHD mortality. We found a simple additive effect between noise and black carbon on coronary mortality. These findings suggest that both noise and traffic-related fine particulate air pollution indicated by black carbon may be partly responsible for the observed associations between exposure to road traffic and adverse cardiovascular outcomes.

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