Aircraft noise and cardiovascular disease near Heathrow airport in London: small area study

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Abstract

Objective To investigate the association of aircraft noise with risk of stroke, coronary heart disease, and cardiovascular disease in the general population.

Design Small area study.

Setting 12 London boroughs and nine districts west of London exposed to aircraft noise related to Heathrow airport in London.

Population About 3.6 million residents living near Heathrow airport. Risks for hospital admissions were assessed in 12 110 census output areas (average population about 300 inhabitants) and risks for mortality in 2378 super output areas (about 1500 inhabitants).

Main outcome measures Risk of hospital admissions for, and mortality from, stroke, coronary heart disease, and cardiovascular disease, 2001-05.

Results Hospital admissions showed statistically significant linear trends (P<0.001 to P<0.05) of increasing risk with higher levels of both daytime (average A weighted equivalent noise 7 am to 11 pm, \( L_{Aeq,16h} \)) and night time (11 pm to 7 am, \( L_{night} \)) aircraft noise. When areas experiencing the highest levels of daytime aircraft noise were compared with those experiencing the lowest levels (>63 dB \( v \leq 51 \) dB), the relative risk of hospital admissions for stroke was 1.24 (95% confidence interval 1.08 to 1.43), for coronary heart disease was 1.21 (1.12 to 1.31), and for...
cardiovascular disease was 1.14 (1.08 to 1.20) adjusted for age, sex, ethnicity, deprivation, and a smoking proxy (lung cancer mortality) using a Poisson regression model including a random effect term to account for residual heterogeneity. Corresponding relative risks for mortality were of similar magnitude, although with wider confidence limits. Admissions for coronary heart disease and cardiovascular disease were particularly affected by adjustment for South Asian ethnicity, which needs to be considered in interpretation. All results were robust to adjustment for particulate matter (PM$_{2.5}$) air pollution, and road traffic noise, possible for London boroughs (population about 2.6 million). We could not distinguish between the effects of daytime or night time noise as these measures were highly correlated.

Conclusion High levels of aircraft noise were associated with increased risks of stroke, coronary heart disease, and cardiovascular disease for both hospital admissions and mortality in areas near Heathrow airport in London. As well as the possibility of causal associations, alternative explanations such as residual confounding and potential for ecological bias should be considered.

Methods

We carried out analyses comparing rates of hospital admissions for cardiovascular disease and mortality in neighbourhoods (small areas) exposed to different levels of aircraft noise related to Heathrow airport. We used a standard noise metric, the $A_{eq}$ sound pressure level ($L_{eq}$), denoted as $L_{eq}$. The human ear is more sensitive to some frequencies than others. The $L_{eq}$ deviates lower frequencies compared with medium and higher frequencies, and uses a set of mathematical curves to adjust the sound pressure level to the relative loudness perceived by human hearing. We defined daytime noise ($L_{eq, day}$) as the average $A$ weighted equivalent noise from 7 am to 11 pm and night time noise ($L_{eq, night}$) from 11 pm to 7 am.

Study area and population

The study area comprised 12 London boroughs and nine districts west of London exposed to aircraft noise related to Heathrow airport, defined as being partly or wholly within the 2001 50 dB noise contour for Heathrow airport during the daytime ($L_{eq, day}$) supplied by the Civil Aviation Authority (fig 1). Additionally, we had confounder data for particulate air pollution and road traffic noise for the 12 London boroughs (data for districts outside London were not readily comparable with the data available for London).

We defined neighbourhoods (small areas) by using the national census geographical units, which are census output areas and super output areas. The study area comprised 12 110 census output areas (average 297 inhabitants, area 0.13 km$^2$) and 2378 super output areas (1510 inhabitants, area 0.65 km$^2$). The census output area as the unit of analysis for hospital admissions and the super output area, an aggregate of on average five census output areas, for mortality as the numbers of deaths were insufficient for meaningful analyses at census output area level. We used Office for National Statistics annual mid-year population estimates by age and sex for 2001-05 at London borough or district level, which we then disaggregated to census output areas and super output areas using the UK 2001 census age-sex distribution.

Aircraft noise data

From the Civil Aviation Authority we obtained aircraft noise data related to Heathrow airport for 2001 on 10 m × 10 m grids. The noise data had been modelled using the UK Civil Aircraft Noise Contour Model ANCON, which uses information on flight paths of arriving and departing aircraft along with factors such as height, speed, and engine power to derive noise at ground level.

We calculated population weighted annual average noise levels for daytime and night time aircraft noise for census output areas and super output areas. This was done because the noise grid was smaller than the area of the census output area or super output areas and populations are not evenly distributed (for example, a census output area has on average 125 addresses and six postcodes that may cluster to one or other side of the census output area) so a simple area averaging would not accurately represent population exposures (see supplementary appendix).

Health data

We extracted post coded data on hospital admissions (main reason for admission, first episode of stay in a given year) and deaths (by underlying cause) for the study area, 2001-05, from Office for National Statistics and Department of Health data held by the UK Small Area Health Statistics Unit at Imperial College London. Data were obtained for stroke (ICD-10 codes I61, I63-I64, international classification of diseases, 10th revision), coronary heart disease (ICD-10 I20-I25), and cardiovascular disease (ICD-10 Chapter I) and then linked these by postcode (average 23 households) to census output area and super output area.

Data on potential confounders

We included ethnicity, deprivation, and a smoking proxy at census output area and super output area level as potential
confounders. Area level ethnic composition and deprivation from the 2001 census were obtained from the Office for National Statistics. For the two major ethnic groups in London, we categorised areas by South Asian ethnicity (census term “Asian or Asian British,” for which we included only “Indian,” “Pakistani,” and “Bangladeshi”) and black ethnicity (census term “Black or Black British,” which includes “Black Caribbean,” “Black African,” and “Other Black”). We used the following cut points: the national average (%) for England and Wales at census output area level (4% for South Asian, 2% for black ethnicity), double the national average (8%, 4%), and 50% South Asian or black ethnicity—areas where these comprised the majority ethnic group. This gave us four categories for each ethnicity, where the reference categories were less than or equal to the national average (%) for that ethnic group (≤4% for South Asian and ≤2% for black ethnicity). The deprivation score used was Carstairs index, categorised in fifths. As a proxy measure for area level smoking we used smoothed lung cancer mortality (ICD-10 codes C33-C34) relative risk estimates, 2005, for census output areas and super output areas,23 since data on individual smoking or smoking prevalence were not available.

For the 12 London boroughs within the study area we also obtained data on air pollution and daytime road noise. For air pollution, the Environmental Research Group at King’s College London provided estimates of annual mean particulate matter of 10 microns or less (PM$_{10}$) at spatial resolution of 20 m × 20 m for 2001, using dispersion modeling as detailed in the London Emissions Toolkit and London Air Pollution Toolkit.24 We obtained data on daily average road traffic noise for 2001 from the Department for Environment, Food and Rural Affairs (Defra), expressed in continuous A weighted equivalent sound pressure levels ($\text{L}_{\text{eq},16\text{h}}$) on 10 m × 10 m grids at 1 dB resolution between ≥50 dB and ≤75 dB. Road traffic noise data (major roads) had been generated to comply with the European Noise Directive 2002/49/EC (http://ec.europa.eu/environment/noise/directive.htm) and modeled using the calculation of road traffic noise method at a height of 4 m above ground using characteristics of the road network.23 We linked the air pollution and road noise data to census output area and super output area using population weighting (see supplementary appendix).

Statistical analyses

Correlations between aircraft noise and potential confounders were assessed using Goodman Kruskal tau rank correlation coefficients.

For the entire study area we carried out a small area analysis of aircraft noise and the three cardiovascular outcomes, adjusted for potential confounders at area level (census output area or super output area): age, sex, South Asian and black ethnicity, deprivation, and smoking proxy (lung cancer mortality risk). We conducted a sensitivity analysis for the 12 London boroughs (London area) additionally including particulate air pollution (PM$_{10}$) and road noise as potential confounders.

We grouped daytime aircraft noise and road noise into six categories from ≤51 to >63 dB in increments of 3 dB, which represents a doubling in sound intensity that is just perceptible as a change in loudness to the human ear. For aircraft noise, 57 dB $\text{L}_{10\text{min}}$ is taken as the point at which noticeable community annoyance starts to occur25 26; the Civil Aviation Authority attempts to minimise areas exposed to this level of noise or higher, measured as the daytime $\text{L}_{10\text{min}}$, over a 92 daytime summer period.27 Our $\text{L}_{10\text{min}}$ aircraft noise categories include a 57 dB cut point, although we use an annual not summertime average (fig 1). Night time aircraft noise affected fewer areas (fig 1), and 5 dB categories (≤50, >50-55, and >55 dB) were used.

To aid comparisons between daytime and night time aircraft noise, we also ran daytime analyses using the same 5 dB categories. The correlation between daytime and night time aircraft noise categories was almost perfect ($\tau \geq 0.98$, see supplementary table 2) so we did not include these together in the statistical models, but analysed them separately.

To allow for small numbers and unstable rates of hospital admissions and mortality we used random effects models to produce smoothed relative risk maps. To examine the effects of noise we fitted Poisson regression models with an additional random effect term to account for over-dispersion and residual heterogeneity, using the R software (www.r-project.org) and tested for linear trend across noise categories using the median noise value for each category.

Results

Figure 1 shows the study area; the population (2001 census) was 3.6 million. During 2001-05, 189 226 first episodes of hospital stay in a given year for cardiovascular disease (16 983 stroke, 64 448 coronary heart disease) and 48 347 cardiovascular disease related deaths (9803 stroke, 22 613 coronary heart disease) occurred in the study area (table 1). Supplementary figures 1 and 2 show the maps of hospital admissions at census output area level and mortality at super output area level, respectively. Only 2% or fewer of the study population lived in areas exposed to the highest category of daytime (>63 dB) or night time (>55 dB) aircraft noise (see supplementary table 1). The area affected by night time noise was less extensive than that for daytime noise (fig 1). Supplementary figure 3 shows the spatial distributions of the confounder data. Areas with a high proportion of South Asian and black ethnicity population were concentrated in the north eastern and eastern part of the study area, respectively, which were also areas with higher deprivation and higher risks of lung cancer. Within the London area, higher levels of PM$_{10}$ were found in the eastern part towards central London; distributions of both PM$_{10}$ and road noise differed from that of aircraft noise (supplementary figure 3 and figure 1). Correlations between aircraft noise and potential confounders are shown in supplementary table 2 where $\tau=1$ denotes perfect positive correlation and $\tau=-1$ denotes perfect negative correlation. Correlations between confounders and aircraft noise were all ≤0.30. In the London boroughs, aircraft noise was modestly correlated with PM$_{10}$ ($\tau=0.2$ for daytime noise and $\tau=-0.3$ for night time noise) but not with road traffic noise ($\tau \leq 0.02$).

Hospital admissions

Figure 2 and supplementary table 3 show the results for hospital admission for daytime and night time noise adjusted for age and sex, and with additional adjustment for ethnicity, deprivation, and the smoking proxy. For each of stroke, coronary heart disease, and cardiovascular disease the pattern was of increasing risk of admission with increasing aircraft noise, and all linear tests for trend were statistically significant (P<0.001 to P<0.05). The risk of coronary heart disease in particular, and to a lesser extent cardiovascular disease, was noticeably reduced by adjustment for multiple confounders, in particular South Asian ethnicity.

In multiple adjustment models, for daytime aircraft noise (>63 dB $\nu \leq 51$ dB) the relative risk for stroke was 1.24 (1.08 to 1.43),...
for coronary heart disease was 1.21 (1.12 to 1.31), and for cardiovascular disease was 1.14 (1.08 to 1.20). Corresponding relative risks for night time noise (>55 dB v ≤50 dB) were 1.29 (1.14 to 1.46), 1.12 (1.04 to 1.20), and 1.09 (1.04 to 1.14). Results using the same categories for daytime as for night time noise (supplementary table 3) suggested higher relative risks for night time noise.

Mortality

Figure 3i and supplementary table 4 show the results for mortality for daytime and night time noise. The relative risks of mortality were numerically similar to those for hospital admissions at the higher noise levels, although confidence intervals were wider, reflecting the smaller numbers of events. In multiple adjusted models, for daytime aircraft noise (>63 dB v ≤51 dB) the relative risk for stroke mortality was 1.21 (95% confidence interval 0.98 to 1.49), for coronary heart disease was 1.15 (1.02 to 1.30), and for cardiovascular disease was 1.16 (1.04 to 1.29). The corresponding relative risks for night time aircraft noise (>55 dB v ≤50 dB) were 1.23 (1.02 to 1.49), 1.11 (0.99 to 1.24), and 1.14 (1.03 to 1.26). Results using the same categories for daytime as for night time noise (supplementary table 4) suggested higher relative risks for night time noise. Tests for linear trend across noise categories in the fully adjusted models were significant (P<0.05) for daytime noise and coronary heart disease but not for stroke or cardiovascular disease, nor night time noise.

Sensitivity analyses

Results were materially unchanged with additional confounder adjustment for particulate air pollution and road traffic noise in the 12 London boroughs (data not shown).

Discussion

In this small area study covering a population of 3.6 million people living near Heathrow airport in London, we identified significant excess risks of stroke, coronary heart disease, and cardiovascular disease, especially among the 2% of the population affected by the highest levels of daytime and night time aircraft noise.

Strengths and weaknesses of this study

Strengths of this study include the large general population sample, inclusion of both incident events (hospital admissions) and mortality, and wide range of aircraft noise levels, providing sufficient statistical power to detect modest associations. Common to some other epidemiological studies,11,12 we analysed aircraft noise separately from other transport noise as it is currently unclear whether noise may be additive or whether aspects of noise such as sound frequency and number and duration of noisy events may be important. Limitations include inability to adjust for confounders at individual level. We were able to adjust at small area level for ethnicity, deprivation, and a smoking proxy (and additionally for particulate air pollution and road traffic noise for a subset of 2.6 million people), but we did not have access to individual level information on confounders such as smoking; therefore results at the area level may not be applicable to individuals (ecological fallacy). Admissions for coronary heart disease and to a lesser extent for cardiovascular disease were particularly affected by adjustment for South Asian ethnicity, which itself is strongly associated with risk of coronary heart disease25; hence these risk estimates should be interpreted cautiously. We restricted our hospital admission analyses to the first admission within one calendar year; as we did not link across years it is possible that some may be readmissions if they occurred in different calendar years. However, point estimates at higher noise levels were similar for mortality and hospital admissions, making it less likely that this was an important source of bias.

We examined exposures to aircraft noise in 2001 and health outcomes in 2001-05. We were unable to distinguish between short and longer term effects of noise in the present study and this needs to be examined in further research. Some studies19, 20 have suggested larger effect estimates with longer duration of residence, but this may reflect exposure misclassification among more recent residents. Our data on noise exposure are left censored because of concerns about the accuracy of noise models at low levels. It is difficult to determine the resulting misclassification bias; this may also have affected the size of our risk estimates by restricting the range of noise levels across which effect sizes were estimated. A further potential source of bias is that we did not have information on migration in and out of the study areas.

Possible explanations and implications in the context of previous studies

Potential for causality of the observed associations needs to be considered in the context of previous studies, including consideration of biological plausibility and coherence. Much of the research effort concerning adverse effects of noise on cardiovascular health has focused on effects on blood pressure and risk of hypertension, hypertension being the leading cause of stroke and a major risk factor for heart disease.26 Acute exposure to noise activates the neuroendocrine system, leading to short term increases in heart rate or blood pressure, or both,27, 28 and in stress hormone levels;29 neuroendocrine effects are also seen with chronic exposures30 offering potential mechanisms by which environmental noise may be related to cardiovascular risk. Although these effects have mainly been studied at high exposure levels in the occupational10,30 or experimental setting,31 they may also occur at ambient environmental noise levels.32 In a study conducted near four European airports (including Heathrow), noise disturbance by aircraft noise at night was associated with short term increases in blood pressure of 6-7 mm Hg.33 Increased risks of stroke and coronary heart disease would be expected if such physiological changes were to lead to sustained raised blood pressure.34 A meta-analysis published in 200935 of five studies (totalling nearly 45 000 participants) of aircraft noise and risk of long term hypertension gave a pooled relative risk estimate of 1.13 (95% confidence interval 1.00 to 1.28) per 10 dB increase. A subsequent study of approximately 5000 adults in Sweden found long term effects on hypertension risk only in subgroup analyses, but half the study population had a family history of diabetes, which may affect generalisability.36 The previous literature concerning aircraft noise and cardiovascular disease and mortality is sparse and not fully consistent. In a cross sectional study of people living near seven European airports (including Heathrow), a significant association was observed between night time average aircraft noise and self reported heart disease and stroke (odds ratio 1.25, 95% confidence interval 1.03 to 1.51) in those who had been living in the same place for 20 or more years.37 A census based study of 4.6 million adults aged more than 30 years in Switzerland reported an association with mortality from myocardial infarction in those exposed to the highest level of aircraft noise and who had lived at least 15 years in their place of residence; no associations were seen with stroke or cardiovascular
mortality. A study of adults aged 45-85 years living in Vancouver, Canada did not find associations of aircraft noise with coronary heart disease mortality, neither did a population based study of about 57,000 adults aged 50-64 years in Denmark with stroke mortality. These previous studies had lower population exposures to aircraft noise than in London.

As with our findings for aircraft noise, significant associations have been reported for road traffic noise and heart disease. A meta-analysis of 24 population studies of road traffic noise found a dose-response association with hypertension, with a combined odds ratio of 1.03 (95% confidence interval 1.01 to 1.06) per 5 dB increase of road traffic noise, in the range 45-75 dB. We were unable to distinguish between night time and daytime noise as they were highly correlated and so their effects could not be differentiated. More research is needed to determine if night time noise that disrupts sleep may be a mechanism underlying observed associations.

**Conclusions**

How best to meet commercial aircraft capacity for London and other major cities is a matter of active debate, as this may provide major economic benefits. However, policy decisions need to take account of potential health related concerns, including possible effects of environmental noise on cardiovascular health. Our results suggest that high levels of aircraft noise are associated with an increased risk of stroke, coronary heart disease, and cardiovascular disease. As well as the possibility of causal associations, alternative explanations should be considered. These include the potential for incompletely controlled confounding and ecological bias, as we did not have access to individual level confounder data such as ethnicity and smoking. Further work to understand better the possible health effects of aircraft noise is needed, including studies clarifying the relative importance of night time compared with daytime noise, as this may affect policy response.

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Data sharing: Data are available from the data providers on application with appropriate ethics and governance permissions, but we do not hold data provider, ethics, or governance permissions to share the dataset with third parties.
What is already known on this topic

Few studies have examined aircraft noise and risk of incident or fatal cardiovascular disease or stroke

Previous studies have found an increased risk of hypertension associated with aircraft noise and increased risk of hypertension, stroke, and coronary heart disease with road traffic noise

These findings are consistent with those from studies of occupational noise exposure, and experimental studies examining short term effects of noise on the cardiovascular system

What this study adds

Areas with high levels of aircraft noise related to Heathrow airport in London had increased risks of stroke, coronary heart disease, and cardiovascular disease

Interpretation should consider not only causal associations but also possible alternative explanations such as residual confounding and ecological bias

26 Critchley JB, Ollehead JB. The use of Leq as an aircraft noise index. Civil Aviation Authority, Directorate of Operational Research and Analysis 1990.

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Table

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*ICD-10 codes (international classification of diseases, 10th revision).
Figures

Fig 1 Contextual maps of study area and Heathrow airport showing (top) London boroughs and districts outside London overlaid with the 2001 annual average aircraft daytime (7 am-11 pm, $L_{Aeq,16h}$) noise contours; (bottom) annual average night time noise contours (11 pm-7 am, $L_{night}$).
Fig 2 Relative risks (95% confidence intervals) for associations between hospital admissions for stroke, coronary heart disease, and cardiovascular disease in 2001-05 and annual population weighted average daytime aircraft noise (relative to ≤51 dB) and night time aircraft noise (relative to ≤50 dB) in 2001, census output areas.
Fig 3 Relative risks (95% confidence intervals) for associations between mortality from stroke, coronary heart disease, and cardiovascular disease in 2001-05 and annual population weighted average daytime aircraft noise (relative to ≤51 dB) and night time aircraft noise (relative to ≤50 dB) in 2001, super output areas.