Road traffic noise: A risk factor for myocardial infarction?

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SUMMARY
A population-based cohort study was done to establish an association between road traffic noise and incident myocardial infarction (MI). The study was based on a Diet, Cancer and Health cohort. The cohort comprised 57 053 residents of Copenhagen and Aarhus, Denmark who were aged 50– 64 years. Of these, 566 and 900 residents were excluded due to the presence of cancer and coronary artery disease, respectively. After further excluding persons with incomplete residential address and missing data on covariates, a total of 50 614 participants were studied. The enrolment was done from 1993 to 1997. Baseline characteristics such as sex, smoking status, smoking duration, smoking intensity, intake of fruit, vegetables and alcohol, body mass index, physical activity, calendar year, education, railway and airport noise, air pollution, baseline diastolic and systolic blood pressure, total cholesterol and self-reported diabetes were assessed at the time of enrolment and considered as potential confounders. The average follow-up period was 9.8 years.

Exposure to road traffic noise, for all present and historical residential cohort members who lived between 1988 and event/censoring, was calculated for the years 1990, 1995, 2000 and 2005. The road traffic noise was calculated by the Nordic prediction method as the equivalent continuous A-weighted sound pressure level (L_{Aeq}). ‘A-weighting’ is a widely used scale to measure sound pressure levels, which correlates with the subjective response of the auditory system. It was expressed as L_{den}—a composite index of noise exposure during day (L_{d}), evening (L_{e}) and night (L_{n}). The exposure to railway noise from 1993–2000 was calculated by the same method. The information available from local authorities about noise zones (5 dB categories) was used to determine the noise impact from airports and airfields. The concentration of oxides of nitrogen (NOx) in the air for each year (1998– 2006) at each address was calculated by the Danish AirGIS modelling system.

The outcome was either incident MI (ICD 10: 121.0–121.9) identified from the Danish National Hospital Delivery and Danish Causes of Death Registry, or sudden cardiac death caused by an MI (ICD 10: 146.0–146.9) after validation by medical records. Cases with MI were ascertained from a review of the medical records from baseline through 2003, and as a diagnosed case from ward thereafter. Fatal MI was diagnosed as death within 30 days of diagnosis.

Left truncation at age of enrolment and right censoring at the age of MI (event), death, emigration or end of follow-up, whichever came first, was done. Left truncation means that the individuals who already had the event, viz. MI, were not included in the study. Right censoring means that there were individuals in whom the event did not occur till the end of the follow-up period of the study. The Cox proportional hazards model with age as the underlying time was used for analysis. The exposure to road traffic noise and NOx at a given age were calculated as time-weighted averages for the preceding 5 years (considering all present and historical addresses in that period) or as yearly exposure at the residence. Time-weighted averages were used to estimate a person’s daily exposure to road traffic noise and NOx, taking into account the average levels of noise and NOx and time spent in each area. To establish the association between MI and road traffic noise, incidence rate ratios (IRRs) were calculated for yearly road traffic noise at diagnosis, and time-weighted mean road traffic noise for 5 years preceding diagnosis. The association between exposure to road traffic noise and incident MI was estimated after adjusting for confounders stated earlier.

There was a strong positive correlation (R_{Spearman} =0.96, p=0.0001) between the distribution of road traffic noise exposure (L_{Aeq}) at the enrolment address and the time-weighted 5-year mean L_{den} preceding enrolment (considering historical addresses). The L_{Aeq} and NOx in the study period (R_{Spearman} =0.62, p=0.0001) were also significantly correlated.

High risk for MI was associated with higher level of road traffic noise in a linear dose–response manner. Every 10 dB increase in road traffic noise led to a 12% higher risk for MI. For cases with fatal MI, the IRR per 10 dB yearly increase in road traffic noise was 1.25 (95% CI 1.07–1.46) which reduced to 1.17 (95% CI 0.96–1.43) after adjustment for confounders. There was no significant effect modification present between road traffic noise and incident MI across the strata of exposure variables such as age, sex, smoking status, years of education and exposure to railway noise.

The authors concluded that residential exposure to road traffic noise was positively associated with a risk for MI in a linear dose–response manner.
COMMENT

This study is one of the first to establish an association of road traffic noise with incident MI. A cohort study design is good to study the association between a disease and a potential risk factor. The study population, risk factors and outcome were clearly addressed and potential confounders were adjusted. Only those cases for which full information was available were included in the analysis. The baseline characteristics of missing data and attritions should have been reported to make a comparison with the remaining cohort. Further, as this study was a sub-study of the Diet, Cancer and Health cohort, patients with cancer were excluded at the time of recruitment of the cohort. Although this number was about 1%, it is difficult to say how this would have affected the results of the study.

Industrialization, urbanization and increasing vehicular growth rate have resulted in escalating noise pollution to which road traffic noise contributes the most. Inordinate noise exposure has detrimental effects on cardiovascular, respiratory, neuroendocrine and digestive systems, and also results in fatigue and loss of efficiency at work.

In India, coronary heart disease is considered an epidemic condition, and one of the major causes of disease burden and deaths. Its risk factors include obesity, hypertension, high cholesterol, low high-density lipoprotein (HDL) cholesterol and diabetes, which are present more in urban areas and their prevalence has increased significantly in urban (R² 0.45–0.74) and slowly in rural areas (R² 0.19–0.29).²

Studies have documented that noise pollution is also escalating in India. One such study proved that the population in the town of Asansol was exposed to significantly high noise levels, which was caused mostly by road traffic.³Studies from Lucknow, Balasore, Surat and Kolhapur have also reported that noise levels were higher than permissible limits.⁴⁻⁵ In a study from Delhi, the time-averaged noise spectra revealed that noise intensities were significantly higher in the frequency range of 0.5–2 kHz for all types of vehicles.⁶

Studies involving measurement of environmental noise levels are fraught with a certain degree of uncertainty. This study is from Denmark, and cannot be generalized. Further, the biological plausibility of the relationship between road traffic noise and incident MI, though explained by the authors of the article, may find detractors. Despite these caveats, two points need to be highlighted, viz. the incidence of coronary artery disease is increasing, and that road traffic noise is potentially modifiable. Public health interventions often prioritize conditions which are serious, increasing in magnitude and have potentially modifiable risk factors. Coronary artery disease is one such condition. The present study does make a case for considering the inclusion of road traffic noise as a potential risk factor for this condition. The beneficial effect of controlling road traffic noise on other documented adverse health outcomes would be an added bonus.

In India, though studies have documented high noise levels, few reports have been published on their harmful effect on health. Among all noise-generating sources in a study in the city of Jaipur, road traffic was the major source of noise, followed by factory/machines. About 52% of the population was suffering from frequent irritation, 46% of the respondents felt that they had high blood pressure and 48.6% observed loss of sleep due to noise pollution. The Lₚₙ values were higher (range 73–86) compared to the permissible values of 65 dB(A).⁷

One module on noise pollution and its control has documented the impact of noise on health which includes annoyance, affecting blood pressure, pulse rate and blood cholesterol; loss of hearing and loss of concentration affecting work performance; neurological effects including pain, ringing in the ears, feeling of tiredness and sleeplessness.⁸

A study on traffic noise assessment and its negative health effect on roadside residents showed that noise has a significant (α=0.05) effect on hearing loss, sleep disturbances, abnormal heart beat and speech communication.⁹ Singh and Davar also support these results, adding stress, high blood pressure, distraction affecting productivity and a general reduction in the quality of life as the impact of noise pollution on human health.¹⁰

The limited studies done so far in India show that noise pollution has an adverse effect on the health of exposed residents. Hypertension, abnormal heartbeat and stress are among those documented adverse effects of noise pollution that may result in MI. It also needs to be mentioned that the role of noise pollution could be that of a causative, contributory or aggravating factor for subclinical heart disease. This study cannot answer this question.

In view of paucity of data in this area from India, more studies need to be done in various parts of the country to generate an evidence-base to inform public health interventions. Further research is indicated to document the detrimental effects of road traffic noise on health.

REFERENCES


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