Associations of road traffic noise with mortality and hospital admissions in London

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Summary

Background and aims

Previously published studies have found associations of road noise with hypertension, which is a risk factor for cardiovascular disease, especially for stroke. We aimed to examine the chronic effects of road traffic noise on mortality and hospital admissions for cardiovascular disease and stroke in a large general population.

Methods

The study population consisted of 8.61 million inhabitants in London. We assessed small-area level associations of day- (7:00-22:59) and night-time (23:00-06:59) road traffic noise with all-cause and cardiovascular mortality and cardiovascular hospital admissions in all adults (25+ years) with Poisson regression models applying the Integrated Nested Laplace Approximation (INLA) approach in the Bayesian framework. We adjusted the models for age and sex, area-level deprivation, ethnicity, smoking, air pollution and a random effect.

Results

Mean daytime exposure to road traffic noise was 55.6 dB. Daytime noise was associated with all-cause and cardiovascular mortality in adults; relative risks (RR) for all-cause mortality were 1.04 (95% CI 1.00-1.07) in areas with daytime road noise >60 dB vs. <55 dB. Exposure to daytime road traffic noise also increased the risk of hospital admission for stroke with RR 1.05 (95% CI 1.02-1.09) in areas >60 dB vs. <55 dB. Night-time noise was not associated with road traffic noise in adults of all ages.

Conclusions

This is the largest study to date to investigate environmental noise and cardiovascular disease. It suggests that road traffic noise is associated with small increased risks of all-cause mortality and cardiovascular disease.

1. Introduction

The environmental burden of disease from traffic noise was recently estimated to be the second largest preceded only by airborne particulate matter.[1] In Europe's largest city, London, over 1.6 million people are exposed to daytime road traffic noise levels >55 decibels (dB)[2] which the WHO defines as a level of community noise that causes health problems,[3] and a level that in the United Kingdom has been estimated to annually cause over 500 additional cases of hypertension-related myocardial infarctions and nearly 800 cases of stroke.[4]

Health effects of noise exposure are hypothesised to occur via several pathways.[5, 6] Exposure to noise may affect the autonomic nervous system increasing heart rate, blood pressure and concentrations of noradrenaline, a stress hormone.[7] Noise can also affect the hypothalamus-pituitary-adrenal axis leading to increased levels of cortisol, another stress hormone.[5] In the long-term these reactions are suggested to promote low grade inflammation and cardiovascular morbidity.[8] Another suggested pathway is via sleep disorders[6, 7] some of which have been linked to an increased risk of mortality.[9] Recently, long-term exposure to night-time road traffic noise was linked to development of atherosclerosis,[10] the pathology underlying a range of important cardiovascular diseases.

Epidemiological studies have reported associations between chronic exposure to environmental noise and health outcomes including annoyance, sleep problems, increase in blood pressure, and hypertension.[7, 11-13] Few studies, however, have specifically examined associations between road traffic noise and more severe health outcomes such as hospital admissions and mortality from cardiovascular diseases. Of these studies most[14-19] but not all[20,21] have reported positive associations. The independent long-term effects of road traffic noise and air pollutants remain to be confirmed,[22] and there is a lack of studies identifying susceptible population subgroups.[1, 5]

Therefore, among 8.6 million Londoners, we aimed to determine small-area effects of long-term exposure to road traffic noise, independent of air pollution, on all-cause and cardiovascular mortality as well as on cardiovascular hospital admissions in adult populations.

2. Methods

2.1 Study area and population

The study area was London, defined as the area of Greater London inside the M25 orbital motorway. We used small area census geographical units called Census Output Areas (COA) with a mean population of 300 (>40 households)[23] for the hospital admission analyses, and census geographical unit Lower Layer Super Output Areas (LSOA) with a mean population of 1500 (>400 households)[24] for the mortality analyses. We included 27 686 COAs and 5358 LSOAs in the analyses.

2.2 Data

We used the first registered emergency hospital episode of each year and underlying cause of death for mortality for the following (International classification of diseases 10th revision ICD10) codes: all cardiovascular causes (I00–I99), ischemic heart disease (IHD, I20-I25), and stroke (I61, I63, I64) and also, examined deaths from all natural causes (A00-R99). These data include age, sex, and postcode of residential address at the time of admission or death. We used annual mid-year population estimates by sex and 5-year age bands to calculate standardised mortality and admission rates for each area.

Hospital admission data were derived from Hospital Episode Statistics and are held by the UK Small Area Health Statistics Unit (SAHSU). The mortality and population data were supplied by the Office for National Statistics (ONS), derived from the national mortality registrations and the Census and are

held by SAHSU. Data use was covered by approval from the National Research Ethics Service - reference 12/LO/0566 and 12/LO/0567 - and by National Information Governance Board and Ethics and Confidentiality Committee approval for section 251 support (NIGB - ECC 2-06(a)/2009).

2.3 Traffic noise exposure

Annual road traffic noise levels for the years 2003-2010 were modelled at geometric centroids of ~190 000 postcode locations using the TRAffic Noise EXposure (TRANEX)[2] model. TRANEX was used to estimates noise levels at 1m from the facade of the nearest residential dwelling, with 0.1 dB(A) noise level resolution. Exposures to day-(7:00-22:59) and night- (23:00-06:59) time noise were estimated using $L_{Aeq,16hr}$ and L_{night}. For the hospital admission analyses, noise data were aggregated to COAs by taking the median across all annual postcode address centroid noise levels within a COA, and median of these annual medians COA-level values over 2003-2010. Similarly we calculated LSOA level noise estimates for the mortality analyses. Noise estimates were categorised by 5dB increments – <55 (reference), 55-60, and >60dB – as well as by tertiles.

2.4 Confounders

We adjusted for socio-economic deprivation of the area (Carstairs deprivation index, based on unemployment, overcrowding, car ownership, and low social class), an area-level smoking proxy (smoothed age-sex standardized relative risks for lung cancer mortality (ICD10: C33-C34) area percentage of black and South Asian ethnicity (from UK Census data supplied by the Office for National Statistics) and $PM_{2.5}$ concentrations (air pollution data were supplied by the Environmental Research Group at Kings College London).

2.5 Statistical methods

We used ecological Poisson regression models specified in a Bayesian framework that can be implemented through the Integrated Nested Laplace Approximation (INLA) approach, that modelled spatial dependencies between nearby areas. We calculated age and sex standardised expected number of deaths and hospital admissions for each small area and included these as offsets in the models.

We performed all analyses for adults (≥25 years).

3. Results

In the study population of 8.61 million, there were a total of 442 560 deaths among adults (≥25 years), between 2003 and 2010. The total number of hospital admissions from cardiovascular causes was 400 494.

Median daytime exposure at both COA and LSOA level was 55.6 dB, for night-time exposure these figures were 50.2 dB and 50.1 dB, respectively. A map of the study area and the distribution of daytime noise levels are provided in Figure 1. Little variation was observed in noise levels by quintiles of area-level deprivation or tertiles of ethnicity. Correlations between linear day- and night-time road noise estimates were 0.99 at both COA and LSOA level. Correlations between road noise and $PM_{2.5}$ ranged from 0.39 to 0.45, and those between noise and NO_x from 0.42 to 0.48.

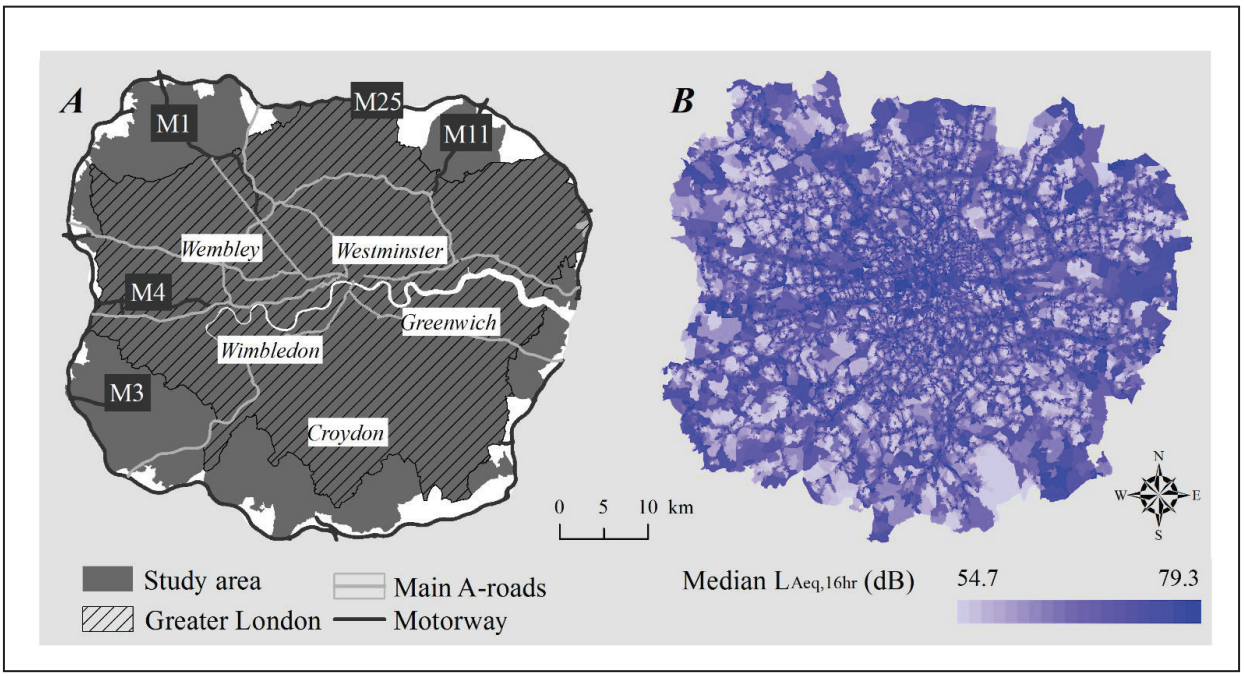

Figure 1 Map of the study area (A) and distribution of median daytime road traffic noise at Census Output Area level (B).

Daytime road traffic noise was significantly associated with all-cause and cardiovascular mortality in adults. Relative risks relative risks (RR) for all-cause mortality were 1.04 (95% CI 1.00-1.07) in areas with daytime road noise >60 dB vs. <55 dB. Associations were mainly positive but non-significant for daytime noise and cardiovascular, IHD, and stroke mortality, but null for night-time noise.

Exposure to daytime road traffic noise increased the risk of hospital admission for stroke with RR 1.05 (95% CI 1.02-1.09) in areas >60 dB vs. <55 dB.

Night-time noise was also associated with increased risk of stroke admission, but not with the other admission outcomes.

4. Conclusions

In the Europe's largest city we found that long-term exposure to daytime road traffic noise was associated with small increased risks of all-cause mortality and cardiovascular disease in the general population.

This was an ecological study design and it is possible that the population-level associations we observed are not representative of those at individual-level. In addition, residual confounding is likely to affect the results as we were only able to adjust for confounders at area- and not individual level. Also, the exposure model used is likely to over-estimate noise at low exposure levels and under-estimate noise in areas with heavily trafficked minor roads, although use of noise categories in analyses will have helped reduce exposure misclassification.

There are few studies with mixed findings[15,16,18,20,21] examining road traffic noise in relation to cardiovascular outcomes to be able to compare our study with. However, our findings are consistent with the larger body of evidence linking traffic noise exposure with hypertension[11].

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