Changes in Residential Proximity to Road Traffic and the Risk of Death From Coronary Heart Disease

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Background: Residential proximity to road traffic is associated with increased coronary heart disease (CHD) morbidity and mortality. It is unknown, however, whether changes in residential proximity to traffic could alter the risk of CHD mortality.

Methods: We used a population-based cohort study with a 5-year exposure period and a 4-year follow-up period to explore the association between changes in residential proximity to road traffic and the risk of CHD mortality. The cohort comprised all residents aged 45–85 years who resided in metropolitan Vancouver during the exposure period and without known CHD at baseline (n = 450,283). Residential proximity to traffic was estimated using a geographic information system. CHD deaths during the follow-up period were identified using provincial death registration database. The data were analyzed using logistic regression.

Results: Compared with the subjects consistently living away from road traffic (>150 m from a highway or >50 m from a major road) during the 9-year study period, those consistently living close to traffic (≤150 m from a highway or ≤50 m from a major road) had the greatest risk of CHD mortality (relative risk [RR] = 1.29 [95% confidence interval = 1.18–1.41]). By comparison, those who moved closer to traffic during the exposure period had less increased risk than those who were consistently exposed (1.20 [1.00–1.43]), and those who moved away from traffic had even less increase in the risk (1.14 [0.95–1.37]). All analyses were adjusted for baseline age, sex, pre-existing comorbidities (diabetes, chronic obstructive pulmonary disease, hypertensive heart disease), and neighborhood socioeconomic status.

Conclusions: Living close to major roadways was associated with increased risk of coronary mortality, whereas moving away from major roadways was associated with decreased risk.

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A growing body of epidemiologic evidence has demonstrated that long-term exposure to ambient air pollution, especially fine particles, is associated with increased cardiovascular morbidity and mortality.[1,2] Several cohort studies suggest that coronary heart disease (CHD) is more strongly associated with fine particulate air pollution than are other cardiovascular outcomes.[3,4] In metropolitan areas, road traffic is a major contributor to air pollution.[5,6] A European study estimated that approximately half of the adult mortality from air pollution was attributed to traffic-related air pollution.[7] Because exposure to traffic-related air pollution is extensive worldwide, the corresponding adverse cardiovascular effects may represent an important public health problem.[1]

The concentrations of traffic-related air pollutants decrease exponentially from major roadways and typically approach background concentrations within about 150 meters.[6,8] The distances from residences to major roadways may therefore reflect spatial variability in the concentrations of traffic-related air pollutants. Although traffic proximity may also be associated with other exposures such as traffic noise, it can serve as a simple and policy-relevant surrogate for exposure to traffic-related air pollution.[9,10] This metric has been widely used in epidemiologic studies of the health effects of traffic-related air pollution.[9–19]

There have been a number of epidemiologic studies examining the associations between residential proximity to traffic and adverse cardiovascular outcomes including arterial atherosclerosis[11,12] and CHD morbidity and mortality.[13–19] Although most of these studies have reported associations, the findings are not entirely consistent. One critical limitation of these studies is the assumption that baseline residential exposure status is consistent during the entire follow-up period; residential relocation after baseline enrollment has generally been ignored. This unrealistic assumption may

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result in exposure misclassification, and thus bias effect estimates toward the null.

We conducted a large population-based cohort study with detailed residential history information to investigate the association between residential proximity to road traffic and the risk of CHD mortality. Specifically, we examined the following factors: (1) whether residential proximity to traffic was associated with higher levels of exposure to traffic-related air pollution; (2) whether living close to traffic was associated with an increased risk of CHD mortality; and (3) whether changing residences, and therefore changing proximity to traffic, was associated with an altered risk of CHD mortality.

METHODS

Study Design

This population-based cohort study was conducted in metropolitan Vancouver, Canada. We used linked administrative databases from British Columbia's universal health insurance system to assemble a population-based cohort (eAppendix, http://links.lww.com/EDE/A405). This study included 2 stages: a 5-year exposure period (January 1994–December 1998), and a 4-year follow-up period (January 1999–December 2002). Mortality information during the follow-up period was identified from the provincial death registration database. CHD mortality was compared between study subjects with different residential-traffic-exposure profiles to determine the relationship between residential proximity to road traffic and the risk of CHD mortality. This study was approved by the institutional review board of The University of British Columbia.

Study Cohort

All metropolitan Vancouver residents who met the following criteria at baseline (January 1999) were included in the cohort: (1) registered with the provincial health insurance plan, which provides universal coverage to the resident population; (2) age 45–85 years; and (3) without previous diagnosis of CHD. A small number (4%) of study subjects who moved to other regions of the province during the 5-year exposure period were included, all other subjects remained in the study region during the exposure period.

Residential Proximity to Road Traffic

We categorized residential proximity to traffic based on individual residential histories (eAppendix, http://links.lww.com/EDE/A405), and whether a 6-digit residential postal code (area centroid) was located within 50 m or 150 m of a highway or a major road during the 5-year exposure period and the 4-year follow-up period. The study subjects were divided into 4 groups:

1. Not exposed to traffic: consistently living away from traffic until the end of follow-up;
2. Consistent exposure to traffic: consistently living close to traffic until the end of follow-up;
3. Moved close to traffic: changing residence from nonexposed to exposed to traffic during the exposure period and retaining this exposure status until the end of follow-up;
4. Moved away from traffic: changing residence from exposed to nonexposed to traffic during the exposure period and retaining this nonexposure status until the end of follow-up.

Subjects with more than one change in exposure status during the exposure period were excluded; those who changed their exposure status during the follow-up period were also excluded.

Depending on road types (highway or major road) and distance from major roadways, residential proximity to traffic was divided into 5 categories: (1) $\leq$50 versus $>$50 m from a highway; (2) $\leq$150 versus $>$150 m from a highway; (3) $\leq$50 versus $>$50 m from a major road; (4) $\leq$150 versus $>$150 m from a major road; (5) $\leq$150 m from a highway or $\leq$50 m from a major road versus $>$150 m from a highway or $>$50 m from a major road. Subjects living within a specific distance were assigned to the exposure group, while the rest were assigned to the nonexposure group (eAppendix, http://links.lww.com/EDE/A405).

Traffic-related Air Pollution Assessment

We used high-resolution land-use regression models to evaluate exposure levels to traffic-related air pollutants. Because the air pollution measurements did not cover the whole study region, air pollution data were available only for a subgroup of the cohort.

Using detailed residential history and corresponding monthly concentrations of traffic-related air pollutants during the 5-year exposure period, average concentrations of air pollutants were calculated for each subject. Detailed methods for the measurement of air pollutants in this study have been described elsewhere.[20,21] A brief description of traffic-related air pollution assessment is available in the eAppendix (http://links.lww.com/EDE/A405).

Coronary Heart Disease Mortality

A case of CHD death was defined as a death record in the provincial death registration database with CHD (ICD-9 codes 410–414, 429.2 and ICD-10 codes I20–I25) as the cause of death. A small proportion of deaths were identified using provincial hospitalization records: a hospitalization death record with CHD as the principal diagnosis for a hospital admission.

Subjects who had a hospitalization record with CHD as the principal or primary diagnosis before baseline (on the basis of data available from January 1991 to December 1998) were regarded as previously-diagnosed CHD cases, and were excluded from the analysis.

Pre-existing Comorbidities

Chronic obstructive pulmonary disease (COPD)[22] (ICD-9: 490, 491, 492, 496; ICD-10: J40–J44), diabetes[23] (ICD-9, 250; ICD-10, E10–E14), and hypertensive heart disease[23] (ICD-9: 401–404; ICD-10: I10–I14) are independent risk factors for CHD. In addition, these chronic diseases and CHD share common behavioral risk factors such as cigarette smoking. In an effort to control the influence of the pre-existing comorbidities and these common behavioral risk factors, all diagnoses (not restricted to principal or primary diagnosis) in a hospitalization record were used to identify subjects with these comorbidities. One hospitalization record with the diagnosis of any of these diseases during January 1991 to December 1998 was defined as the presence of pre-existing comorbidities.

Neighborhood Socioeconomic Status

Individual-level income data were not available in this study. We used neighborhood-income quintiles from the 2001 Statistics Canada Census data to approximate a subject's socioeconomic status (SES). Neighborhood-income quintiles were assigned to study subjects through their residential postal codes (eAppendix, http://links.lww.com/EDE/A405).

Statistical Analysis

We compared the baseline characteristics among the exposure groups using a χ^2 test for dichotomous variables, one-way analysis of variance (ANOVA) for continuous variables, and Tukey's post hoc analysis for pair-wise comparisons of continuous variables. Similarly, in a subgroup analysis for the subjects with air pollution data, we used ANOVA and Tukey's post hoc analysis to determine whether residential traffic-exposure profiles were associated with exposure levels to traffic-related air pollutants.

To determine the association between residential proximity to traffic (predictor variable) and the risk of CHD mortality (dependent variable), we first performed bivariable logistic regression analysis using the nonexposed group as the reference category. Then we performed multivariable logistic regression analysis to adjust for age (quintiles), sex, neighborhood income quintiles, and pre-existing comorbidities including diabetes, COPD, or hypertensive heart disease (yes or no). These analyses were repeated for different combinations of road types (highway or major road) and distances (50 or 150 m).

To examine the influence of age and sex on the risk of CHD mortality associated with traffic exposure, we performed stratification analyses by age ($<$65 years, $\geq$65 years) and sex, using the exposure category $\leq$150 m from a highway or $\leq$50 m from a major road.

The exposure category "$\leq$50 versus $>$50 m from a highway" had the largest effect estimates. We therefore used this category to perform a sensitivity analysis in which we compared the relative risks of CHD mortality using various distances from a highway and various frames of reference.

All analyses were performed using SAS 9.1 (SAS Institute Inc., Cary, NC).

RESULTS

We use the road traffic exposure category "$\leq$150 m from a highway or $\leq$50 m from a major road versus $>$150 m from a highway or $>$50 m from a major road" to present the overall results of this study. At baseline in January 1999, there were 488,785 subjects who met the inclusion criteria. At the end of follow-up, 38,502 persons (8%) were lost to follow-up, mainly due to moving out of the province or dying from other diseases. This left 450,283 subjects with complete data; 210,128 persons (47%) changed their residences at least one time during the 9-year study period, and 68,726 persons (15%) changed their exposure status. We excluded 12,619 persons (3%) with multiple changes in exposure status and 22,871 (5%) who changed their exposure status during the follow-up period. This left 414,793 subjects for analysis: 328,609 (79%) who consistently lived away from traffic, 52,948 (13%) who consistently lived close to traffic, 15,747 (4%) who moved close to traffic, and 17,489 (4%) who moved away from traffic (Table 1).

The baseline characteristics of these subjects are summarized by the 4 exposure groups in Table 1. Fewer than half (46%) of the subjects were male; the average age (SD) was 59 (11) years (range, 45–83 years). Overall, compared with those consistently living away from traffic, persons who consistently lived close to traffic were older and more likely to have lower neighborhood SES and pre-existing comorbidities.

Based on the land-use regression data that incorporated high spatial resolution, persons who consistently lived close to traffic were exposed to elevated concentrations of black carbon, $PM_{2.5}$, NO_2, and NO during the 5-year exposure period (Table 2). Furthermore, those once living close to traffic were also exposed to higher concentrations of black carbon, NO_2, and NO; this increment was even larger for those who moved their residences close to traffic.

During the follow-up period, 3133 people (3097 from the death registration database and 36 from hospitalization records) died of CHD, for an overall mortality rate of 7.6 per 1000 subjects. Compared with subjects consistently living away from traffic, those consistently living close to traffic were 69% (95% confidence interval [CI] = 1.55–1.85) more likely to die of CHD during the follow-up period. For those who moved away from traffic during the exposure period, there was a 4% increase in the risk of CHD mortality (0.87–1.25) during the follow-up period compared with the unexposed. For those moving closer to traffic during the exposure period, the risk of CHD mortality increased 23% (1.03–1.46) as compared with the unexposed. Adjustment for

TABLE 1. Baseline Characteristics[a] of Study Subjects by Exposure Groups[b]

	Not Exposed to Traffic (n = 328,609)	Moved Close to Traffic (n = 15,747)	Moved Away From Traffic (n = 17,489)	Consistent Exposure to Traffic (n = 52,948)
Men	46	46	47	45
Age (years); mean (SD)	58.7 (10.4)	58.6 (10.2)	57.6 (10.0)	61.0 (10.9)
Age quintiles (years)				
45–48	19	19	21	15
49–53	22	21	23	18
54–60	21	21	21	19
61–69	20	21	20	22
70–83	19	18	15	26
Comorbidity				
Diabetes	1.9	2.1	2.0	2.5
COPD	1.0	1.2	1.2	1.5
Hypertensive heart disease	3.7	4.0	3.9	4.6
Any of the above	5.6	6.4	6.1	7.2
Income quintiles[c]				
1	15	25	20	27
2	18	19	19	20
3	19	21	20	19
4	22	18	22	16
5	26	17	20	19

[a]Percent, unless otherwise specified.
[b]Traffic exposure was defined as ≤150 m from a highway or ≤50 m from a major road.
[c]Quintile 1 represents the lowest and Quintile 5 the highest neighborhood income quintile.

TABLE 2. Average Concentrations of Traffic-related Air Pollutants by Exposure Groups

	Not Exposed to Traffic (n = 306,296) Mean (SD)	Moved Close to Traffic (n = 13,285) Mean (SD)	Moved Away From Traffic (n = 14,582) Mean (SD)	Consistent Exposure to Traffic (n = 50,502) Mean (SD)
Black carbon (10^{-5}/m)	1.1 (0.7)	2.3 (1.1)	1.9 (0.9)	3.0 (1.5)
$PM_{2.5}$ ($\mu g/m^3$)	4.0 (1.6)	4.2 (1.6)	4.1 (1.6)	4.3 (1.8)
NO_2 ($\mu g/m^3$)	31.3 (7.9)	33.9 (7.5)	33.0 (7.6)	35.5 (7.9)
NO ($\mu g/m^3$)	28.8 (8.2)	39.5 (13.4)	34.8 (10.7)	45.9 (16.6)

This is a sub-group analysis for the subjects (93%) with land-use regression data. Traffic exposure was defined as ≤150 m from a highway or ≤50 m from a major road.

baseline age, sex, pre-existing comorbidities, and neighborhood SES generally reduced the relative risks but did not change the overall pattern of the results: the risk of CHD mortality increased by 29% (1.18–1.41), 14% (0.95–1.37), and 20% (1.00–1.43), respectively, for those consistently living close to traffic, moving away from traffic, and moving close to traffic, respectively (Table 3).

Similar CHD mortality patterns were observed when the analysis was repeated using different road types and distances (Table 3, Fig. 1). Figure 1 shows that the risk of CHD mortality was strongly dependent on road types (traffic volume) and the distances from major roadways. For example, for those consistently living close to traffic, the risk of CHD mortality rapidly decreased when the distance from traffic increased from 50 to 150 m, or when road type changed from a highway (21,000–114,000 vehicles/day) to a major road (15,000–18,000 vehicles/day). Overall, compared with consistently living away from traffic, consistently living close to traffic was associated with the highest risk of CHD mortality (Fig. 1); moving closer to traffic was associated with an increased risk but lower risk compared with consistently living close to traffic. Moving away from traffic was associated with a decreased risk but higher risk compared with consistently living away from traffic.

For those consistently living within 150 m from a highway or 50 m from a major road (vs. consistently living >150 m from a highway or >50 m from a major road), the risk of CHD mortality was higher for men than for women and higher for the younger (<65 years) than for the older group (≥65 years) (Fig. 2).

TABLE 3. Association of Road Traffic Exposure With Coronary Heart Disease Mortality

Exposure Category	Not Exposed to Traffic[a]	Moved Close to Traffic	Moved Away From Traffic	Consistent Exposure to Traffic
≤150 m Highway or ≤50 m major road				
No. deaths/total number	2271/328,609	131/15,747	124/17,489	607/52,948
Crude RR (95% CI)	1.00	1.23 (1.03–1.46)	1.04 (0.87–1.25)	1.69 (1.55–1.85)
Adjusted RR (95% CI)[b]	1.00	1.20 (1.00–1.43)	1.14 (0.95–1.37)	1.29 (1.18–1.41)
≤50 m Highway				
No. deaths/total number	3164/434,602	26/2304	21/2729	73/4343
Crude RR (95% CI)	1.00	1.55 (1.05–2.29)	1.05 (0.69–1.62)	2.33 (1.84–2.94)
Adjusted RR (95% CI)[b]	1.00	1.44 (0.97–2.13)	1.09 (0.71–1.69)	1.54 (1.21–1.96)
≤150 m Highway				
No. deaths/total number	2851/397,341	59/7016	62/8484	257/20,085
Crude RR (95% CI)	1.00	1.18 (0.91–1.53)	1.02 (0.80–1.32)	1.80 (1.59–2.05)
Adjusted RR (95% CI)[b]	1.00	1.22 (0.94–1.59)	1.11 (0.86–1.44)	1.36 (1.19–1.55)
≤50 m Major road				
No. deaths/total number	2674/370,505	90/10,534	88/12,935	330/31,073
Crude RR (95% CI)	1.00	1.20 (0.97–1.48)	0.95 (0.77–1.18)	1.49 (1.33–1.67)
Adjusted RR (95% CI)[b]	1.00	1.16 (0.93–1.43)	1.07 (0.86–1.33)	1.15 (1.02–1.29)
≤150 m Major road				
No. deaths/total number	1752/247,483	157/19,724	170/25,781	1024/112,093
Crude RR (95% CI)	1.00	1.17 (1.00–1.38)	0.97 (0.83–1.14)	1.35 (1.25–1.46)
Adjusted RR (95% CI)[b]	1.00	1.24 (1.05–1.46)	1.09 (0.93–1.28)	1.11 (1.02–1.19)

The total number of subjects in each traffic exposure category is different due to exclusion of subjects with multiple changes in exposure status and subjects who changed their exposure status during the follow-up period.

[a]Reference category.

[b]Adjusted for age, sex, neighborhood socioeconomic status, and pre-existing comorbidities.

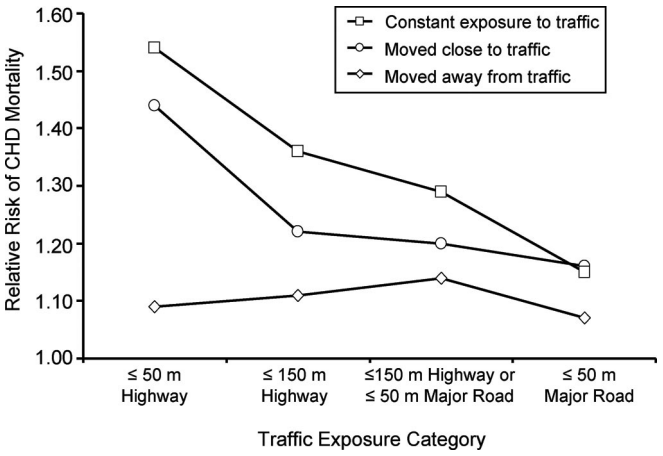

FIGURE 1. Association of road traffic exposure with coronary heart disease mortality by road types and distances. RRs adjusted for age, sex, neighborhood SES, and pre-existing comorbidities.

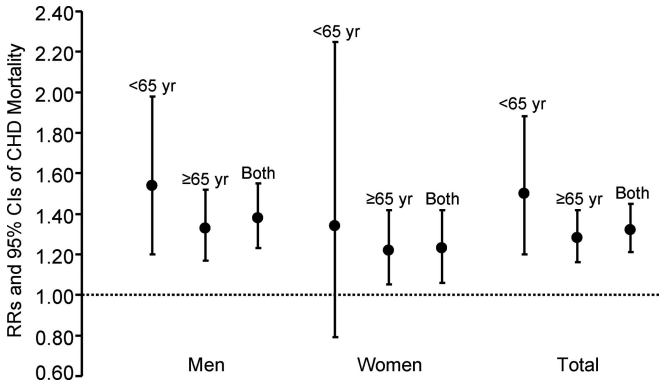

FIGURE 2. Association of road traffic exposure with coronary heart disease mortality by sex and age (traffic exposure was defined as ≤150 m highway or ≤50 m major road). Adjusted for neighborhood SES and pre-existing comorbidities; the combined analyses ("Both") were additionally adjusted for age (<65 years, ≥65 years); for the total group, the analyses were additionally adjusted for age (<65 years, ≥65 years) and sex.

In the sensitivity analysis examining the effects of distances and reference groups, for those who moved away from traffic during the exposure period, the effect estimates were very close among the 3 groups (Fig. 3). However, for those who moved close to or consistently lived close to traffic, the effect estimates changed in response to different distances and references used in the analysis, indicating that the observed association between residential proximity to traffic and the risk of CHD mortality was sensitive to distances from highways and the references used for comparison.

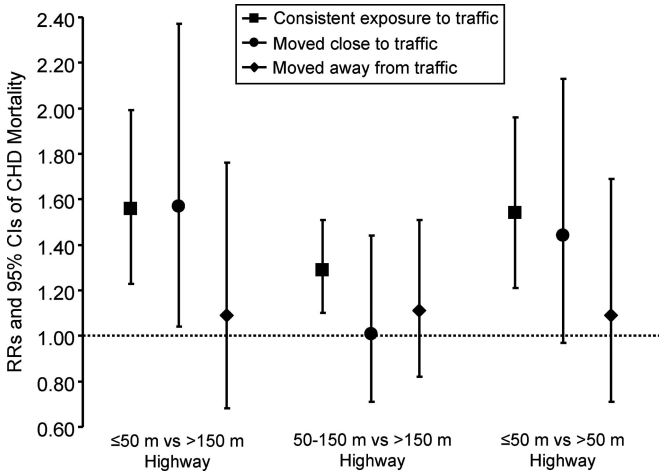

FIGURE 3. Association of road traffic exposure with coronary heart disease mortality by distances from highways. Adjusted for age, sex, neighborhood SES, and pre-existing comorbidities.

DISCUSSION

In this large population-based cohort study with detailed residential-history information, living close to road traffic was associated with an increased risk of CHD mortality. More importantly, a change in residential proximity to traffic was associated with an altered risk of CHD mortality: moving close to traffic was associated with a relatively increased risk, whereas moving away from traffic was associated with a relatively decreased risk.

Previous studies examining the associations between residential proximity to traffic and cardiovascular outcomes have not reported entirely consistent findings. A cross-sectional study carried out in Germany with 4494 participants found that living close to a major road was associated with more severe coronary artery calcification.[11] In contrast, a recent study with 1147 participants in the United States found no appreciable association between residential proximity to a major road and abdominal aortic calcification.[12] In a 13-year cohort study of 13,309 people in the United States, Kan et al[13] found that residential traffic intensity was associated with an increased risk of fatal and nonfatal coronary events. Similarly, in a large case-control study, Tonne et al[14] reported that living near a major road was associated with a 5% increase in the risk of acute myocardial infarction. A 13-year cohort study of 4800 women in Germany also found that living within 50 m of a major road was associated with increased cardiopulmonary mortality.[15] In contrast, in a 9-year Dutch cohort study with 117,528 participants, Beelen et al[16] did not find an association between residential proximity to a major road or residential traffic intensity and cardiovascular mortality. Several studies have also reported associations between exposures to traffic-related air pollutants such as nitrogen oxides and adverse cardiovascular outcomes.[17–19] Because of differences in traffic characteristics, study populations, meteorological and geographic conditions, study design, and statistical methods, it is difficult to quantitatively compare the results from different studies. Although the findings from these previous studies are not fully consistent, the present study and most previous studies suggest that residential proximity to traffic is associated with increased risk of CHD mortality. Traffic-related air pollution and other factors such as traffic noise may be responsible for the observed association.

Compared with previous reports, this study has several important strengths: First, this population-based cohort study may be regarded as a natural experiment in which we took advantage of detailed residential histories to investigate the relationship between changes in traffic exposure status and the risk of CHD mortality. Changes in residential proximity to traffic were associated with an altered risk of CHD mortality in an exposure-response fashion.

Second, we used various road types (highway or major road) and distances ($\leq$50 or $\leq$150 m) from major roadways to assess residential proximity to traffic. The observed association was consistent across various combinations of road types and distances. The effect estimate was dependent on road types (traffic volume) and distances in a dose-response fashion (Table 3, Fig. 1).

Third, residential proximity to traffic was consistent with land-use-regression-model estimates for the concentrations of black carbon, nitrogen dioxide (NO_2), and nitric oxide (NO) (Table 2). These results are consistent with those of previous studies, and suggest that residential proximity to traffic is a simple and specific surrogate that reflects spatial variability of traffic-related air pollution.[5,6] In a separate analysis of associations between these 4 pollutants and the risk of CHD mortality, we found that an interquartile range elevation in the concentrations of black carbon was associated with a 6% (95% CI = 1.02–1.09) increase in the risk of CHD mortality after adjustment for all the covariates and 3 other copollutants ($PM_{2.5}$, NO_2, and NO); the corresponding relative risk for $PM_{2.5}$, NO_2, and NO was 1.00 (0.97–1.04), 1.04 (1.00–1.09), and 1.02 (0.97–1.08), respectively (Gan WQ, Koehoorn M, Daves HW, Demers PA, Tamburic L, Brauer M. Submitted paper).

Fourth, this study found that 47% of study subjects changed their residences at least once during the 9-year study period, leading to a change in the residential traffic exposure status in 15% of the subjects. When residential proximity to traffic at the original address (January 1994) was used to evaluate traffic exposure status (and subsequent residential relocations were ignored), the corresponding adjusted RRs (95% CI) for the 5 exposure categories were: 1.19 (1.10–1.29), 1.34 (1.10–1.64), 1.27 (1.13–1.42), 1.09 (0.99–1.21), and 1.06 (0.99–1.14) (Table 3, from the first to the fifth row). Thus, previous studies that have not accounted for residential relocation may have suffered from substantial exposure mis-

classification. This may result in underestimations of the true adverse health effects, and even false-negative results.

This study had several limitations that should be considered when interpreting these findings. The study cohort was constructed using linked administrative databases that did not include certain important information about individual cardiovascular risk factors (such as active or passive smoking status, body mass index, and individual SES). To partially control for these unmeasured risk factors, we adjusted for age, sex, neighborhood SES, and pre-existing comorbidities including diabetes, COPD, and hypertensive heart disease. Because these comorbidities and CHD share common behavioral risk factors, adjusting for these pre-existing comorbidities was presumably able to reduce the influence of uncontrolled factors, such as cigarette smoking, to some extent.[24] However, these approaches cannot eliminate all confounding effects caused by unmeasured cardiovascular risk factors.

Cigarette smoking is the single most important risk factor for CHD.[25] If smokers are more likely to live near (or move closer to) major roadways, the observed association may be confounded by the effects of cigarette smoking. However, previous epidemiologic studies have demonstrated that the association of air-pollution exposure with the severity of atherosclerosis[11,26] or the risk of CHD mortality[3,27] was independent of cigarette-smoking status and even stronger among never-smokers.[3,11,26,27] For example, Pope et al[3] reported that for each 10 $\mu g/m^3$ increase in annual average concentration of $PM_{2.5}$, the adjusted relative risk of CHD mortality was 1.22 for never smokers, 1.15 for former smokers, and 1.16 for current smokers. Given these findings and the lack of evidence to suggest that cigarette smoking is related to changes in residential proximity to traffic, it is less likely that the observed associations were due to confounding effects of cigarette smoking.

Low SES is a risk factor for CHD[28] and is also related to other cardiovascular risk factors such as cigarette smoking, obesity, and hypertension.[29–31] In some locations, people with low SES are more likely to live close to major roadways.[32] Individual SES is thus a possible confounder for the observed association. In the present study, we used neighborhood-income quintiles to approximate the major differences of economic status between subjects with various traffic-exposure profiles. Although this method may induce a degree of SES misclassification, some evidence has suggested that this approximation is acceptable for group comparisons.[33] In addition, some studies have found that neighborhood SES is associated with the risk of CHD independent of individual SES, indicating that adjustment for neighborhood SES may also reduce the influence of uncontrolled factors related to neighborhood disadvantages.[31] We used neighborhood income quintiles derived from the 2001 census data, which may not accurately reflect the original neighborhood SES for subjects who changed their residences during the exposure period (January 1994–December 1998). Nevertheless, there is evidence that the levels of neighborhood SES are well correlated for those who change their residences.[31]

Residential proximity to traffic is a relatively crude surrogate for exposure to traffic-related air pollution. Many factors, such as wind direction, presence of street canyons, and specific residence characteristics, may influence actual residential exposure levels.[34,35] Moreover, in the present study, residential proximity to traffic was estimated using the postal code centroid rather than the actual residential address. In urban areas, a 6-digit postal code typically represents one side of a city block or individual multiunit structures and is therefore fairly precise. Still, this assessment of traffic proximity will inevitably induce exposure misclassification. Furthermore, as in previous studies, our exposure assessment can only approximately reflect the exposure levels at subjects' residences, which may not precisely reflect actual individual exposure levels. Mobility,[36] outdoor activity, and indoor infiltration of air pollutants[37] may differ across study subjects. Nevertheless, all these factors presumably cause nondifferential exposure misclassification, leading to underestimations of the true adverse effects of residential proximity to traffic.

Finally, residential proximity to traffic signifies exposure not only to traffic-related air pollutants but also to traffic-related noise. Some studies have indicated that traffic-noise levels are at least moderately correlated with the concentrations of nitrogen oxides[38] and also with increased risk of CHD.[39] Therefore, it is possible that the increased risk of CHD mortality observed in the present study may be associated with both traffic-related air pollution and traffic noise. We cannot disentangle the effects of these 2 traffic-related pollutants in the current analysis.

An enormous number of people are regularly exposed to traffic; therefore, traffic-related air pollution may represent an important public-health problem. Using a large population-based cohort study with detailed residential history information, we observed that living close to traffic was associated with an increased risk of coronary mortality, whereas moving away from traffic was associated with a decreased risk.

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;121:2331–2378.
2. Pope CA III, Dockery DW. Health effects of fine particulate air pollution: lines that connect. *J Air Waste Manag Assoc*. 2006;56:709–742.
3. 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;109:71–77.
4. Miller KA, Siscovick DS, Sheppard L, et al. Long-term exposure to air pollution and incidence of cardiovascular events in women. *N Engl J Med*. 2007;356:447–458.

5. 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;14:228–239.
6. Zhu Y, Hinds WC, Kim S, Sioutas C. Concentration and size distribution of ultrafine particles near a major highway. *J Air Waste Manag Assoc*. 2002;52:1032–1042.
7. Kunzli N, Kaiser R, Medina S, et al. Public-health impact of outdoor and traffic-related air pollution: a European assessment. *Lancet*. 2000;356: 795–801.
8. Zhou Y, Levy JI. Factors influencing the spatial extent of mobile source air pollution impacts: a meta-analysis. *BMC Public Health*. 2007;7:
9. Adar SD, Kaufman JD. Cardiovascular disease and air pollutants: evaluating and improving epidemiological data implicating traffic exposure. *Inhal Toxicol*. 2007;19:135–149.
10. Jerrett M, Arain A, Kanaroglou P, et al. A review and evaluation of intra-urban air pollution exposure models. *J Expo Anal Environ Epidemiol*. 2005;15:185–204.
11. Hoffmann B, Moebus S, Mohlenkamp S, et al. Residential exposure to traffic is associated with coronary atherosclerosis. *Circulation*. 2007; 116:489–496.
12. Allen RW, Criqui MH, Diez Roux AV, et al. Fine particulate matter air pollution, proximity to traffic, and aortic atherosclerosis. *Epidemiology*. 2009;20:254–264.
13. Kan H, Heiss G, Rose KM, Whitsel EA, Lurmann F, London SJ. Prospective analysis of traffic exposure as a risk factor for incident coronary heart disease: the Atherosclerosis Risk in Communities (ARIC) study. *Environ Health Perspect*. 2008;116:1463–1468.
14. Tonne C, Melly S, Mittleman M, Coull B, Goldberg R, Schwartz J. A case-control analysis of exposure to traffic and acute myocardial infarction. *Environ Health Perspect*. 2007;115:53–57.
15. Gehring U, Heinrich J, Kramer U, et al. Long-term exposure to ambient air pollution and cardiopulmonary mortality in women. *Epidemiology*. 2006;17:545–551.
16. Beelen R, Hoek G, van den Brandt PA, et al. Long-term effects of traffic-related air pollution on mortality in a Dutch cohort (NLCS-AIR study). *Environ Health Perspect*. 2008;116:196–202.
17. Rosenlund M, Bellander T, Nordquist T, Alfredsson L. Traffic-generated air pollution and myocardial infarction. *Epidemiology*. 2009;20:265–271.
18. Rosenlund M, Picciotto S, Forastiere F, Stafoggia M, Perucci CA. Traffic-related air pollution in relation to incidence and prognosis of coronary heart disease. *Epidemiology*. 2008;19:121–128.
19. Nafstad P, Haheim LL, Wisloff T, et al. Urban air pollution and mortality in a cohort of Norwegian men. *Environ Health Perspect*. 2004;112:610–615.
20. Brauer M, Lencar C, Tamburic L, Koehoorn M, Demers P, Karr C. A cohort study of traffic-related air pollution impacts on birth outcomes. *Environ Health Perspect*. 2008;116:680–686.
21. Henderson SB, Beckerman B, Jerrett M, Brauer M. Application of land use regression to estimate long-term concentrations of traffic-related nitrogen oxides and fine particulate matter. *Environ Sci Technol*. 2007; 41:2422–2428.
22. Hole DJ, Watt GC, Davey-Smith G, Hart CL, Gillis CR, Hawthorne VM. Impaired lung function and mortality risk in men and women: findings from the Renfrew and Paisley prospective population study. *BMJ*. 1996;313:711–715.
23. Pearson TA, Blair SN, Daniels SR, et al; American Heart Association Science Advisory and Coordinating Committee. 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;106:388–391.
24. Pope CA III, Ezzati M, Dockery DW. Fine-particulate air pollution and life expectancy in the United States. *N Engl J Med*. 2009;360:376–386.
25. Ockene IS, Miller NH; For the American Heart Association Task Force on Risk Reduction. Cigarette smoking, cardiovascular disease, and stroke: a statement for healthcare professionals from the American Heart Association. *Circulation*. 1997;96:3243–3247.
26. Kunzli N, Jerrett M, Mack WJ, et al. Ambient air pollution and atherosclerosis in Los Angeles. *Environ Health Perspect*. 2005;113: 201–206.
27. Pope CA III, Burnett RT, Thun MJ, et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA*. 2002;287:1132–1141.
28. Kaplan GA, Keil JE. Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation*. 1993;88:1973–1998.
29. Luepker RV, Rosamond WD, Murphy R, et al. ; The Minnesota Heart Survey. Socioeconomic status and coronary heart disease risk factor trends. *Circulation*. 1993;88:2172–2179.
30. Auer JW, Berent R, Eber BC. Low socioeconomic status and coronary artery disease. *Lancet*. 2002;359:979–980.
31. Diez Roux AV, Merkin SS, Arnett D, et al. Neighborhood of residence and incidence of coronary heart disease. *N Engl J Med*. 2001;345:99–106.
32. Gunier RB, Hertz A, Von Behren J, Reynolds P. Traffic density in California: socioeconomic and ethnic differences among potentially exposed children. *J Expo Anal Environ Epidemiol*. 2003;13:240–246.
33. Domínguez-Berjón F, Borrell C, Rodríguez-Sanz M, Pastor V. The usefulness of area-based socioeconomic measures to monitor social inequalities in health in Southern Europe. *Eur J Public Health*. 2006; 16:54–61.
34. Xie SD, Zhang YH, Li Q, Tang XY. Spatial distribution of traffic-related pollutant concentrations in street canyons. *Atmos Environ*. 2003;37: 3213–3224.
35. Restrepo C, Zimmerman R, Thurston G, et al. A comparison of ground-level air quality data with New York State Department of Environmental Conservation monitoring stations data in South Bronx, New York. *Atmos Environ*. 2004;38:5295–5304.
36. Nethery E, Leckie SE, Teschke K, Brauer M. From measures to models: an evaluation of air pollution exposure assessment for epidemiological studies of pregnant women. *Occup Environ Med*. 2008;65:579–586.
37. Hystad PU, Setton EM, Allen RW, Keller PC, Brauer M. Modeling residential fine particulate matter infiltration for exposure assessment. *J Expo Sci Environ Epidemiol*. 2009;19:570–579.
38. Davies HW, Vlaanderen JJ, Henderson SB, Brauer M. Correlation between co-exposures to noise and air pollution from traffic sources. *Occup Environ Med*. 2009;66:347–350.
39. Babisch W, Beule B, Schust M, Kersten N, Ising H. Traffic noise and risk of myocardial infarction. *Epidemiology*. 2005;16:33–40.