LONG-TERM EXPOSURE TO TRAFFIC NOISE AND TRAFFIC-RELATED AIR POLLUTION AND CORONARY HEART DISEASE MORTALITY

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Background and Aims: In metropolitan areas, road traffic is a major contributor to ambient air pollution, and also the dominant source of environmental noise. Therefore, observed associations between air pollution and adverse cardiovascular outcomes may be confounded by coexistent traffic noise or vice versa. This population-based cohort study investigated the independent and joint effects of traffic noise and traffic-related air pollution on coronary heart disease (CHD) mortality.

Methods: This 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 = 445,795). Individual exposures to traffic noise and traffic-related air pollutants including black carbon, PM$_{2.5}$, NO$_2$, and NO were estimated at subjects' residences using a noise prediction model (Cadna A) and land use regression models, respectively. CHD deaths during the follow-up period were identified from the provincial death registration database.

Results: Traffic noise level was moderately correlated with black carbon (0.44), but weakly correlated with PM$_{2.5}$ (0.14), NO$_2$ (0.33), and NO (0.39). After adjusting for baseline age, sex, preexisting comorbidity, neighborhood socioeconomic status, PM$_{2.5}$, NO$_2$, and black carbon, a 10 dBA elevation in residential noise was associated with a 9% (95% CI: 1-17%) increase in CHD mortality; subjects in the highest noise decile (>70 dBA) had a 19% (95% CI: 2-40%) increase in CHD mortality compared with those in the lowest decile (≤57 dBA). After similar adjustments including noise, an interquartile range ($0.97\times10^{-5}/m$) elevation in black carbon concentration was associated with a 4% (95% CI: 1-8%) increase in CHD mortality. We did not find any interaction between traffic noise and black carbon on CHD mortality.

Conclusions: Traffic noise and black carbon may both be independently responsible for observed associations between exposure to road traffic and adverse cardiovascular outcomes.