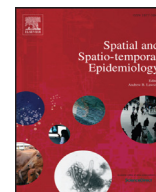


Contents lists available at [ScienceDirect](#)

Spatial and Spatio-temporal Epidemiology

journal homepage: www.elsevier.com/locate/sste

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

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ARTICLE INFO

Article history:

Received 29 November 2015

Revised 17 March 2016

Accepted 21 March 2016

Available online xxx

Keywords:

Aircraft

Noise

Cardiovascular disease

Hospital admissions

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 co-variables, 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.

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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 dis-

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ease. 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 min 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 30-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 20 km 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 Fig. 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 55 dB(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 55 dB(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

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