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Acute effects of aircraft noise on cardiovascular admissions – an interrupted time-series analysis of a six-day closure of London Heathrow Airport caused by volcanic ash

ABSTRACT

Acute noise exposure may acutely increase blood pressure but the hypothesis that acute exposure to aircraft noise may trigger cardiovascular events has not been investigated. This study took advantage of a six-day closure of a major airport in April 2010 caused by volcanic ash to examine if there was a decrease in emergency cardiovascular hospital admissions during or immediately after the closure period, using an interrupted daily time-series study design. The population living within the 55 dB(A) noise contour was substantial at 0.7 million. The average daily admission count was 13.9 (SD 4.4). After adjustment for covariates, there was no evidence of a decreased risk of hospital admission from cardiovascular disease during the closure period (relative risk 0.97 (95% CI 0.75 – 1.26)). Using lags of 1-7 days gave similar results. Further studies are needed to investigate if transient aircraft noise exposure can trigger acute cardiovascular events.

1. Introduction

Noise is recognised as being one of the main local environmental problems in Europe and it has been estimated that 55% of the UK population live in dwellings where outdoor environmental noise levels exceed guideline values suggested by the World Health Organisation (Commission of the European Communities, 1996; Skinner and Grimwood, 2005; Berglund et al., 1999). Air transport may be a major source of environmental noise in the vicinity of major airports. Advances in technologies that have produced quieter aircraft are offset by the growth of the air travel industry, which in the UK has been forecast to double by 2030 (Department for Transport, 2009). To meet this level of growth more airports or runways, larger aircraft and an increase in the number of evening and night-time flights is required. All of these measures have the capacity to worsen noise levels around major airports.

There is an increasing body of evidence on the adverse effects of transportation noise on cardiovascular risk (Babisch 2006). Recent studies have found significant associations between aircraft noise and cardiovascular disease risk (Hansell et al., 2013; Correia et al., 2013; Floud et al., 2013). Two of these were small-area level ecological correlation studies which found significant associations between aircraft noise and cardiovascular admissions and mortality (Hansell et al., 2013; Correia et al., 2013). The third used a cross-sectional study design and found an association between exposure to aircraft noise and self-reported heart disease and stroke (Floud et al., 2013). The study designs used largely imply that chronic exposure to aircraft noise increases the risk of cardiovascular disease. Chronic exposure to noise is associated with hypertension, which could explain the underlying causal mechanism (Jarup et al., 2008).

Whilst there is a body of evidence documenting the associations between chronic noise exposure, hypertension and cardiovascular disease as described above, noise exposure also has acute effects on blood pressure, causing transient increases in systolic and diastolic blood pressure observed in laboratory and field conditions (Baudrie et al., 2001; Andren et al., 1980; Isling and Michalak, 2004; Lusk et al., 2004; Haralabidis et al.,2008; Chang et al., 2015). One study which recorded ambulatory blood pressure at 15 minute intervals documented transient increases in blood pressure in normotensive adults specifically in relation to aircraft flight events near airports Haralabidis et al., 2008). In addition, people with pre-existing hypertension have larger acute rises in blood pressure in relation to noise stress (Chang et al., 2015).

Transient rises in blood pressure are recognised as one of the mechanisms by which a range of acute precipitating factors such as bouts of exercise, stressful events and diurnal rises in blood pressure could trigger a cardiovascular event by causing rupture of a vulnerable atheromatous plaque (Bentzon et al., 2014). Therefore, in addition to chronic exposure to aircraft noise being associated with increased risk of cardiovascular disease, it is plausible that aircraft-related noise may also exert acute effects by triggering acute cardiovascular events. However, the potential for acute exposure to aircraft noise to trigger cardiovascular events has not been investigated previously.

We took advantage of a "natural experiment" situation to investigate the hypothesis. We used an unplanned 6-day closure of London Heathrow Airport to investigate whether there was evidence of a reduction in cardiovascular admissions amongst the population living near the airport. The airport is situated close to the centre of London, the European Union's most

populous city. By 2010 rankings, Heathrow was the world's 4th busiest airport by passenger volume and 13th by aircraft movements (Airports Council International, 2014).

Large areas of European airspace were closed as a result of the volcanic ash cloud caused by a major eruption of Iceland's Eyjafjallajökull volcano, which began on 14th April 2010. From 15th April 2010 the National Air Traffic Services imposed restrictions on all UK non-emergency flights. The dynamic nature of the ash cloud resulted in some regional differences in the duration and time at which flight restrictions were applied to individual airports (NATS, 2010). London Heathrow Airport restrictions were in force from 12:00 on Thursday 15th April 2010 to 21:34 on Tuesday 20th April 2010. Scheduled flights resumed on Wednesday 21st April 2010. This 6-day closure of London Heathrow Airport provided a rare opportunity to investigate acute effects of aircraft noise on cardiovascular disease.

2. Materials and methods

We used an interrupted daily time series study design to investigate whether the closure of Heathrow airport was associated with a decrease in acute cardiovascular hospital admissions arising from the population living in the vicinity of the airport. The six-day period in which exposure to aircraft noise was absent was compared with the thirty-day periods immediately before and after the closure period when exposure to aircraft noise would have been at usual levels. The study timeframe therefore comprised a continuous 66-day period from Tuesday 16th March 2010 to Thursday 20th May 2010.

The main study area of interest was the area which fell within the 55 dB(A) noise contour for the airport. This contour was used to select the population exposed to potentially harmful levels of aircraft noise related to the airport. The digitised 2011 noise contour was obtained from the Civil Aviation Authority in a format that was compatible with Geographical Information Systems.

We also used admissions from a "control" area in the analysis. The control area admissions were used as a proxy to adjust for the effects of other unmeasured factors which varied on a daily or short term basis. These factors could have influenced daily variation in admissions and therefore potentially confounded any association between aircraft noise and cardiovascular admissions.

The control area was selected using a 20km buffer constructed around the 55 dB(A) noise contour. The control area excluded populations living close to London's four main commercial airports (Heathrow, Gatwick, Stansted and Luton). The extent of the 55 dB(A) Heathrow airport noise contour and control area, with the location London's main commercial airports and major road networks, is shown in Figure 1.

Routinely collected data on hospital admissions were used to extract emergency cardiovascular admissions with a date of admission within the study timeframe for the analysis. Cardiovascular admissions were defined as admissions with ICD-10 codes I00-I99 in the primary diagnosis field in the first episode of the admission. Admission records contained the UK census-based lower super-output area (LSOA) code as the geographical identifier of area of usual residence. An LSOA typically contains 1500-2000 people. The LSOA population centroid was used to allocate admissions to the 55dB(A) and control areas.

Statistical analysis was carried out using Poisson models. These were used to model the natural log of daily admission counts in the 55dB(A) area as a function of day of the week

(relative to Sunday), the Easter holiday weekend days (2nd to 5th April 2010), airport closure, trend (days) and log of admission counts in the control area. Cochrane-Orcutt methods were used to deal with residual temporal autocorrelation. Models were fitted using combinations of covariates and for lag periods of 0-7 days relative to the airport closure days. Models were assessed for fit and parsimony. The results are presented as regression coefficients with 95% confidence intervals. Exponentiation of these coefficients gives the relative risk (RR) of admission, relative to the baseline for categorical variables and per unit increase for continuous variables.

3. Results

The total population within the 55 dB(A) study area was 724,250 in 433 LSOAs, based on mid-2010 population estimates. The mean (SD) daily count of emergency admissions for cardiovascular disease in this area was 13.9 (4.4). The population in the control area was 8,891,068 in 4604 LSOAs and the mean (SD) admission count in the control area was 189.3 (35.8).

Figure 2 shows daily admission counts over the 66-day study period in the 55 dB(A) and control areas. The control area counts clearly show that admissions tended to be lower at weekends and over the extended weekend period at Easter. A similar pattern is seen in the 55 dB(A) area, although the pattern is less clear as the counts involved are much smaller. Visual inspection of the figure shows no obvious reduction in admissions within the 55 dB(A) study area during the six-day closure period.

Results of the statistical analysis using a lag of 0 days are shown in Table 1. Using lags of 1-7 days gave similar results (not shown). Results for three models are shown. The first is the standard interrupted time series model without inclusion of counts in the control area as a covariate. After adjustment for day of the week, Easter and overall trend, no significant reduction in admissions during the closure period relative to the 30-day periods on either side of the closure period was observed (RR 1.01 (95% CI, 0.78 – 1.31)).

Table 1: Model coefficients for the effects of the six-day closure period in April 2010 and other covariates on (log) daily emergency cardiovascular admissions amongst people living within the 55dB(A) aircraft noise contour of London Heathrow airport.

Model Parameter	Model-1	Model-2	Model-3	Model-4
Intercept	1.923(1.679 to 2.167)	-1.28(-5.059 to 2.5)	-2.633(-4.606 to -0.659)	-2.456(-4.444 to -0.467)
Closure period	0.01(-0.25 to 0.27)	-0.002(-0.261 to 0.256)	-0.019(-0.273 to 0.236)	-0.026(-0.285 to 0.232)
Trend (days)	**0.006(0.002 to 0.01)	*0.005(0.001 to 0.009)	0.004(0 to 0.008)	0.004(0 to 0.008)
Monday	**0.358(0.11 to 0.606)	0.053(-0.381 to 0.486)	-	-
Tuesday	***0.571(0.313 to 0.828)	0.291(-0.124 to 0.707)	-	-
Wednesday	***0.541(0.285 to 0.797)	0.242(-0.192 to 0.675)	-	-
Thursday	***0.587(0.333 to 0.841)	0.277(-0.166 to 0.719)	-	-
Friday	***0.601(0.336 to 0.866)	0.32(-0.101 to 0.742)	-	-
Saturday	**0.42(0.172 to 0.668)	**0.376(0.128 to 0.624)	*0.261(0.043 to 0.478)	*0.247(0.027 to 0.467)
Easter Friday	0.013(-0.553 to 0.578)	0.18(-0.41 to 0.771)	-	-
Easter Saturday	-0.511(-1.083 to 0.062)	*-0.652(-1.24 to -0.064)	*-0.77(-1.339 to -0.201)	**-0.829(-1.402 to -0.257)
Easter Sunday	*0.73(0.157 to 1.302)	*0.603(0.02 to 1.187)	0.445(-0.094 to 0.983)	-
Easter Monday	-0.202(-0.768 to 0.364)	0.024(-0.592 to 0.64)	-	-
"Control" area (log) admission counts	-	0.662(-0.118 to 1.442)	***0.965(0.586 to 1.343)	***0.934(0.552 to 1.316)
Residual SE (df)	0.270 (52)	0.266 (51)	0.270 (58)	0.274 (59)
R-square (adjusted)	0.985	0.986	0.987	0.985

Significance Level: * p<0.05; ** p<0.01; *** p<0.001

Model coefficients are from interrupted daily time series Poisson regression models (six-day closure period in April 2010 compared with 30-day periods immediately before and after the closure period). The coefficients provided indicate the covariates included in each model. The Easter days were not in the closure period.

The second model included admission counts in the control area as a proxy to adjust for other factors potentially contributing to daily variation in admissions. Again, there was no evidence of a significant reduction in admissions during the closure period relative to the 30-day periods on either side of the closure period (RR 1.00 (95% CI, 0.77 – 1.29)). This second model also shows that inclusion of admission counts in the control area largely captured day of the week effects (hence most of the day of the week effects and the control day admissions effect are non-significant).

The third model is a more parsimonious model which only includes significant day of the week variables (Saturday and Easter Saturday) in addition to control day admissions and overall trend. Again, it can be seen that there was no significant reduction in admissions in the closure period relative to the 30-day periods on either side (RR 0.97 (95% CI, 0.75 – 1.26)).

4. Discussion

We investigated the hypothesis that aircraft noise might trigger acute cardiovascular events by taking advantage of a six-day closure of London Heathrow airport caused by volcanic ash in the atmosphere over northern Europe to investigate the hypothesis. However, we found no significant evidence of a reduction in emergency cardiovascular admissions during the six-day closure period compared with admissions in 30-day periods immediately before and after

the closure period (RR 0.97 (95% CI, 0.75 – 1.26). Using lags of 1-7 days to investigate delayed effects gave similar results.

A potential explanation as to why we did not find any association is that there was insufficient power to detect an effect. However, closures of major airports situated within major city boundaries are rare events. The population living within the 55 dB(A) noise contour of Heathrow was substantial, with 0.7 million residents normally exposed to aircraft noise over this level. Whilst US airports were closed for a period following the 9/11 terrorist attacks in 2001, we have not found any peer-reviewed publications examining if closure was associated with decreases in cardiovascular events. In any case, the traumatic events may have had adverse psychological and clinical effects resulting in transient increases in cardiovascular events which would have masked any underlying association.

An alternative explanation is that aircraft related noise does not trigger acute cardiovascular events. However, transient increases in blood pressure have been clearly documented in several studies including studies where exposure was carried out under controlled laboratory conditions and studies using ambulatory blood pressure monitoring to assess work related and environmental noise (Baudrie et al., 2001; Andren et al., 1980; Isling and Michalak, 2004; Lusk et al., 2004; Haralabidis et al.,2008; Chang et al., 2015). Haralabidis et al found that transient aircraft events were associated with acute rises in systolic and diastolic blood pressure of 6.2 mmHg and 7.4 mmHg respectively, with blood pressure measured at 15 minute intervals using non-invasive ambulatory monitoring (Haralabidis et al.,2008). Furthermore, Chang et al have found that acute rises in blood pressure in response to noise are more marked in people with underlying hypertension (Chang et al., 2015). They reported a transient increase of 0.3 mmHg in systolic blood pressure per unit increase in dB(A). This would be equivalent to a rise in systolic blood pressure of 16.5 mmHg for a 55 dB(A) increase in noise levels. It is recognised that a range of transient factors (e.g. sudden emotional stress) can trigger acute cardiovascular events and the triggering pathways include activation of the sympathetic nervous system with increased heart rate and blood pressure leading to plaque rupture (Bentzon et al., 2014).

Current evidence linking aircraft noise to cardiovascular disease is generally interpreted as evidence of a chronic exposure effect (Babisch, 2006; Hansell et al., 2013; Correia et al., 2013; Floud et al., 2013; Jarup et al., 2008). Hansell et al found that people living near Heathrow airport and exposed to high levels of aircraft noise had increased rates of cardiovascular admissions and mortality (Hansell et al., 2013). In the USA, Correia et al found higher cardiovascular hospital admission rates amongst the Medicare registered population aged 65 or more years experiencing higher levels of residential exposure to aircraft noise (Correia et al., 2013). Whilst studies that examine prevalence of conditions such as hypertension are consistent with this chronic exposure hypothesis, ecological and cohort studies examining cardiovascular event rates cannot distinguish between a chronic exposure effect of noise (e.g. noise exposure causing acceleration of atherosclerotic disease resulting in higher rates of cardiovascular events) and acute recurrent aircraft noise events triggering cardiovascular events, which would also result in higher rates in more noise exposed areas or cohorts. Thus, cohort and ecological studies would capture both acute and chronic exposure effects of noise on cardiovascular events and would not be able to distinguish between them, a situation similar to that observed in ecological studies examining the link between air pollution and cardiovascular disease (Maheswaran et al., 2012; 2014).

There are a number of limitations to our study which need to be considered. We have already mentioned the limited power of the study to detect any association between aircraft noise and

acute cardiovascular events. There may have been errors in coding of emergency admissions. Acute cardiovascular events resulting in death before admission to hospital will not have been captured by hospital admissions data. Unmeasured confounders that contributed to daily variation in admissions may have masked any underlying association. However, we used admissions in the control area to attempt to adjust for such factors. For example, if the ash cloud had any effect on cardiovascular events, this effect would also have been observed in the control area, and adjusted for using this approach in the analysis. The daily time series design essentially means that only factors which vary on a short term (e.g. daily) basis would be able to confound any association between noise and acute health effects, and factors which vary on a longer term basis e.g. smoking prevalence or socioeconomic status, would not act as confounders in this situation. We decided apriori to use the 55 dB(A) noise contour to define the study population. An alternative approach would have been to use more contours. However, we felt that this approach would have further reduced the power of the study.

Although we found no evidence of an acute effect of aircraft noise on cardiovascular events, this hypothesis cannot be ruled out. As pressure for airport expansion increases, a greater understanding of the health risks associated with living close to an airport is needed and further studies are indicated. The ash cloud affected large parts of the northern European airspace and a multinational study would increase study power. In addition, time series studies carried out using a fine temporal resolution would be needed to detect an acute effect. This will require aircraft noise around airports to be continuously monitored and the precise onset times of cardiovascular events in populations living near airports to be recorded.

In summary, we found no evidence of a significant reduction in emergency hospital admissions for cardiovascular disease amongst an exposed population of approximately 0.7 million people during a six-day period in which London Heathrow, a major European airport, was closed. Further studies are needed to investigate if transient aircraft noise exposure can trigger acute cardiovascular events.

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Figure 1: Map showing the extent of the 55 dB(A) noise contour study area around London Heathrow Airport and the extent of the "control" area. Inset map shows the location of the study area within the UK.
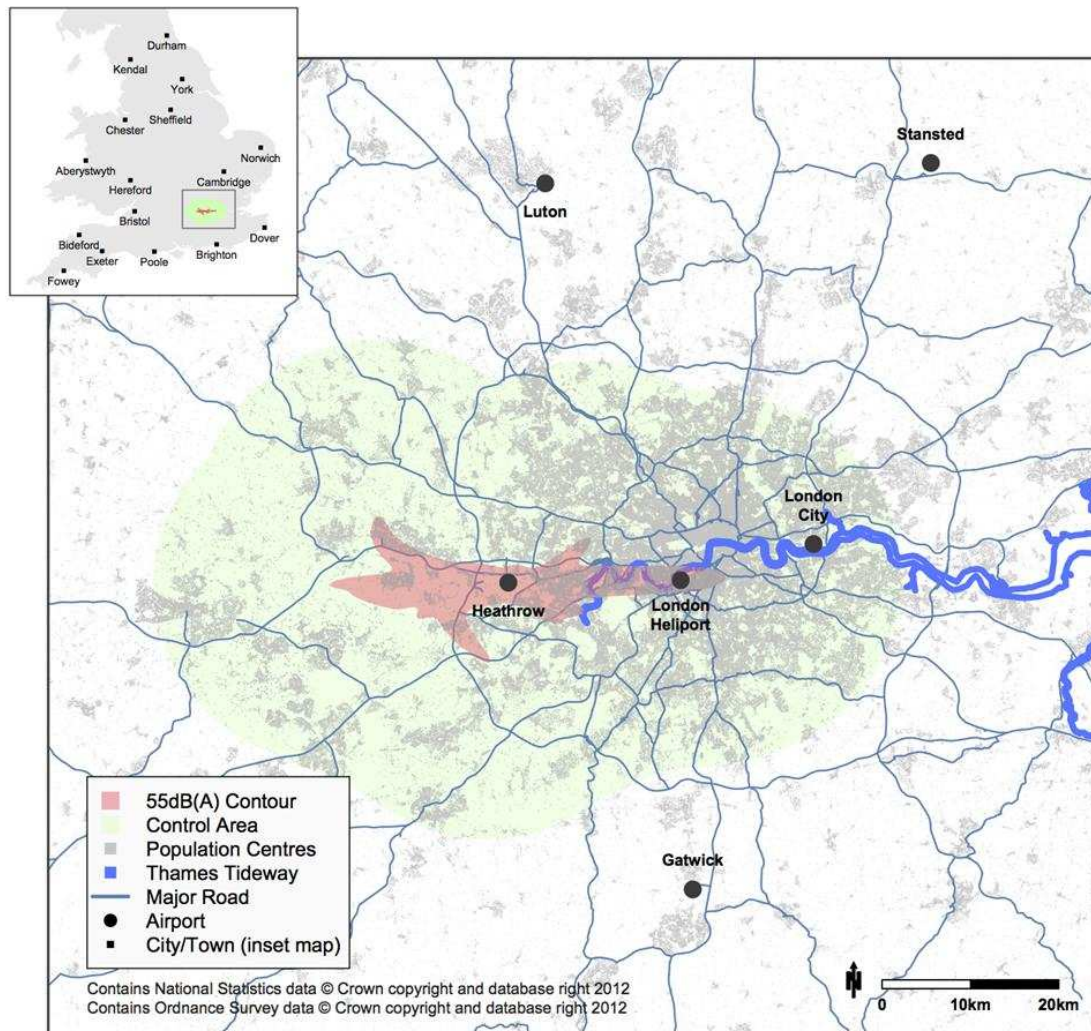
Figure 2: Daily counts of cardiovascular emergency hospital admissions from the 55dB(A) aircraft noise contour study area for London Heathrow Airport and from the "control" area over the 66-day study time-frame, March-May 2010 (admission counts are shown on a log scale).