



Full length article

Long-term exposure to aircraft noise and cardiovascular disease hospitalization and mortality near major airports in the UK, 2006–2015 – A small area study

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ABSTRACT

The environmental disease burden from transport noise in Europe is considered second only to air pollution, but the majority of epidemiological studies relate to road noise. We examined associations between annual average day-evening-night (Lden) and night-time (Lnight) aircraft noise in 2006 and 2011 and cardiovascular disease (CVD) hospitalization and mortality. We used a small area design covering a population of 3.1 million living near London Heathrow, Gatwick, Birmingham and Manchester international airports in 2006–2015. Statistical analysis used Bayesian Poisson regression in linear and categorical analyses. We observed strong evidence of associations between aircraft noise and hospital admissions; for coronary heart disease admissions near London Heathrow, we found an increased risk of 0.44 % (95 % CrI 0.16 %, 0.73 %) and for all-CVD admissions near other airports an increased risk of 0.34 % (95 % CrI 0.04 %, 0.64 %) per 5 dB Lden for noise levels above 50 dB Lden (the cut-off level for available data). However, results were not fully consistent across airports and no associations were seen with stroke hospitalisation and mortality, nor with CVD or CHD mortality. Associations were smaller and less clear than our previous Heathrow study of similar design during 2001–5. Differences over time are likely to relate to changes in population, therefore population confounder structure, over time, as well as reductions in population aircraft noise exposure. Given the increasing evidence base showing associations between noise and cardiovascular disease, we recommend use of large cohorts with better control of confounding at individual-level to provide quantification of exposure–response relationships.

1. Introduction

The World Health Organization classified the burden of disease from

transport noise as the second highest environmental risk in Europe after air pollution (World Health Organization. [Environmental noise guidelines for the European region.](#), 2018). There is good epidemiological

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(Kempen et al., 2018) and experimental (Münzel et al., 2021; Schmidt et al., 2013; Schmidt et al., 2021) evidence on adverse effects of noise on the cardiovascular system. Cardiovascular outcomes have been included in recent European and national burden of disease calculations for transport noise by the European Environment Agency (Agency, 2020) and by the UK Health Security Agency (Jephcote et al., 2023). However,

- Lden: annual-average, 24-hour noise indicator with separate weightings for the daytime period of 07:00–19:00 (Lday + 0 dB), evening period of 19:00–23:00 (Leve + 5 dB), and night-time period of 23:00–07:00 (Lnight + 10 dBA).

$$L_{den} = 10 \cdot \log_{10} \left(\frac{\left[12 \cdot 10^{\left(\frac{L_{day}}{10} \right)} \right] + \left[4 \cdot 10^{\left(\frac{L_{eve}+5}{10} \right)} \right] + \left[8 \cdot 10^{\left(\frac{L_{night}+10}{10} \right)} \right]}{24} \right)$$

much of the evidence to date relates to road transport with fewer studies on aircraft noise. The systematic review underpinning the WHO 2018 Environmental Noise Guidelines for the European Union rated the quality of evidence supporting an association between aircraft noise and coronary heart disease (CHD) as ‘Low’ using modified GRADE criteria (Kempen et al., 2018); indicating that further research is warranted. More recent *meta*-analyses have found small positive associations of aircraft noise exposure with cardiovascular disease (CVD) not all of which are statistically significant (Vienneau, 2019; Cai et al., 2021), but the evidence base remains limited due to the small number of studies.

A disproportionate number of people are affected by aircraft noise in England in comparison to other European countries (Civil Aviation Authority, 2014), with airports such as London Heathrow, one of the ten busiest airports worldwide, sited close to densely populated urban areas. In a previous small area study published in 2013, we found positive associations between aircraft noise from London Heathrow airport 2001–5 and CVD mortality and morbidity in 12,110 census output areas (COAs), home to 3.6 million inhabitants (Hansell et al., 2013).

Here, we extend the previous small area analysis to consider aircraft noise exposure for the years 2006–15 near London Heathrow and additionally examine the effect of aircraft noise exposure near three other major international airports in England – London Gatwick, Manchester, and Birmingham.

2. Methods

We conducted a small area study, comprising all COAs around London Heathrow, London Gatwick, Manchester, and Birmingham airports that were partly or wholly within the 2006 and 2011 50 dB Civil Aviation Authority (CAA) aircraft noise contours of Lden aircraft noise. Area boundaries used were those of the 2011 national Census. A-weighting is a frequency weighting applied to sound levels to reflect the loudness perceived by the human ear.

2.1. Aircraft noise data

The UK Civil Aviation Authority (CAA) provided 100 m x 100 m annual-average estimates of ground level aircraft noise around London Heathrow, London Gatwick, Manchester, and Birmingham airport for 2006 and 2011. The night and day-evening-night noise metrics used in this analysis are quantified in decibels that are A-weighted (dB(A); referred to as dB hereafter) to the audible frequencies of the human ear to noise between 500 Hz and 6 kHz:

- Lnight: Night-time annual-average noise indicator for the 8-hour period of 23:00 to 07:00.

These were modelled using the UK Civil Aircraft Noise Contour Model ANCON, which includes information on flight paths of arriving and departing aircraft and factors such as height, speed, and engine power to derive noise at ground level (Ollerhead et al., 1999). CAA datasets are provided truncated to a lower noise limit of 45 dB for Lnight and 50 dB for Lden. We used six exposure categories for Lden – ≤50 dB (reference), >50–|54 dB, >54–|57 dB, >57–|60 dB, >60–|63 dB and > 63 dB and four categories for Lnight – ≤45 dB (reference), >45–|50 dB, >50–|55 dB, and > 55 dB.

Values from the 100 m x 100 m aircraft noise surfaces were extracted at the central delivery points of postcode locations (centroids) and attributed with residential population headcounts in the 2011 UK Census. A typical postcode unit in England contains 43 residents (SD = 39) within 18 occupied households (SD = 15). Population-weighted noise exposures were calculated for COA communities by taking the logarithmic average of the postcode points located within. COAs in England have residential populations of between 100 and 625 persons, typically 309 residents (SD = 83), within an area that covers 0.78 km² (SD = 3.27 km²). Therefore, approximately 7 postcodes are usually located within each COA.

2.2. Health data

Hospital admissions data were obtained from the national Hospital Episode Statistics (HES) dataset held by the UK Small Area Health Statistics Unit (SAHSU), provided by National Health Service (NHS) Digital. Only primary admissions recorded as the first episode of stay each year were included. Mortality data were obtained from the Office for National Statistics (ONS) dataset held by SAHSU. We extracted data on cardiovascular hospital admissions and mortality in 2006–10 and in 2011–2015 for individuals whose residential postcode at time of event fell within the study area, using the 2006 and 2011 noise contours as described above. Disease categories selected were: all cardiovascular disease (CVD) (International Classification of Disease 10th edition (ICD-10) Chapter I), CHD (ICD-10:I20–I25) and Stroke (ICD-10 codes: I61, I63–I66).

2.3. Data on potential confounders

Population stratification by age group in 5-year intervals (0–4, 5–9, 10–14, 15–19, 20–24, 25–29, 30–34, 35–39, 40–44, 45–49, 50–54, 55–59, 60–64, 65–69, 70–74, 75–79, 80–84 and 85 + years) and sex (male and female) comprising 36 strata were included in the expected counts of health outcomes (see details in the Statistical Analysis subsection). We adjusted for ethnicity, deprivation, road and rail-transport noise and long-term ambient nitrogen dioxide (NO₂) exposure. We

obtained 2011 Census data on area-level ethnicity and deprivation from Nomis, the official online delivery service provided by ONS. Fifthths of the Carstairs index (Morris and Carstairs, 1991) were used as an area-level deprivation indicator. We modelled annual-average road-transport noise exposure metrics at postcode centroid locations for 2013 (i.e., the nearest year available to correspond with data on aircraft noise) in accordance with the European Commission ‘Common Framework for Noise Assessment Methods’ (CNOSSOS-EU) (Morley et al., 2015). The model considered contributions from annual-average daily traffic counts on major roads within 1 km and minor roads within 100 m (i.e., to reflect the influence of local road traffic in residential areas), along with information relating to the surface roughness of land cover, building geometries and heights, wind profiles, and average temperatures. The UK Department for Environment, Food and Rural Affairs (Defra) provided 10 m x 10 m annual-average noise exposure surfaces originating from major railway lines in 2011, defined as railways running more than 30,000 passenger vehicle trips per year. Annual average NO₂ concentrations in µg/m³ at 200x200m resolution from all sources of outdoor air pollution were extracted from a 2009 Land Use Regression (LUR) surface of the UK (Gulliver et al., 2013). For more information about the confounders see Online Supplement Text S1.

2.4. Statistical analyses

We specified a Bayesian hierarchical Poisson model with spatial random effects. This framework provides a powerful regression technique to address issues of data sparsity, borrowing strength across airport locations and small areas and accounting for unknown spatial confounding.

Separately for each outcome (CVD, CHD, and stroke hospitalizations and deaths, $j = 1, \dots, 6$), we estimated the expected number of cases E_{ijt} in COA i for each year $t(1, \dots, 10)$ by age and sex classes $k(k = 1, \dots, 36)$. The expected number of hospital admissions/deaths is the denominator for the morbidity/mortality ratio of each health outcome and is calculated for each COA adjusting for age-sex and year, using an internal standardization procedure, and considering the entire study region as reference population as follows:

$$E_{ijt} = \sum_{k=1}^K \lambda_{jkt} N_{ikt} \quad i = 1, \dots, n(\text{COA}) \quad (1)$$

$j = 1, \dots, 6$ (outcomes)

$k = 1, \dots, 36$ (age – sex strata)

$t = 1, \dots, 10$ (2006, ..., 2015), where λ_{jkt} is the rate over the entire study region and N_{ikt} is the population in each COA-year-age-sex stratum. Then E_{ijt} are summed over the two periods p (where $p = 1$ covers 2006–10, and $p = 2$ covers 2011–15):

$$E_{ij1} = \sum_{t=1}^5 E_{ijt} \text{ and } E_{ij2} = \sum_{t=6}^{10} E_{ijt} \quad (2)$$

Then, separately for Heathrow and for the other airports, we assumed that the number of events (hospitalizations or deaths) in the i th COA during exposure period p follows a Poisson distribution:

$$O_{ijp} \sim \text{Poisson}(\lambda_{ijp} E_{ijp}) \quad (3)$$

The parameter λ_{ijp} represents the relative risk of CVD, CHD, or stroke hospitalization or mortality. To simplify the notation, we omit j from the model, but we use the same model for all 6 outcomes:

$$\log(\lambda_{ip}) = \alpha_0 + X_{ip}\beta + Z_i\gamma + b_i \quad (4)$$

where α_0 are intercept terms that capture the baseline risk of hospital admission and deaths for each outcome event. For the other airports analysis we allowed the intercept to vary by airport α_{0s} ($s = 1, \dots, 3$). The

terms β denote the vector of regression coefficients for the different categories of the weighted annual average of day-evening-night time (Lden) or night time aircraft noise denoted as X_{ip} . Note that X_{i1} corresponds to the 2006 exposure metrics, while X_{i2} corresponds to the 2011 exposure metrics. We also examined the linear effect of aircraft noise on health for noise levels above 50 dB for Lden and 45db for Lnight (aircraft exposure data are not supplied by CAA below these levels as noise estimates are considered too uncertain). The effects of the two main noise exposures were assumed not to change between the two periods, which was supported by testing for the suitability of a different effect between the two periods. The term γ is a vector of fixed effect regression coefficients for the set of multiple confounders, denoted as Z_i . The term b_i accounts for spatial variability and is defined as a reparametrisation of the Besag-York-Mollie model (Besag et al., 1991; Riebler et al., 2016; Konstantinoudis et al., 2020). For more information about this spatial prior see Online Supplement Text S2.

Fitting the model to Heathrow airport separately (from the other three airports considered in this study) allowed a comparison with previous Heathrow-based analyses (Hansell et al., 2013), and with airports in other parts of the UK which have a different confounder mix to Heathrow.

All statistical regression analyses were implemented using the Integrated Nested Laplace Approximation within the R-INLA software environment (Rue et al., 2009; Blangiardo et al., 2013) in R Statistical Software version 4.2.1 (R Core Team R. R., 2013). We present the minimally and fully adjusted effect of noise on mortality and hospital admissions. We note that the term minimally adjusted here refers to the lack of additional covariates in the regression model, while the effects are still indirectly adjusted for age, sex and time, through the calculation of the expected values in (1)–(2). When presenting the results we label “no evidence” of an effect if the regression coefficient is negative or positive, but close to 0 with the credible interval spanning across negative and positive values, while with “weak evidence” of an effect we refer to small but positive regression coefficients, with a credible interval just crossing 0 on the negative side. Finally, we label “strong evidence” of an effect if the regression coefficient is positive, and the credible interval does not cross 0.

2.5. Sensitivity analysis

We ran a series of sensitivity analyses to examine robustness of the results. First, we stratified by periods 2006–10 and 2011–15 to assess if associations near London Heathrow had changed over time, using the fully adjusted model. Second, to examine whether the random effect specification was supported by the data, we considered alternative models, either not accounting for the correlation among neighbouring areas or removing the random effect completely. We compared the Deviance Information Criterion (DIC), the Watanabe-Akaike Information Criterion (WAIC) and assessed the posterior distribution of the hyperparameters. We examined for potential that the effect of aircraft noise is modified by age, by focusing on populations older than 55 years. Lastly, to ensure comparability with the previous study focusing on Heathrow (Hansell et al., 2013); we repeated the analysis using the same noise categories as previously.

3. Results

3.1. Study area and population

The study area comprised 8,147 COAs near London Heathrow, and 1,713 around London Gatwick, Manchester, and Birmingham airports (Online Supplement Table S1). The population in the study area around all four airports at the 2011 Census was 3,138,937, of whom 2,619,234 (83.4 %) lived near London Heathrow (Online Supplement Table S2). Table 1 shows the totals, mean and standard deviation of the different health outcomes per COA near London Heathrow and the other airports.

Table 1

Total number of cases, mean and standard deviation (SD) across 8,147 COAs near London Heathrow, and 1,713 COAs near other (London Gatwick, Manchester, and Birmingham) airports during 2006–2010, 2011–2015 and 2006–2015. Statistical Disclosure Control procedure was applied to subnational estimates: * = < 5 counts per cell; all counts were rounded to the nearest 5 or 0.

Hospital Admissions									
	All cardiovascular disease (ICD-10 Chapter 1) ^a			Coronary heart disease (ICD-10 120–125) ^a			Stroke (ICD-10 161, 163, 164) ^a		
	N	Mean	SD	N	Mean	SD	N	Mean	SD
Heathrow									
2006–2010	137,410	16.9	9.5	41,355	*	*	10,750	*	*
2011–2015	144,570	17.7	9.8	36,970	*	*	12,235	*	*
2006–2015	281,980	17.3	9.7	78,325	*	*	22,980	*	*
Other airports									
2006–2010	39,690	23.2	10.1	11,450	*	*	3,480	*	*
2011–2015	40,820	23.8	10.3	11,195	*	*	3,435	*	*
2006–2015	80,510	23.6	10.2	22,645	*	*	6,920	*	*
Mortality									
Heathrow									
2006–2010	24,590	*	*	10,915	*	*	4,520	*	*
2011–2015	20,605	*	*	9,085	*	*	3,840	*	*
2006–2015	45,200	*	*	20,000	*	*	8,365	*	*
Other airports									
2006–2010	8,840	*	*	4,290	*	*	1,485	*	*
2011–2015	7,050	*	*	3,470	*	*	1,285	*	*
2006–2015	15,890	*	*	7,760	*	*	2,770	*	*

^a ICD-10 codes (international classification of diseases, 10th revision).

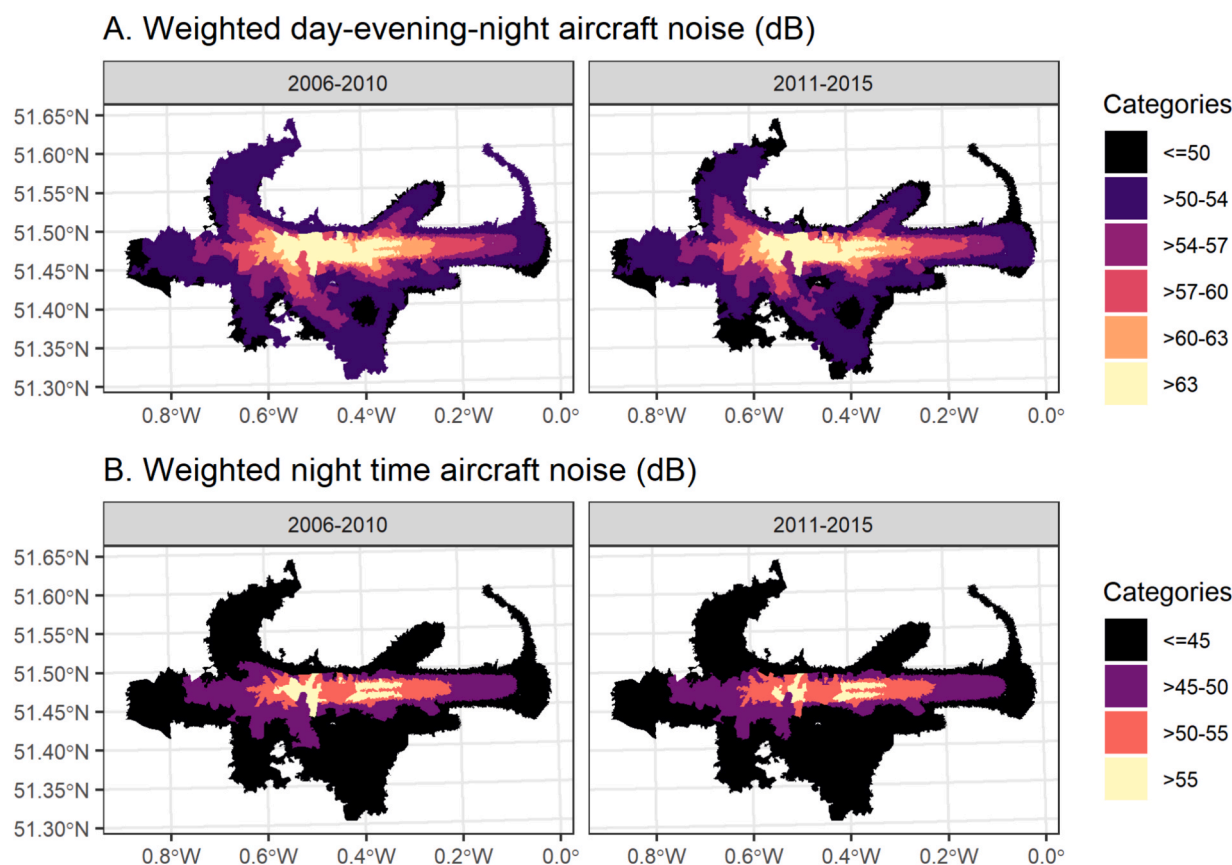


Fig. 1. The spatial distribution of the weighted day-evening-night (panel A) and the weighted nighttime aircraft noise (panel B) in dB near London Heathrow in 2006 and 2011.

In 2006–2015 there were 281,980 hospital admissions for CVD, 78,325 for CHD and 22,980 for stroke near Heathrow airport, and respective figures for mortality were 45,200 CVD, 20,000 CHD and 8,365 S (Table 1). Comparing 2006–10 with 2010–15, hospital admissions for each outcome either increased or stayed at similar levels, whereas mortality decreased.

3.2. Aircraft noise exposure

Online Supplement Fig. 1, S1-5, 7, 9 and 11 summarise and map the spatial extent of the aircraft noise contours in 2006 and 2011. For Lden, the 2006 and 2011 population-weighted exposures differed by less than 1 dB in > 70 % of the COAs around Birmingham, Gatwick, and Heathrow

airport – with > 90 % of these COA reporting differences of 1.5 dB or lower (see [Supplementary Text S3](#)). Changes in exposure were less pronounced for Lnight than Lden. For Lden, >50 % of COAs have seen at least a 1 dB reduction in Lden noise levels around Manchester airport and the biggest change in categorised noise levels categories was at Heathrow with a shift of 10.6 % drop in population exposed to > 50–54 dB and 13.3 % rise in population in the ≤50 dB category comparing 2011 with 2006 ([Table S17](#)). Only small changes were seen in higher noise categories.

3.3. Confounder distribution

The distribution of confounders (Carstairs deprivation index, ethnicity, railways noise exposure, road transport noise exposure and NO₂ exposure) differed by airport (Online [Supplement Table S1](#) and [Figs. S6, 8, 10 and 12](#)). We observed highest concentrations of Black and South Asian ethnicity populations in the northeast and northwest of London Heathrow and Birmingham airports respectively, corresponding to the locations of the most deprived areas and highest NO₂

concentrations, but mostly not the areas with the highest aircraft noise ([Fig. 1](#) and [Supplement Figs. S6–12](#)). There was a strong correlation ($r > 0.8$) between Lden and Lnight for all airports (Online [Supplement Figs. S13–14](#)). Correlations between aircraft noise metrics and the potential confounders (deprivation, ethnicity, road traffic noise, rail noise and NO₂) were generally low to moderate (Online [Supplement Figs. S13–14](#)) with highest correlations seen with South Asian ethnicity for London Heathrow (0.6 with Lden and 0.5 with Lnight in 2006 and 0.6 with both Lden and Lnight in 2011).

3.4. Heathrow

In the fully adjusted models for London Heathrow, we found no evidence of positive associations of all CVD hospital admissions with Lden ([Fig. 2](#), [Table 2](#) and Online [Supplement Table S3](#)). However, for CHD, apparent increased hospital admission risks of 4.56–8.35 % with increasing exposure were observed in categorical analyses ([Fig. 2](#), Online [Supplement Table S3](#)), corresponding to an increase of 0.44 % (95 % CrI 0.16 %, 0.73 %) risk for every 5 dB increase in Lden in linear models

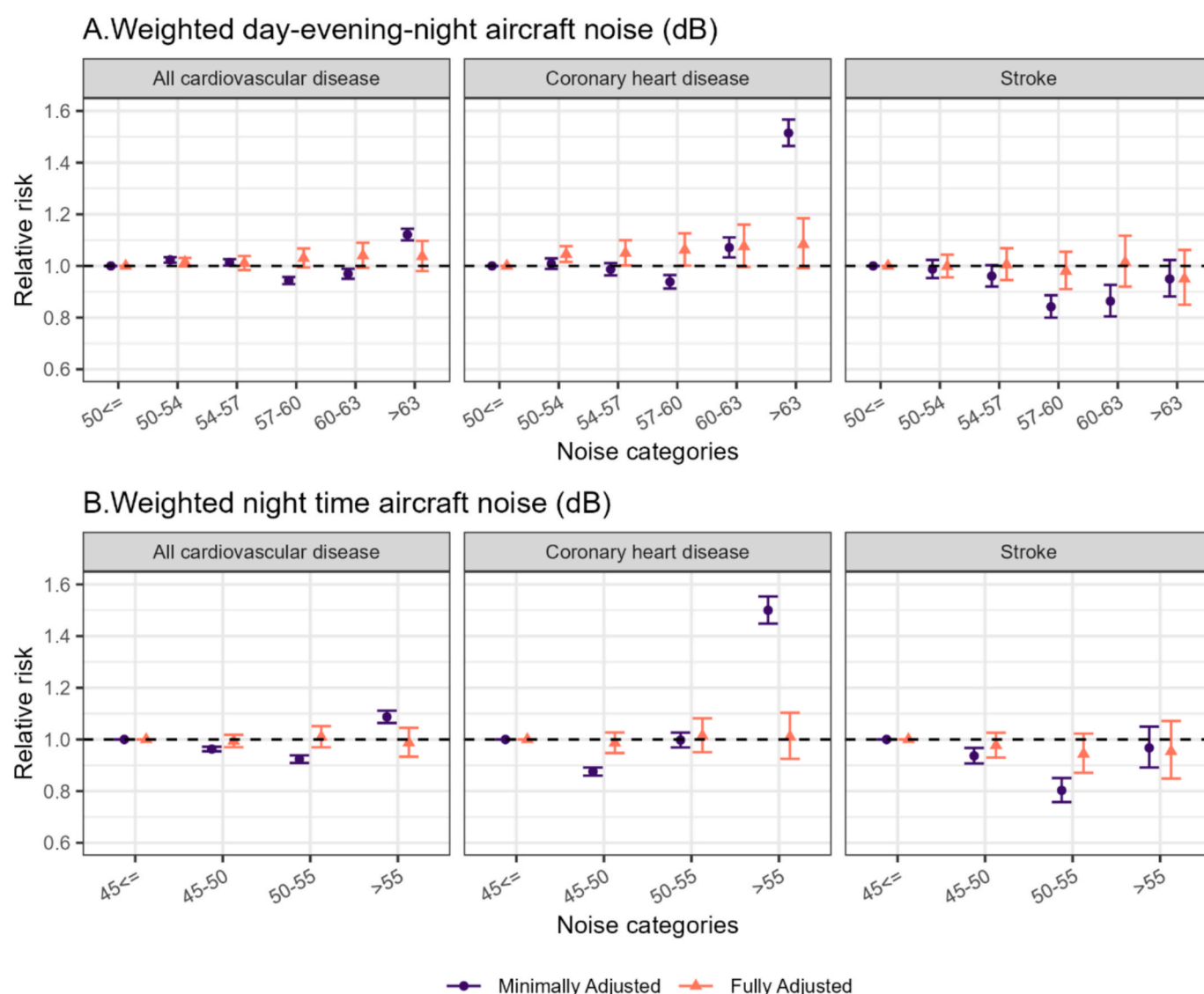


Fig. 2. Median relative hospitalisation risk and 95% credible intervals for all cardiovascular disease, coronary heart disease and stroke using the minimally and fully adjusted models in London Heathrow during 2006–2016. Panel A. shows associations with long-term exposure to annual average population weighted day-evening-night aircraft noise. Panel B. shows associations with long-term exposure to annual average population weighted night time aircraft noise. The minimally adjusted model does not include additional covariates but accounts for temporal trends, age and sex through the expected counts (see (1)–(2) in the Methods section), whereas the fully adjusted model additionally accounts for ethnicity, deprivation, road noise, rail noise, NO₂ exposure and spatial confounding.

Table 2

Median and 95 % Credible Intervals (in parenthesis) of the posterior distribution of the linear effect of aircraft noise (Lden and Lnight) on all cardiovascular disease, coronary heart disease and stroke hospital admissions and mortality near London Heathrow and other airports (London Gatwick, Manchester and Birmingham). The results are from the fully adjusted models accounting for temporal trends, age, sex, ethnicity, deprivation, road noise, rail noise, NO₂ exposure and spatial confounding. As aircraft exposure data are not supplied below 50 dB for Lden and 45db for Lnight, the linear effect refers to relative risks for noise levels higher than these thresholds.

	Lden ^a		Lnight ^b	
	London Heathrow	Other airports	London Heathrow	Other airports
Hospital admissions				
All	1.00142	1.00335	0.99955	1.00254
CVD ^c	(0.99983, 1.00301)	(1.00035, 1.00635)	(0.99703, 1.00206)	(1.00022, 1.00488)
CHD ^d	1.00443 (1.00155, 1.00731)	0.99724 (0.99184, 1.00267)	0.99926 (0.99517, 1.00336)	1.00119 (0.99722, 1.00519)
Stroke	0.99966 (0.99552, 1.00381)	1.00717 (0.99825, 1.01616)	0.99696 (0.99224, 1.0017)	1.00404 (0.99797, 1.01015)
Mortality				
All	1.00065	1.00053	1.00256	1.00111
CVD ^c	(0.99728, 1.00404)	(0.99385, 1.00726)	(0.99860, 1.00654)	(0.99623, 1.00601)
CHD ^d	1.00021 (0.99556, 1.00487)	0.99732 (0.98869, 1.00603)	1.00470 (0.99938, 1.01005)	1.00077 (0.99488, 1.00670)
Stroke	1.00130 (0.99471, 1.00793)	1.00161 (0.98693, 1.01652)	0.99560 (0.98902, 1.00222)	0.99921 (0.98928, 1.00924)

^a Annual average population weighted day/evening/night time aircraft noise,

^b Annual average population weighted night-time aircraft noise,

^c All cardiovascular disease,

^d Coronary heart disease.

(Table 2 and Online Supplement Table S4 and Fig. S15) for noise levels above 50 dB. We observed a large attenuation of the effect of noise levels on CHD hospital admissions once we accounted for the selected confounders. No associations were seen for stroke after adjustment. There was no evidence of associations with Lnight after adjustment (Table 2 and Online Supplement Table S5 and Fig. S15).

There was no evidence of associations of Lden with mortality for any outcome (Fig. 3) in either categorical (Online Supplement Table S6) or linear (Table 2 and Online Supplement Table S4) analyses. For CVD mortality and Lnight there was weak evidence of association, but we did detect a positive increased risk of 0.25 % (95 % CrI −0.14 %, 0.65 %) for every 5 dB increase in linear analyses for noise levels above 45 dB (Table 2 and Online Supplement Table S4), similarly reported in all noise strata (Online Supplement Table S7). For CHD mortality and Lnight, strong evidence of an association was seen in categorical analyses for 45–<50 dB only, while linear analyses showed weak evidence of a small increased risk of 0.47 % for every 5 dB increase above 45 dB (95 % CrI −0.062 %, 1.01 %) (Table 2 and Online Supplement Table S4). There were no associations with stroke mortality.

3.5. Other airports

In the other airports (Fig. 4, Online Supplement Table S8–S12 and Fig. S16), a complex pattern of associations emerged. Categorical analyses for Lden suggested increased risks for all CVD admissions in categories from 50–60 dB Lden (vs. <=50 dB), with increased risk of 6.44 % (95 % CrI 1.14 %, 11.70 %) for 57–60 dB in fully adjusted models, but not at higher noise levels. In linear analyses strong evidence of an increased risk of 0.33 % (95 % CrI 0.03 %, 0.64 %) per 5 dB Lden was observed for all CVD (Table 2 and Online Supplement Table S9) for noise levels above 50 dB. For CHD admissions, there were no associations in

either categorical or linear analyses (Fig. 4, Table 2 and Online Supplement Table S8–S9). There was no evidence of a positive association between categorical and continuous Lden noise exposure and stroke (Table 2 and Online Supplement Table S8 and Fig. S16).

For Lnight at other airports, categorical analyses (Fig. 2 and Online Supplement Table S10) showed evidence of associations for all cardiovascular diseases and for stroke at middle categories of noise (50–55db); this was also seen in the corresponding adjusted linear analyses for noise as a continuous exposure for all CVD only (0.25 % with 95 % CrI 0.02 %, 0.49 %) (Online Supplement Table S9 and Fig. S16). There was little evidence of an association in categorical or linear analysis for CHD (Online Supplement Table S9–S10 and Fig. S16).

For mortality around other airports, there was no evidence of associations between Lden or Lnight and any mortality outcome in categorical (Fig. 5, Online Supplement Table S11–S12) or linear (Online Supplement Table S9) analyses.

3.6. Sensitivity analysis

In a sensitivity analysis, we examined whether effect estimates varied between 2006–10 and 2011–15 given shrinkage in noise contours over time near London Heathrow, but found similar results (Online Supplement Figs. S17–18).

We also examined how the results change with spatial adjustment: without accounting for space, and accounting for space using a prior without a spatial structure and confirmed that the model with the BYM2 prior was the best performing (Online Supplement Tables S13–15).

We found little evidence of an effect modification by age, when focusing on populations older than 55 years (Online Supplement Figs. S19–22, nearly identical to all-age findings as shown in Figs. 2–5). We also examined how the results change when adjusting for each variable separately, finding that ethnicity, deprivation and properly accounting for spatial autocorrelation resulted in the largest changes in observed effect estimates (Online Supplement Figs. S23–34). Lastly, our results were comparable, when using noise exposure categories identical with the previous Heathrow study (Online Supplement Figs. S35–36).

4. Discussion

The study considered long-term exposure to aircraft noise in 2006 and 2011 and health outcomes occurring between 2006 and 2015 covering a population of 3.1 million residents near London Heathrow, London Gatwick, Manchester, and Birmingham airports in England using a small area design. For Lden, there was strong evidence supporting increased risk of CHD hospital admissions near London Heathrow and for CVD hospital admissions near other airports. Results were not consistent across airports or noise metrics. Associations were more evident for hospital admissions than for mortality (where numbers were smaller). There was weak evidence for Lnight and mortality for CVD and CHD both at London Heathrow and other airports, but no comparable pattern of associations for Lden. There was no clear and consistent association of aircraft noise with stroke hospital admission or mortality. Sensitivity analyses showed similar patterns if only considering ages 55 + years.

4.1. Comparison with previous small area study of Heathrow

Findings were less pronounced in this study than our previous small area analysis of Heathrow considering aircraft noise 2001–5 (Hansell et al., 2013), where we had seen strong evidence of associations with each of CVD, CHD and stroke for hospital admissions and mortality with both daytime and night-time noise. For daytime noise (7 am to 11 pm, LAeq,16 h) in the previous study we found increased risk of hospital admission near Heathrow of 14 % (95 % CrI: 8 % to 20 %) for all CVD, 24 % (95 % CrI: 8 % to 43 %) for stroke and 21 % (95 % CrI: 12 % to 31 %) for CHD, comparing > 63 dB vs. ≤ 51 dB. In the present study using

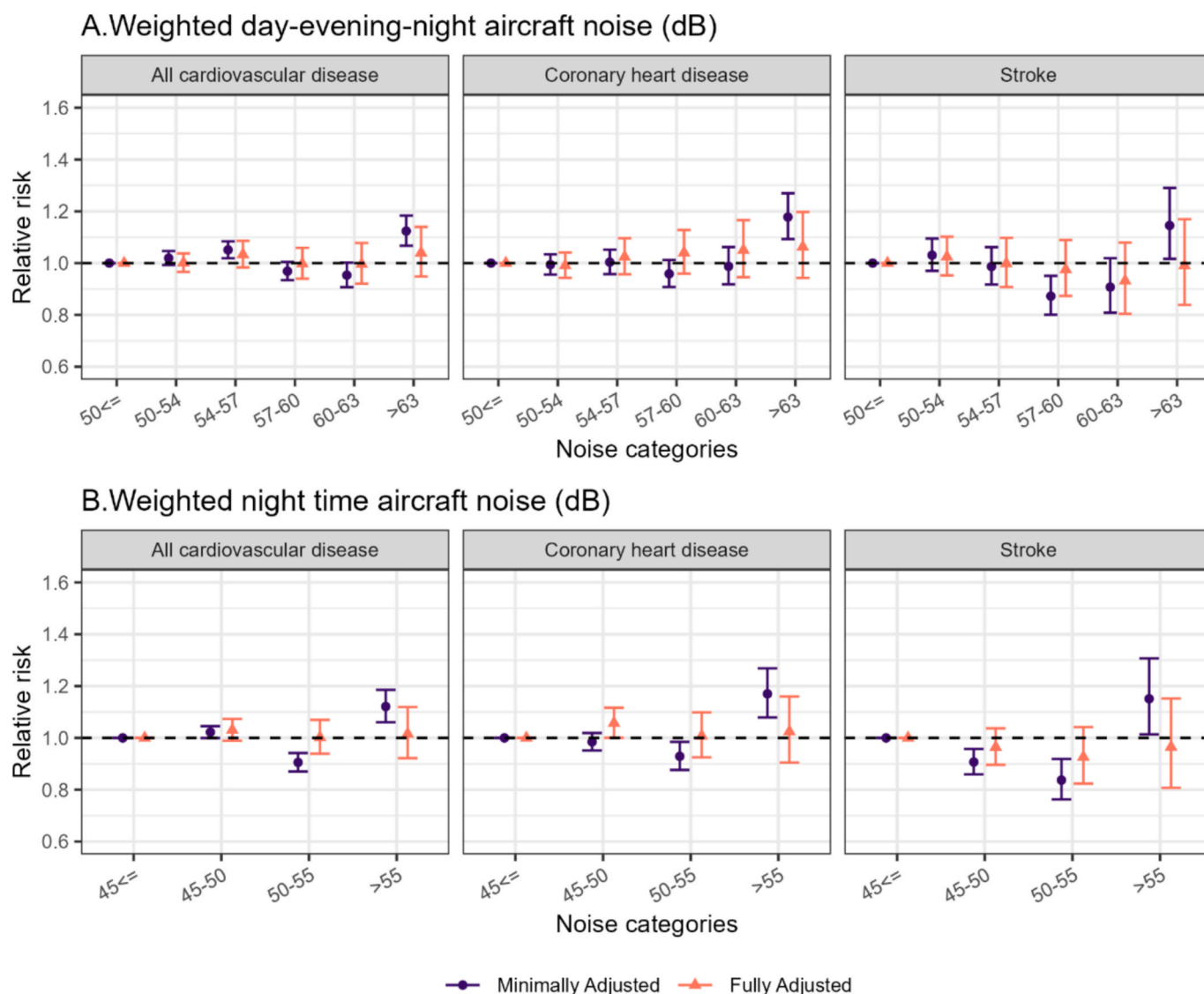


Fig. 3. Median relative **mortality** risk and 95% credible intervals for all cardiovascular disease, coronary heart disease and stroke using the minimally and fully adjusted models in **London Heathrow** during 2006–2016. Panel A. shows associations with long-term exposure to annual average population weighted day-evening-night aircraft noise. Panel B. shows associations with long-term exposure to annual average population weighted day night time aircraft noise. The minimally adjusted model does not include additional covariates but accounts for temporal trends, age and sex through the expected counts (see (1)-(2) in the Methods section), whereas the fully adjusted model additionally accounts for ethnicity, deprivation, road noise, rail noise, NO₂ exposure and spatial confounding.

Lden (which includes daytime as well as penalty-weighted night-time noise) and comparing > 63 dB vs. ≤ 50 dB we saw an increased risk of 8 % (95 % CrI: −0.9 % to 19 %) near Heathrow.

Reasons for differences in exposure–response over time may include changes in confounder structure e.g. ethnic mix in areas, lifestyle factors related to population change in city areas and a smaller therefore different study area than the previous study. The number of COAs near Heathrow airport exposed to appreciable aircraft noise in 2001 in our previous study (Hansell et al., 2013) was 12,110 (population 3.6 million) compared to 8,147 COAs in 2006 and 2011 in the present study (population 2.6 million), demonstrating widespread substantial reductions in aircraft noise exposure to local populations, but also reflecting potential changes in confounder distribution. Confounder adjustment made substantial attenuation to effect estimates compared with minimally adjusted results, with most substantial attenuation of the effect seen when accounting for deprivation and ethnicity (Online Supplement Figs. S23–34). We noted a very large effect of confounder adjustment around Heathrow for CHD at highest noise exposures (Fig. 2, Online Supplement Table S3) with similar issues but to a lesser extent on

stroke, and in the mortality analyses (Fig. 3). These potential residual confounding issues may help explain differences from our previous study (Hansell et al., 2013).

Other factors accounting for less clear associations in the present study for 2006–15 compared with the study of 2001–5 (Hansell et al., 2013) include reductions in aircraft noise exposure, especially at higher noise levels. Additionally, in 2014, Heathrow started to offer the Quieter Homes Scheme (QHS) for the residents living closest to the airport within the 69 dB LAeq and 16-hour aircraft noise contour (Kuhlmann et al., 2020). Home-owner and airport sponsored housing noise insulation may therefore have reduced indoor aircraft noise levels in areas of highest exposure. However, this would only apply to relatively small numbers of individuals within the study area and a limited amount of the study time period. We note that our metrics cover average noise levels and do not take account of other aspects of changes over this time period e.g. increasing numbers of flights and planned respite periods during the day.

Further differences over the time period of the original and current study include reductions in CVD mortality and incidence, related to

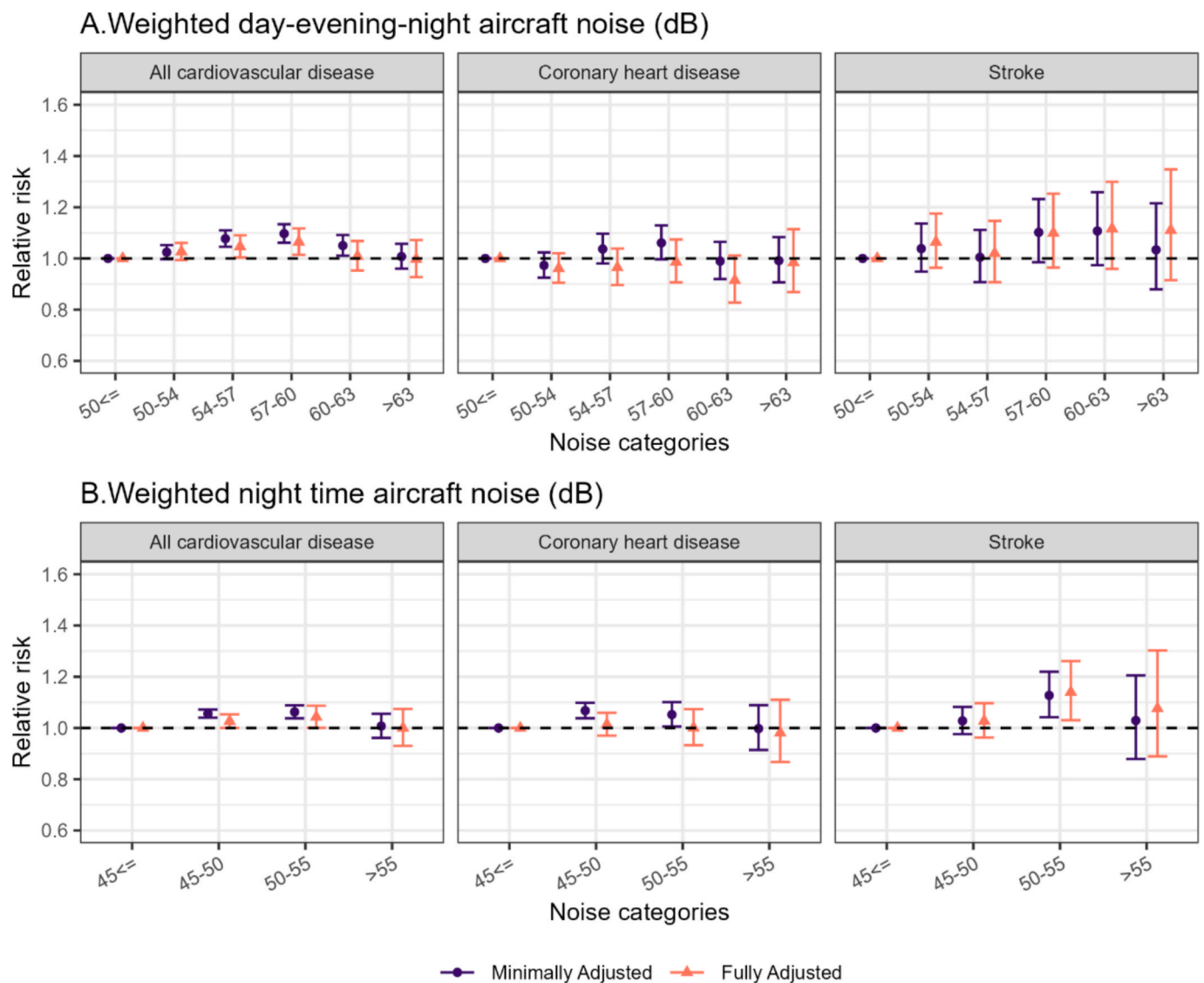


Fig. 4. Median relative **hospitalisation** risk and 95% credible intervals for all cardiovascular disease, coronary heart disease and stroke using the minimally and fully adjusted models in the London Gatwick, Manchester and Birmingham airports during 2006–2016. Panel A. shows associations with long-term exposure to annual average population weighted day-evening-night aircraft noise. Panel B. shows associations with long-term exposure to annual average population weighted night time aircraft noise. The minimally adjusted model does not include additional covariates but accounts for temporal trends, age and sex through the expected counts (see (1)–(2) in the Methods section), whereas the fully adjusted model additionally accounts for ethnicity, deprivation, road noise, rail noise, NO₂ exposure and spatial confounding.

improved reduction in risk factors such as smoking and better treatment of hypertension and heart disease in primary and secondary care, which may have made associations harder to detect (Hansell et al., 2013; Bhatnagar et al., 2016).

There were minor changes in study design compared with our previous study, but we do not think this will have affected findings materially. For example, we used the Lden metric (which includes daytime as well as penalty-weighted evening night-time noise) rather than daytime noise due to data availability; this aids comparability with other recent studies, most of which use Lden. We used slightly wider noise categories and provided a linear effect estimate rather than a linear test for trend across categories. Also, adjustments for road traffic noise used an improved model compared with the previous study.

4.2. Comparison with other studies

In addition to our previous small area study around Heathrow, we have also conducted a study of daily hospital admissions over the same

study area in 2014–18 using the case-crossover design. Similarly to the present study, we found strong evidence that previous evening and night-time exposures were associated with small increased risks for hospital admissions for all CVD but not for CHD or stroke, nor for mortality (Itzkowitz et al., 2023); with the advantage that the case-crossover design, while looking at acute rather than longer term effects of aircraft noise on health, is much less affected by possible confounding by sociodemographic factors. The only other short-term study to date of aircraft noise and cardiovascular outcomes looked at mortality in Switzerland (Saucy et al., 2021), but with higher temporal resolution than our London study (Itzkowitz et al., 2023). This found associations between aircraft noise in the two hours preceding death and both CVD and CHD mortality, but not stroke mortality. The null findings in both studies may have been affected by smaller numbers in CVD subgroup analyses.

Other epidemiological studies have found associations with aircraft noise and cardiovascular admissions and mortality. A US small area study of aircraft noise near 89 airports in 2009 (Correia et al., 2013)

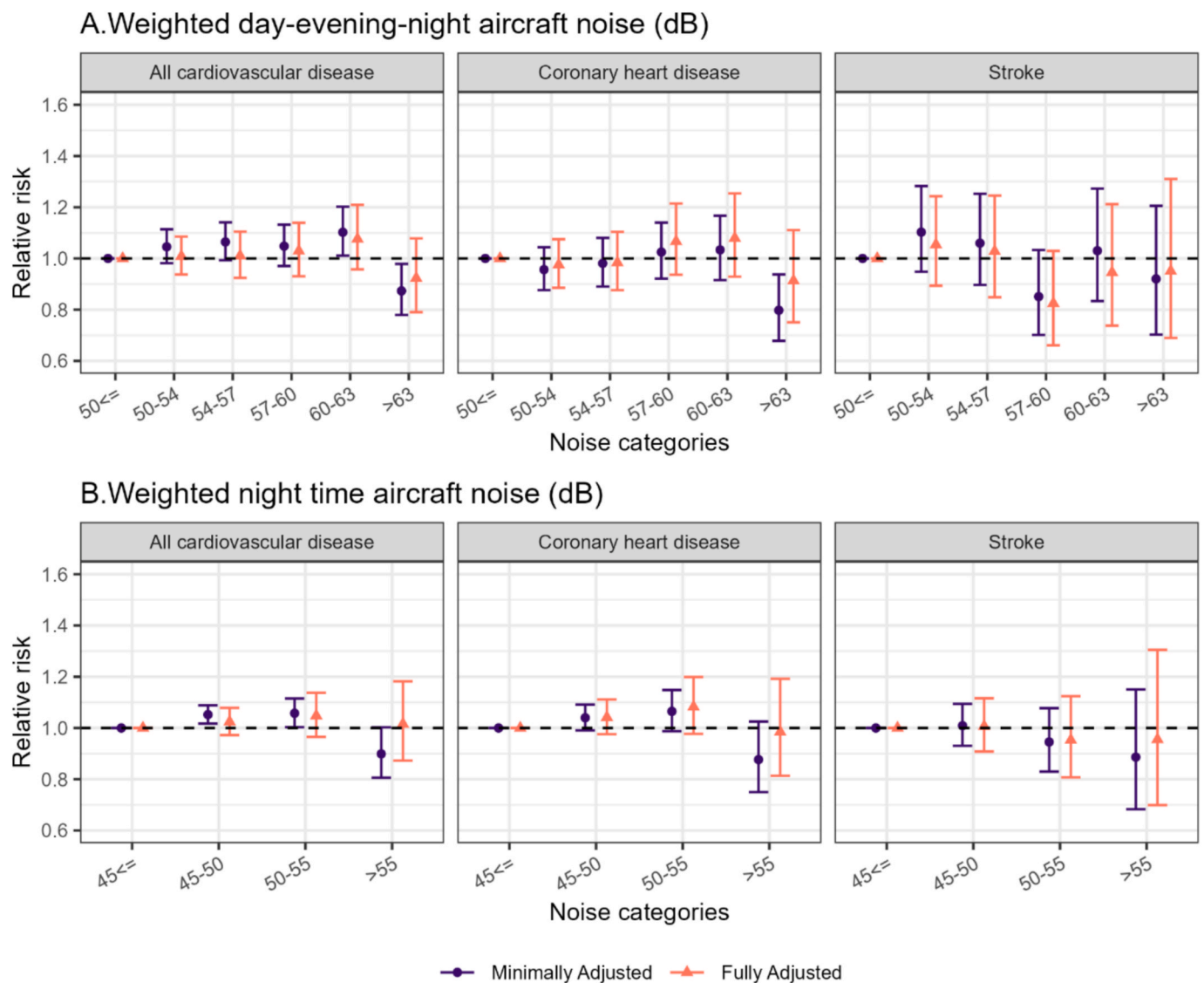


Fig. 5. Median relative mortality risk and 95% credible intervals for all cardiovascular disease, coronary heart disease and stroke using the minimally and fully adjusted models in the London Gatwick, Manchester and Birmingham airports during 2006–2016. Panel A. shows associations with long-term exposure to annual average population weighted day-evening-night aircraft noise. Panel B. shows associations with long-term exposure to annual average population weighted day night time aircraft noise. The minimally adjusted model does not include additional covariates but accounts for temporal trends, age and sex through the expected counts (see (1)-(2) in the Methods section), whereas the fully adjusted model additionally accounts for ethnicity, deprivation, road noise, rail noise, NO₂ exposure and spatial confounding.

found a 3.5 % (95 % CI: 0.2 % to 7.0 %) increase in risk of hospital admission for CVD per 10 dB higher aircraft noise in 6 million older people (>65 years). An epidemiological study of the Swiss National Cohort published in 2022 looking at aircraft noise 2001–2015 and mortality only (Vienneau et al., 2022) found increased risk of myocardial infarction (MI) and ischaemic stroke mortality associated with aircraft noise – in contrast, we did not find any evidence of associations with mortality in our fully adjusted analyses. We note the Swiss study suggested reductions of all CVD and stroke exposure–response coefficients over the five-year periods of 2001–5, 2006–10 and 2011–15.

The 2018 WHO Environmental Noise Guidelines for the European region (World Health Organization. Environmental noise guidelines for the European region., 2018) reported relative risk for CHD incidence (based on hospital admissions) of 1.09 (95 % CI 1.04–1.15) per annual average 10 dB increase in Lden noise for the European Region based on two ecological studies, (Kempen et al., 2018) but one of these was our 2001–5 study around Heathrow airport (Hansell et al., 2013). The WHO meta-analyses did not assess all CVD and did not find associations with

stroke. A meta-analysis by Vienneau et al published in 2019, (Vienneau, 2019) investigating CHD incidence using similar systematic review methods to the WHO but adding in three newer studies (one case-control and two cohort studies), found a small non-significant association between aircraft noise and risk of incident CHD (RR 1.03; 95 % CI: 0.98, 1.09), with high heterogeneity and high risk of bias. A meta-analysis of aircraft noise and mortality published in 2021 (Cai et al., 2021) reported a similar pooled relative risk 1.03 (95 % CI: 0.82 to 1.29) for CHD mortality with high heterogeneity, a statistically significant association for CVD mortality with RR 1.17 (95 % CI: 1.10 to 1.25) and non-significant association for stroke mortality RR 1.06 (95 % CI: 0.93 to 1.20), based on three studies including our previous Heathrow study (Hansell et al., 2013).

A recent meta-analysis by Minikin et al published in 2025 updated the WHO systematic review on heart disease used to formulate the 2018 Environmental Noise Guidelines for the European Union (World Health Organization. Environmental noise guidelines for the European region., 2018; Minkin et al., 2025). There were minor differences in definitions

with the present study – for example, the main outcome was heart disease, which included CHD, heart failure and atrial fibrillation. The meta-analysis found that aircraft noise was associated with an increased risk in heart disease mortality (RR = 1.07; 95 % CI, 1.01, 1.14 based on seven studies, of which three were small area studies) and heart disease incidence (RR = 1.03; 95 % CI 0.99, 1.07 – seven studies of which two were small area studies). For aircraft noise, the effect estimates changed from 1.07 to 1.03 for mortality and from 1.03 to 1.00 for incidence, when the small area studies were excluded. However, the authors noted that the small area studies, particularly the earlier Heathrow study (Hansell et al., 2013), included a relatively large population exposed to higher noise levels (>55 dB Lden) and that this may be important in terms of ability to detect health impacts of noise.

4.3. Biological plausibility

There is good biological plausibility for aviation noise impacts on human health (Hahad et al., 2019; Munzel et al., 2014; Peters et al., 2018). A recent review reported potential effects of noise on alterations of gene networks, epigenetic pathways, gut microbiome, circadian rhythm, signal transduction along the neuronal–cardiovascular axis, inflammation, and oxidative stress (Münzel et al., 2021). Experimental studies in humans have demonstrated association of short-term aircraft noise exposure with increase in endothelial dysfunction and systolic blood pressure, stress hormone release and measures of endothelial function, as well as sleep disturbance (Schmidt et al., 2013; Schmidt et al., 2015; Hahad et al., 2023). Epidemiological studies have demonstrated associations of long-term aircraft noise with cardiovascular risk factors. For instance, Pyko and colleagues (Pyko et al., 2019) found associations of long-term aircraft noise exposure with incident hypertension and with central obesity (Pyko et al., 2015). Our study of aircraft noise and cardiovascular MRI in UK Biobank cohort participants living near the four airports in the present study (Topriceanu et al., 2025) found that higher noise levels were associated with increases in left ventricular mass and walls with poorer left ventricular function, at least in part mediated by hypertension and body mass index. These types of cardiac changes were associated (in individuals not living near the airports) with fourfold increase in major adverse cardiac events (MACE). A further study also using UK Biobank participants, found associations between aircraft noise and disturbed sleep and sleep–wake cycle, suggesting impacts on circadian rhythms (Gong et al., 2024).

4.4. Strengths and Limitations

A strength of the study is its size, with administrative data available on ~ 3.1 million population with ~ 362,000 hospital admissions for CVD and ~ 61,000 CVD deaths. We used English administrative data on hospitalisation and mortality with excellent coverage. Comparing with cohort data, UK routine mortality statistics capture at least 80 % of myocardial infarction and stroke deaths (Delmestri and Prieto-Alhambra, 2020), while hospitalisation data for CVD and stroke has lower sensitivity (70 %) but high specificity (>95 %) (Kivimäki et al., 2017). We conducted separate analyses of Heathrow airport (with 83 % of the population) and the remaining airports combined. This ensured that Heathrow did not dominate the analyses and we were able to compare with our previous published study (Hansell et al., 2013). For other airports, we used of a probabilistic statistical modelling framework that allows borrowing of strength across multiple airports to assess exposure of interest, while accounting for area-specific variability through spatial random effects and airport-specific effects. A limitation is the group-level design, which is prone to ecological bias and limited confounder control, but this is offset to some extent by the high geographical resolution – each COA, the unit of analysis, comprised of 316 individuals on average.

We incorporated a spatially structured random effect at the small area level. This accounts for spatially varying factors we could not

readily get information on, such as access to and quality of clinical care. Additionally, a spatial random effect was included to help account for residual spatial confounding. This may, for example, be due to data quality, availability and choices relating to the selected ecological confounders including their temporal mismatch with the study period, their definition (e.g., groupings) and the use of estimates from exposure models which may introduce potential bias and lack of uncertainty. We examined how the results change with spatial adjustment, comparing our main spatial model with alternative ones (i) not including spatial random effects and (ii) including random effect but not accounting for the neighbourhood structure and confirmed that our final model with the BYM2 prior was the best performing. Nonetheless, we cannot rule out the risk of overadjustment but given the low correlation between the spatial random effects and Lden and Lnight, the risk of such overadjustment is likely small (Online Supplement Table S16).

Our exposure data were annual average aircraft noise data for two time periods, covering both time-weighted 24-hour exposures and night-time exposures. However, the Civil Aviation Authority only provides data to minimum noise values of 50 dB Lden and 45 dB Lnight (see methods), which means our study could not consider lower minimum noise thresholds such as 45 dB Lden and 40 dB Lnight as recommended by WHO Noise Guidelines for the European Region (World Health Organization, 2018). Consequently, our linear analysis should be interpreted as a linear threshold analysis, and we can only report the slopes above the threshold. Similarly, we could not consider more flexible modelling, for instance using splines, as they would only capture the right end tail of the distribution. Further, changes in aircraft flight patterns during the study period may not be fully captured by the Lden and Lnight metrics utilised in our present study e.g. we did not have information on number of flight events, on variability of noise exposure due to operational factors (e.g. runway changeovers), nor on intermittency ratio (percentage of noise energy perceived as individual events) which has been found to be associated with cardiovascular events (Vienneau et al., 2022).

We adjusted for key confounders including road and rail noise and NO₂ air pollution, as road traffic is an important pollution source near English airports. There is no readily available information on ultrafine particle exposures (UFPs), which are emitted by aircraft and likely to be highest near airports where noise levels are highest. However, the role of UFPs in cardiovascular morbidity and mortality is still being established. Further, our previous study measuring aircraft noise and UFPs near Gatwick airport (Tremper et al., 2022) found moderate to low correlations between the two exposures, suggesting that UFP would be unlikely to be an important confounder. We did not have information on individual confounders such as smoking, although we did adjust for deprivation and ethnicity, which are likely to be the important determinants of both lifestyle factors such as smoking and noise exposure. A potential additional issue is that our study was dominated by Heathrow airport (comprising 2.6 m of 3.1 m individuals living in the areas covered by the study). Each of the four airport areas had different confounder structures (Table S1). We have therefore presented results separately for other airports and Heathrow to aid comparisons with our previous Heathrow study.

There is not information on the level of noise insulation measures in homes around UK airports, but generally UK homes are poorly insulated against both temperature extremes and noise and the noise insulation schemes offered by UK airports are extremely limited, benefitting only a small proportion of houses and available only to those near the airport (see for instance for Heathrow (Heathrow. Residential Insulation Scheme. Accessed October 1, 2025). Hence, it is unlikely that not including measurement of insulation would have led to substantial exposure misclassification in the current study.

5. Conclusion

In conclusion, this small area study across four airports in England found some evidence of associations between Lden aircraft noise 2006–2015 and elevated risk of CVD and CHD hospital admissions (but not with mortality) or with stroke. Findings are consistent with our previous case-crossover study of aircraft noise and daily hospital admissions near Heathrow for 2014–18 (Itzkowitz et al., 2023), but contrasts with our earlier small area study for London Heathrow for 2001–2005 (Hansell et al., 2013), which found clear associations for each of CVD, CHD and stroke morbidity and mortality. Differences over time may relate to changes in population and confounder structure, and/or reductions in exposure to aircraft noise. A small area study design is by nature hypothesis-generating and findings need to be investigated further in large cohorts with better control of confounding at individual-level.

6. Ethics

The study was covered by national research ethics approval from the London-South East Research Ethics Committee – reference 22/LO/0256. Access to hospital admission and mortality data was covered by the Health Research Authority – Confidentiality Advisory Group under section 251 of the National Health Service Act 2006 and the Health Service (Control of Patient Information) Regulations 2002 – HRA CAG reference: 20/CAG/0028.

CRediT authorship contribution statement

Garyfallos Konstantinou: Writing – original draft, Visualization, Methodology, Formal analysis. **Glory Atilola:** Writing – original draft, Visualization, Methodology, Formal analysis. **Calvin Jephcote:** Writing – review & editing, Data curation. **Kathryn Adams:** Writing – review & editing, Data curation. **John Gulliver:** Writing – review & editing, Funding acquisition, Data curation, Conceptualization. **Paul Elliott:** Writing – review & editing, Funding acquisition, Conceptualization. **Anna L Hansell:** Writing – original draft, Funding acquisition, Conceptualization. **Marta Blangiardo:** Writing – original draft, Supervision, Methodology, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Marta Blangiardo, Paul Elliott, Glory Atilola, Garyfallos Konstantinou reports financial support was provided by National Institute for Health and Care Research. Marta Blangiardo, Anna Hansell, Paul Elliott, John Gulliver, Glory Atilola reports financial support was provided by UK Research and Innovation Medical Research Council. Anna Hansell, John Gulliver reports financial support was provided by National Institute for Health and Care Research. Anna Hansell reports financial support was provided by British Heart Foundation. Garyfallos Konstantinou reports financial support was provided by UK Research and Innovation Medical Research Council. Anna Hansell, John Gulliver reports a relationship with department of environment, food and rural affairs that includes: consulting or advisory. Anna Hansell reports a relationship with Health Data Research UK that includes: travel reimbursement. John Gulliver reports a relationship with Noise Consultants Limited that includes: consulting or advisory. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.envint.2025.109884>.

Data availability

The aircraft noise exposure data are available from CAA to other academic researchers on request. Health outcomes data were obtained from the UK Small Area Health Statistics Unit (SAHSU), which does not have permission to supply data to third parties. The data can be requested through the Office for National Statistics (<https://www.ons.gov.uk/>).

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