

Does night-time aircraft noise trigger mortality? A case-crossover study on 24 886 cardiovascular deaths

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Aims

It is unclear whether night-time noise events, including from aeroplanes, could trigger a cardiovascular death. In this study, we investigate the potential acute effects of aircraft noise on mortality and the specific role of different night-time exposure windows by means of a case-crossover study design.

Methods and results

We selected 24 886 cases of death from cardiovascular disease (CVD) from the Swiss National Cohort around Zürich Airport between 2000 and 2015. For night-time deaths, exposure levels 2 h preceding death were significantly associated with mortality for all causes of CVD [OR = 1.44 (1.03–2.04) for the highest exposure group ($L_{Aeq} > 50$ dB vs. <20 dB)]. Most consistent associations were observed for ischaemic heart diseases, myocardial infarction, heart failure, and arrhythmia. Association were more pronounced for females ($P = 0.02$) and for people living in areas with low road and railway background noise ($P = 0.01$) and in buildings constructed before 1970 ($P = 0.36$). We calculated a population attributable fraction of 3% in our study population.

Conclusion

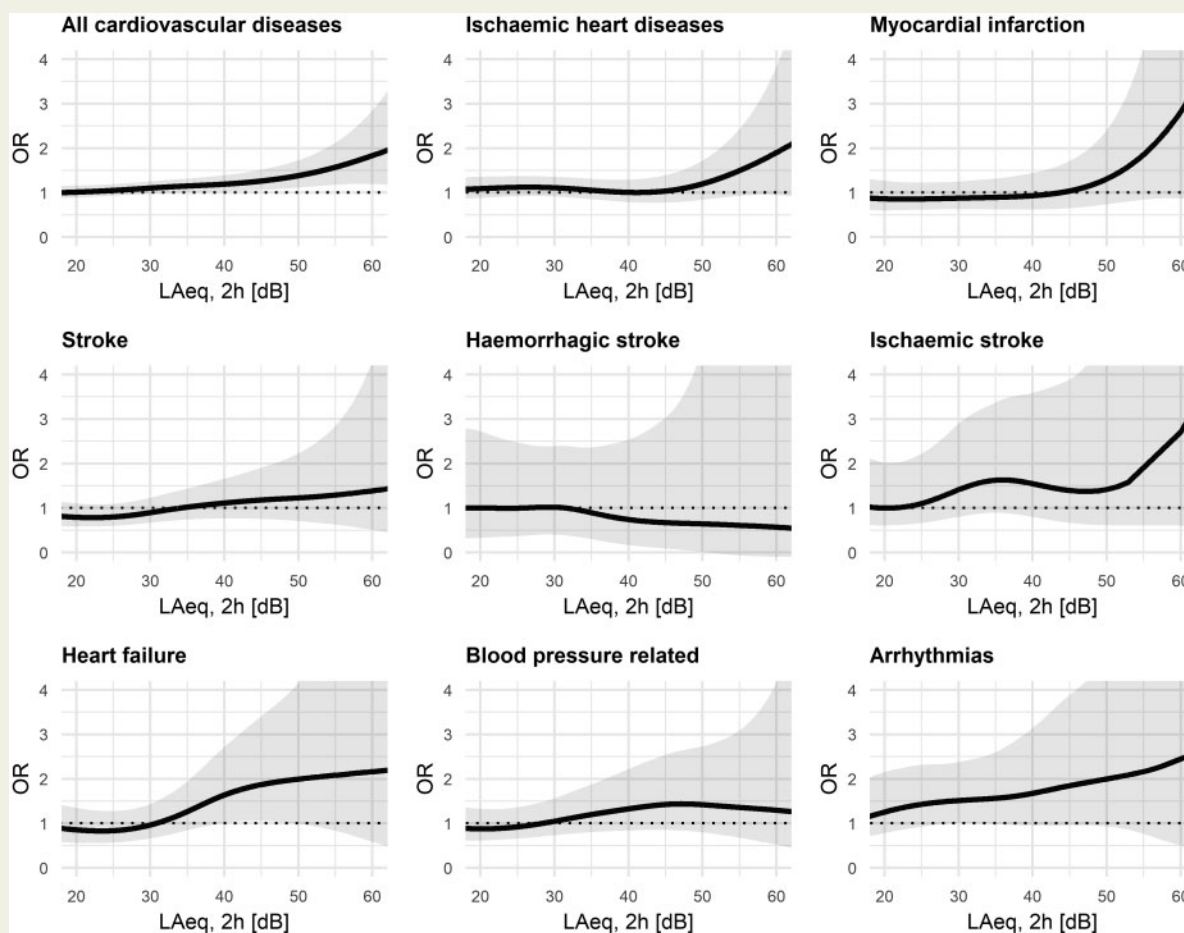
Our findings suggest that night-time aircraft noise can trigger acute cardiovascular mortality. The association was similar to that previously observed for long-term aircraft noise exposure.

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Graphical Abstract



Keywords

Aircraft noise • Cardiovascular mortality • Case-crossover • Acute effects

Introduction

It is estimated that environmental noise exposure contributes to 48 000 new cases of ischaemic heart diseases (IHD) or 156 000 DALYs in Europe each year.^{1,2} A meta-analysis performed for the new WHO noise guidelines considered all studies published until 2015 and reported that the risk for IHD increases by 8% per 10 dB increase in long-term road traffic noise.³ However, evidence for an association between aircraft noise and incidence of IHD and hypertension were rated of very low and low quality, respectively, due to the lack of longitudinal studies.^{2,3} Since then several studies—including longitudinal—and meta-analyses investigated the cardiovascular health effects of aircraft noise,^{4,5} providing the evidence of associations with IHD,⁶ including the incidence of hypertension (OR = 2.26 per 10 dB increase in L_{night} aircraft noise),⁷ myocardial infarction,^{8,9} heart failure,⁹ (ischaemic) stroke,^{5,6,8} and arrhythmias.⁷ It has also been suggested that

night-time noise exposure is particularly relevant for cardiovascular health, through sleep disruption and initiation of physiological stress reaction.^{10–12}

While most epidemiological studies addressed cardiovascular effects of long-term exposure to transportation noise, there is a need to better understand whether noise exposure also acts as a trigger for cardiovascular events and how the timing of noise exposure modulates this response.^{1,13} Experimental studies have shown acute responses to environmental noise on different physiological responses, such as endothelial dysfunction,¹⁴ hypertension,^{15,16} and sleep quality.^{17,18}

In the present study, we aim to investigate if and how night-time aircraft noise can trigger mortality for cardiovascular diseases (CVDs). We used mortality data from the Swiss National Cohort (SNC) over 15 years using a case-crossover design. We conducted separate analysis for night-time and daytime deaths and also tested three different noise exposure metrics to capture the characteristics and evolution of noise over time for various exposure windows.

Methods

Study design

We used a time-stratified case-crossover study design, in which each case of death was matched with up to four control days, chosen within the same month and same day of the week.¹⁹ The case-crossover design adjusts for any individual confounders that do not vary over a short period of time, such as age, smoking, or socio-economic status. This approach is particularly well suited to investigate acute risk effects with minimal bias²⁰ and has been largely applied to air pollution studies over the past two decades.²¹

With a focus on night-time noise exposure, we separately considered deaths occurring during the day (07:00–23:00), and deaths occurring during the night (23:00–07:00). For the night-time deaths, we considered a 2-h exposure window preceding death, as described for other triggers of acute cardiovascular mortality, such as air pollution and coffee intake.²² For daytime deaths, we investigated the effect of five different exposure windows defined a priori within the night preceding the day of event: overall night preceding the day of death (23:00–07:00); late evening (19:00–23:00); reduced air traffic reserved for delayed flights (23:00–23:30); core night (23:30–06:00); and early morning (06:00–07:00).

Study population

The SNC is a long-term cohort based on the linkage of national census and mortality records for the whole Swiss population.²³ It contains personal information as well as household, building, and mortality data, including hour and cause of death.²⁴ Based on a power analysis, we included mortality data from 4 December 2000 to 31 December 2015. We restricted the study population to adults over 30-year old with cardiovascular primary cause of death living near Zürich Airport (ZRH), using the envelope of the Zurich Aircraft Noise Index (ZFI) calculation perimeters from 2000 to 2016 for highly annoyed and highly sleep disturbed persons (Supplementary material online, Figures S1 and S2).²⁵ We considered the following primary causes of death (International Classification of Diseases ICD-10): all CVD (ICD-10: I00–I99), IHD (ICD-10: I20–I25), myocardial infarction (ICD-10: I21–I22), stroke (ICD-10: I60–I64), haemorrhagic stroke (ICD-10: I60–I62), ischaemic stroke (ICD-10: I63), heart failure (ICD-10: I50), blood pressure related death (ICD-10: I10–I15), and arrhythmias (ICD-10: I44–I49).

Aircraft noise exposure

Individual exposure to aircraft noise was estimated at home locations for the relevant time windows on the selected case and control nights and exposure time windows as previously described elsewhere.²⁶ In short, we used a list of all aircraft movements at ZRH between 2000 and 2015 and linked them with pre-existing outdoor aircraft noise exposure calculations at 250 m × 250 m resolution, specific for aircraft type, air route, time, and year.²⁷ From these data, we calculated three noise exposure metrics: (i) average A-weighted equivalent continuous sound pressure level (L_{Aeq}), (ii) maximum sound pressure level (L_{Amax}), and (iii) number of events above threshold 55 dB (NAT₅₅) for the pre-defined time windows defined above. In addition, we extracted long-term night-time exposure to railway and road traffic noise (L_{night}) at home locations for the year of death as calculated by Karipidis et al.²⁸ within the interdisciplinary SiRENE project.

Air pollution and meteorological exposure

We estimated nitrogen dioxide (NO₂), maximum temperature and precipitation data at home locations for all cases and control event dates (2 days averages). We used modelled daily NO₂ at 100 m × 100 m spatial resolution, available from 2005 onwards for Switzerland.²⁹ For the earlier years, we calculated individual NO₂ levels using routinely collected data

from the nearest 'Immissionsdatenbank Luft (IDB)' (IDB Luft, Bern, Switzerland) combined with the annual mean observed in 2005. Modelled daily maximum temperature and precipitation were available at a 2 km × 2 km resolution for the whole of Switzerland for all 15 years of the study.³⁰

Statistical analyses

The association between average aircraft noise and cardiovascular mortality was estimated using conditional logistic regression.^{19,31} We used all primary cardiovascular causes of death as the primary outcome and created separate models for night-time (2 h exposure) and daytime deaths (overall night and four separate night-time exposure windows). We used the L_{Aeq} as primary exposure—modelled as a penalized spline with four degrees of freedom to reflect the shape of the association between acute aircraft noise exposure and mortality—and adjusted for NO₂, maximum temperature, precipitation, and public holiday. We also computed separate models for the 2-h night-time noise exposure prior to death for three noise metrics (L_{Aeq} , L_{Amax} , and NAT₅₅) using categorical exposure variables to compute ORs for the noise exposure groups (10 dB exposure groups with reference at 20 and 40 dB for L_{Aeq} and L_{Amax} , respectively, and NAT₅₅ 0; 1–2; 2–5; 6–15; >15 events) and calculated 'P for trend' values, as an indication for exposure-response relationships.³² We investigated possible modification in the association between night-time 2h- L_{Aeq} and mortality for individual characteristics and subgroups of the population. We reported the change in odds ratio (OR) per 10 dB increase 2h- L_{Aeq} with 95% confidence intervals. Finally, we calculated the population attributable fraction (PAF) for cardiovascular mortality based on the risks calculated in the 2h- L_{Aeq} categorical models³³ (Supplementary material online, Equation S1).

Table 1 Study population characteristics

Population characteristics	Females (N = 13 269)	Males (N = 11 617)
Time of death, n (%)		
Daytime	9108 (70)	8137 (70)
Night-time	4161 (30)	3480 (30)
Age, mean (standard deviation)	84 (9)	78 (12)
Education level, n (%)		
Compulsory or less	6660 (50)	2170 (18)
Upper secondary	5756 (43)	6597 (57)
Tertiary	521 (4)	2629 (23)
Civil status, n (%)		
Single	1154 (9)	923 (8)
Married	2421 (18)	6865 (59)
Divorced	1197 (9)	1022 (9)
Widowed	8497 (64)	2807 (24)
Cause of death, n (%)		
Ischaemic heart diseases	4880 (37)	5641 (48)
Myocardial infarction	1342 (10)	1906 (16)
Stroke	2238 (17)	1512 (13)
Haemorrhagic stroke	469 (4)	361 (3)
Ischaemic stroke	350 (3)	277 (2)
Heart failure	1129 (8)	624 (5)
Blood pressure	1745 (13)	983 (8)
Arrhythmia	727 (5)	665 (6)

Results

We identified 24 886 adult deaths from cardiovascular cause between 2000 and 2015 within our study area in the vicinity of ZRH, of which 7641 occurred during the night and 17 245 during the day (Table 1). The mean L_{Aeq} ranged from 17.6 to 45.2 dB for the different exposure time windows. On average, all three noise metrics were highest in the evening time window (19:00–23:00) and lowest in the core night (23:30–06:00). L_{Amax} was highly correlated with L_{Aeq} (Pearson correlation coefficient = 0.91, $P < 0.001$). A more detailed description of the aircraft noise exposure metrics can be found elsewhere.²⁶

We found an association between 2 h aircraft noise exposure preceding the time of a cardiovascular death during the night (Figure 1). Indication of an association was specifically observed for IHD, myocardial infarction, heart failure, and arrhythmias. The odds of night-time cardiovascular mortality (all causes) was significantly increased for 2h- L_{Aeq} values above 40 dB with P for trend = 0.01 (Table 2). Linear exposure-response relationship was also significant for heart failure (P for trend = 0.05) and suggestive for IHD, without reaching significance ($P = 0.18$). We observed similar trends for 2h- L_{Amax}

exposure as for 2h- L_{Aeq} . For 2h-NAT, we observed a significant increase in odds of mortality for heart failure for exposure values above five events within the 2 h window preceding death (Supplementary material online, Table S1). The odds of mortality were significantly stronger among females than males, especially for arrhythmias (Figure 2). Due to the limited number of observations for some diagnoses (e.g. haemorrhagic and ischaemic strokes), power was insufficient to make any statement about the form shape of the exposure-response curve. Precision was generally lower for the highest exposure groups with fewer observations. Sensitivity analysis with additional adjustment for fine particles did not affect the results (Supplementary material online, Figure S3).

Based on our findings presented in Table 2, and assuming a causal relationship, we estimated that 782 out of 24 886 deaths in our study population could be attributed to aircraft noise thus representing a PAF of 3%.

We found that the association between aircraft noise and night-time cardiovascular deaths was significantly stronger for people living in quiet areas as compared to areas with higher night-time levels of road and railway noise and for people living in older buildings, most

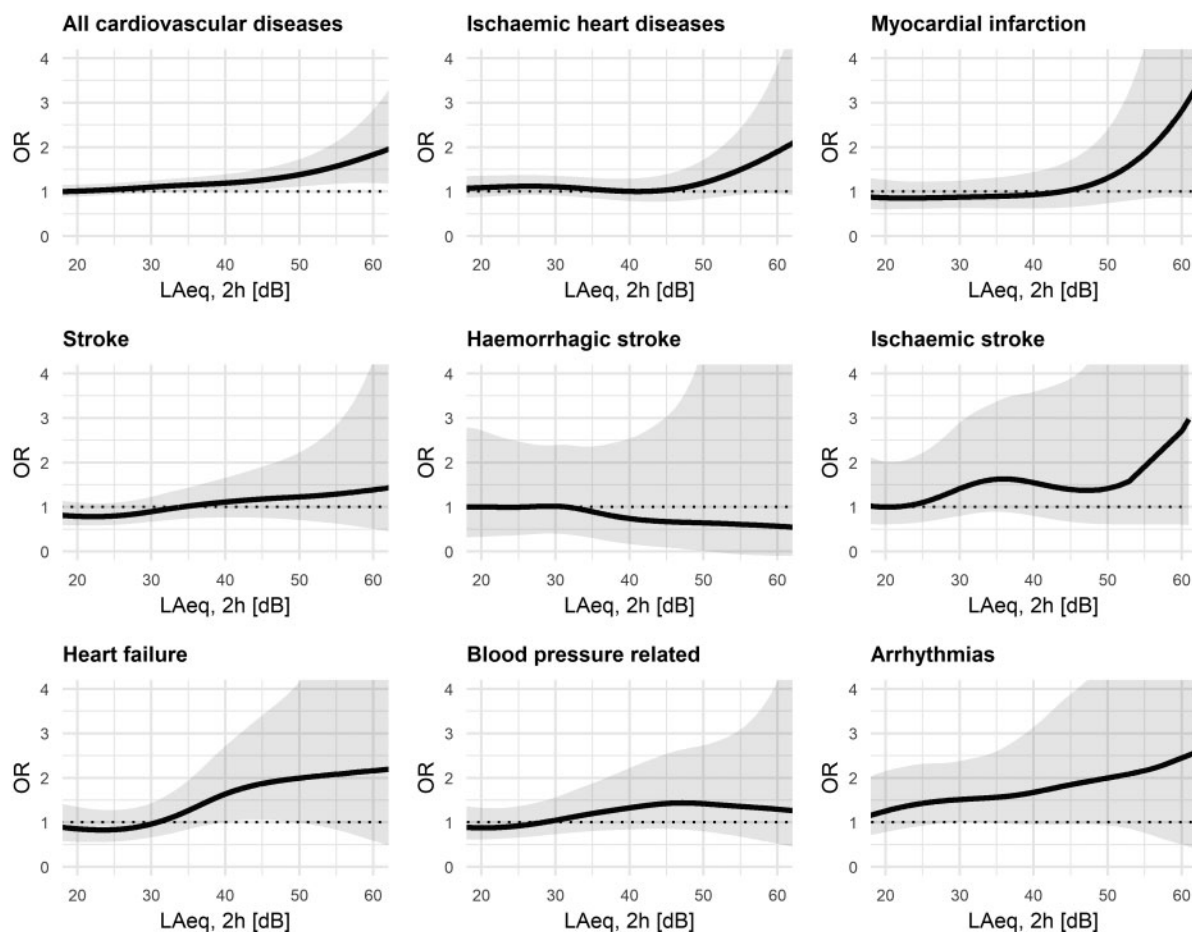
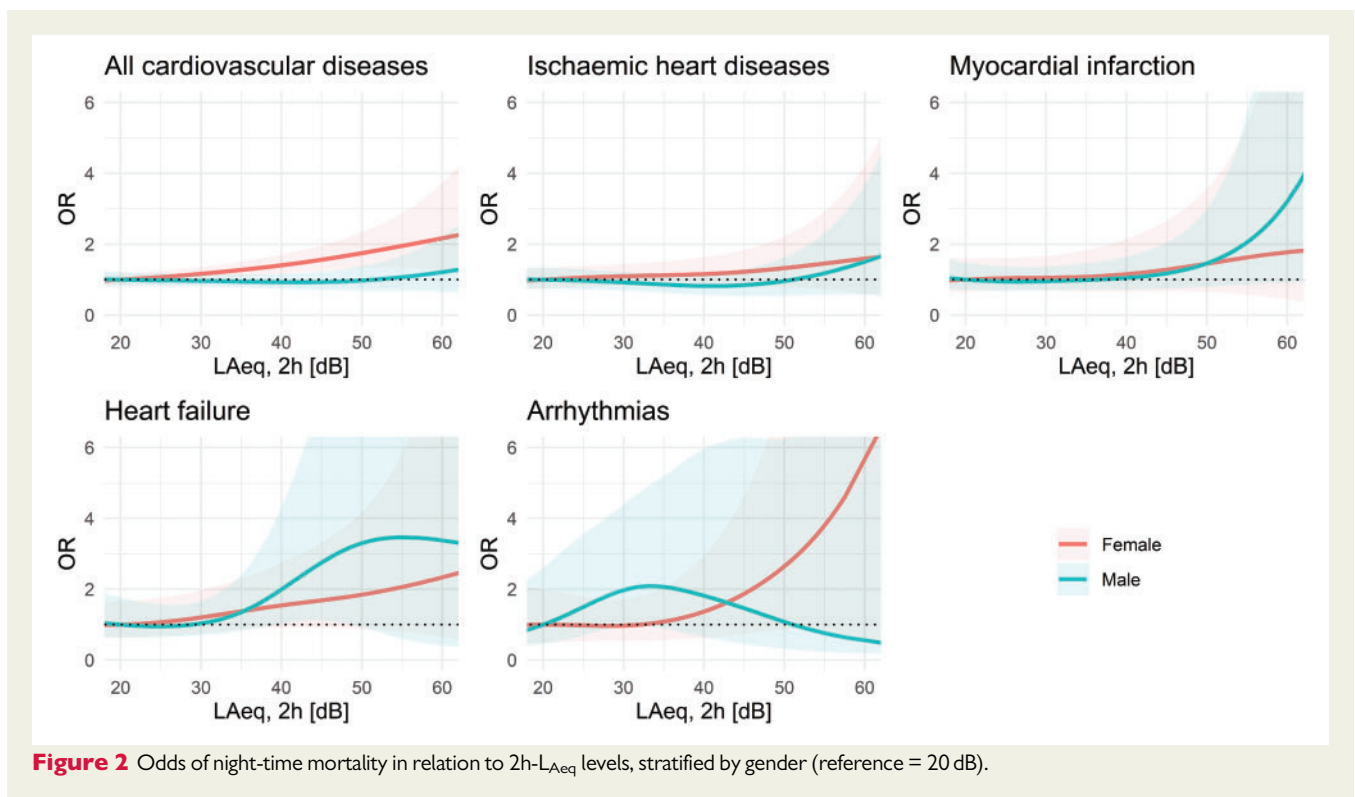


Figure 1 Odds of night-time mortality in relation to 2h- L_{Aeq} levels.

Table 2 Associations between night-time mortality from cardiovascular cause and noise exposure groups 2 h preceding death (2h-L_{Aeq})

Exposure groups	All cardiovascular diseases			Ischaemic heart diseases			Myocardial infarction			Heart failure			
	n	OR	95% CI	n	OR	95% CI	n	OR	95% CI	n	OR	95% CI	
<20 dB	4245	1		1797	1		527	1		229	1		
20–30 dB	824	1.08	(0.92–1.26)	360	1.15	(0.90–1.47)	101	1.11	(0.67–1.79)	69	1.11	(0.63–1.99)	
30–40 dB	1169	1.23	(1.00–1.51)	513	1.1	(0.80–1.51)	156	0.86	(0.45–1.64)	108	2.08	(1.01–4.29)	
40–50 dB	1157	1.33	(1.05–1.67)	479	1.13	(0.78–1.64)	152	0.93	(0.46–1.88)	74	2.07	(0.93–4.61)	
>50 dB	246	1.44	(1.03–2.04)	117	1.64	(0.96–2.79)	35	1.62	(0.62–4.25)	16	3.09	(0.94–10.23)	
Trend		P for trend = 0.01			P for trend = 0.18			P for trend = 0.57			P for trend = 0.05		

Statistically significant results at level alpha = 5% are marked in bold, adjusted for NO₂, temperature, precipitation, and holiday.

**Figure 2** Odds of night-time mortality in relation to 2h-L_{Aeq} levels, stratified by gender (reference = 20 dB).

likely with less efficient sound insulation (Table 3). We observed a stronger association for females [OR = 1.13 (1.04–1.23)] than for males [OR = 0.98 (0.89–1.07)] and found a lower risk of mortality for Swiss as compared to men from the rest of Europe. The association between 2h-L_{Aeq} and mortality tended to be stronger with decreasing education level and socio-economic status, as well as older age. Finally, the association between 2h-L_{Aeq} and mortality was modified by civil status although not significant ($P = 0.39$), with the highest odds of mortality observed for divorced people, while being married showed the lowest risk of mortality for males, and was equally high as divorced for females. Graphical representations of these effect modifications are available in [Supplementary material online, Figure S4](#).

For daytime deaths, no consistent risk increase was observed (Figure 3), although for arrhythmias, the morning exposure window (06:00–07:00) could be critical ([Supplementary material online, Figure S5](#)).

Discussion

This study suggests that aircraft noise events during night may trigger a cardiovascular death within 2 h. The PAF estimate of 3% is comparable to other triggers of cardiovascular mortality, such as anger, positive emotions, sexual activity, and heavy

Table 3 Effect modification of the association between 2h-L_{Aeq} and night-time cardiovascular mortality, stratified by gender

Covariates	All			Females ^a			Males		
	Cases	OR (95%CI)	P-int	Cases	OR (95%CI)	P-int	Cases	OR (95%CI)	P-int
Background noise			0.01			0.19			0.07
Low (Tertile 1)	2683	1.08 (1.02–1.15)		1497	1.15 (1.06–1.25)		1186	1.00 (0.91–1.09)	
Middle (Tertile 2)	2516	1.06 (1.00–1.13)		1337	1.14 (1.04–1.24)		1179	0.98 (0.90–1.08)	
High (Tertile 3)	2442	1.04 (0.98–1.11)		1327	1.11 (1.03–1.22)		1115	0.96 (0.87–1.05)	
Building period			0.36			0.93			0.18
Before 1970	4607	1.09 (1.01–1.18)		2481	1.14 (1.03–1.26)		2126	1.05 (0.94–1.17)	
1970–90	2243	1.04 (0.92–1.16)		1266	1.16 (1.00–1.45)		977	0.87 (0.73–1.05)	
After 1990	656	0.89 (0.71–1.12)		329	1.03 (0.74–1.43)		327	0.79 (0.57–1.09)	
Nationality			0.07			0.93			0.01
Swiss	6727	1.06 (0.99–1.12)		3759	1.14 (1.05–1.24)		2968	0.96 (0.87–1.06)	
Rest of Europe	415	1.37 (1.06–1.78)		161	1.11 (0.76–1.62)		254	1.66 (1.14–2.41)	
Other or unknown	499	0.90 (0.70–1.16)		241	1.07 (0.77–1.50)		258	0.74 (0.51–1.08)	
Education			0.36			0.77			0.27
Compulsory or less	2756	1.09 (0.99–1.20)		2085	1.16 (1.04–1.29)		671	0.94 (0.78–1.13)	
Upper secondary	3756	1.07 (0.98–1.17)		1814	1.09 (0.96–1.24)		1942	1.05 (0.93–1.19)	
Tertiary	949	0.91 (0.75–1.10)		153	1.43 (0.81–2.52)		796	0.85 (0.69–1.05)	
SSEP ^b			0.81			0.76			0.72
Quintile 1 (lowest)	802	1.10 (0.93–1.31)		448	1.24 (1.01–1.52)		354	0.86 (0.63–1.16)	
Quintile 2	1115	1.10 (0.95–1.28)		630	1.23 (1.01–1.51)		485	0.96 (0.77–1.20)	
Quintile 3	1331	1.09 (0.95–1.27)		693	1.08 (0.88–1.33)		638	1.11 (0.91–1.36)	
Quintile 4	1907	1.07 (0.95–1.21)		1056	1.14 (0.97–1.34)		851	1.00 (0.83–1.20)	
Quintile 5 (highest)	2016	0.98 (0.87–1.11)		1007	1.04 (0.87–1.25)		1009	0.93 (0.79–1.10)	
Civil status			0.39			0.72			0.22
Married	2746	1.04 (0.94–1.15)		733	1.22 (1.01–1.48)		2013	0.98 (0.87–1.11)	
Widowed	3572	1.03 (0.95–1.13)		2691	1.10 (0.99–1.22)		881	0.87 (0.73–1.04)	
Single	660	1.14 (0.94–1.40)		374	1.16 (0.90–1.49)		286	1.13 (0.81–1.58)	
Divorced	663	1.22 (1.00–1.51)		363	1.22 (0.94–1.58)		300	1.25 (0.89–1.75)	
Age			0.71			0.79			0.82
≤65	664	1.02 (0.82–1.27)		170	1.07 (0.68–1.68)		494	1.00 (0.78–1.29)	
>65	6977	1.06 (1.00–1.13)		3991	1.14 (1.05–1.24)		2986	0.97 (0.88–1.07)	

Statistically significant results at level alpha = 5% are marked in bold.

^aThe association between L_{Aeq} and all cardiovascular disease mortality with stronger for females than for males (P-interaction = 0.02).

^bSSEP, Swiss neighbourhood index of socioeconomic position.²⁴

meals,²² and to previous estimates for long-term aircraft noise exposure.³⁴

To the best of our knowledge this is the first study investigating acute aircraft noise effects on mortality. Our current findings are broadly in line with our previous study investigating the association between long-term noise and cardiovascular mortality in Switzerland, which also found higher mortality for heart failure, myocardial infarction, and ischaemic stroke associated with aircraft noise and absence of associations for other cardiovascular causes of deaths.³⁵ They also align with studies in other countries on long-term health outcomes observed near airports,^{5,36} and acute physiological responses to night-time aircraft noise. We used a spline to model the odds of mortality, without any a priori assumption on the shape of the association. During night and shorter time windows, we have observed higher day-to-day variation, which is

most relevant for a case-crossover analysis. Except for arrhythmias and all CVD combined, where we observed steadily increasing exposure–response relationships with L_{Aeq}, the shape of the splines suggests a possible threshold in the range of 30–50 dB, which is rather low compared to studies on long-term night-time aircraft noise.^{1,4} However, these 2h-L_{Aeq} values might very well represent audible individual flight events when those occur during night-time exposure windows with only few flights per hour ([Supplementary material online, Figure S6](#)).³⁷

We found suggestive evidence for effect modification by gender, nationality, civil status, and background transportation noise. Existing literature on gender differences and the effects of noise on health is rather inconsistent.^{6,13} Our study suggests a stronger risk of mortality among females for CVDs (all causes) but not for IHD, myocardial infarction, and heart failure. The gender difference observed in all

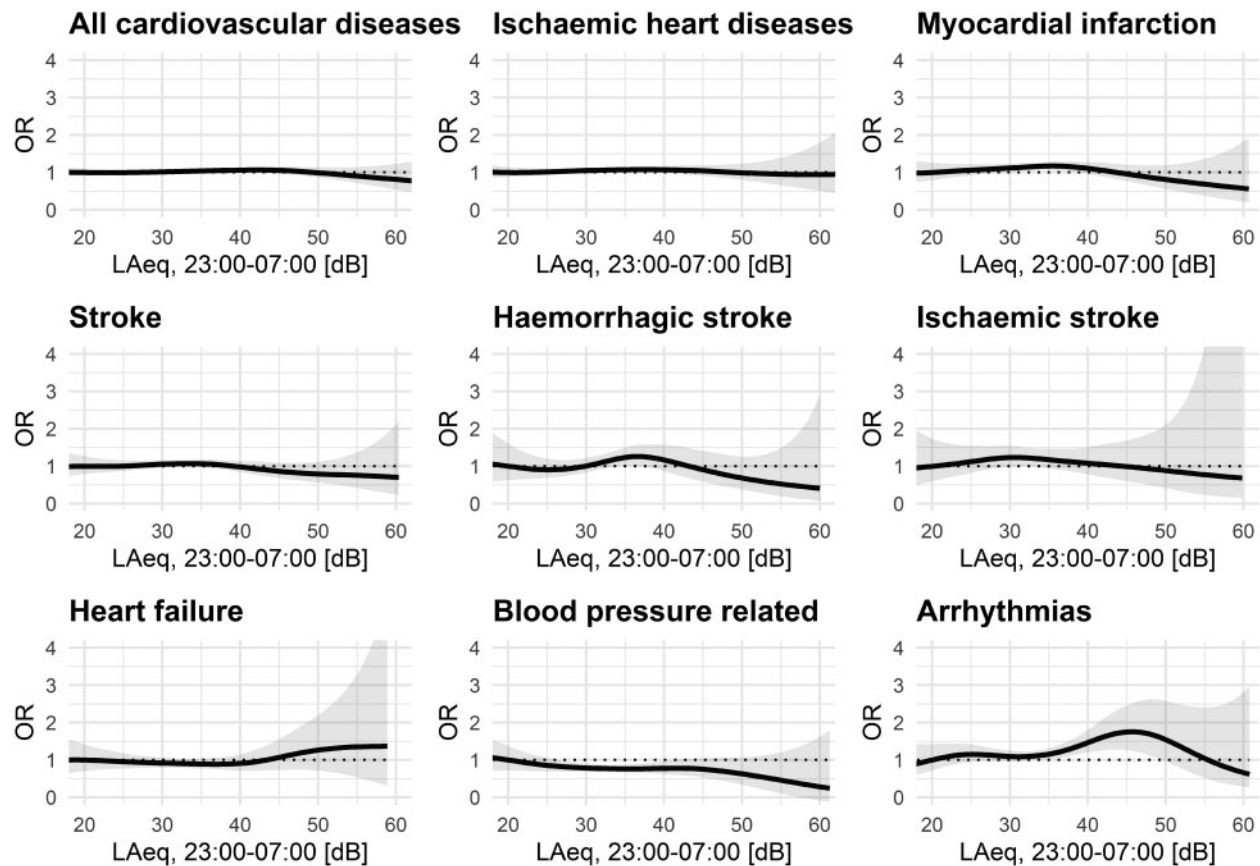


Figure 3 Odds of daytime mortality in relation to night-time L_{Aeq} (23:00–07:00) levels of the preceding night.

causes of cardiovascular mortality is likely to be driven by a stronger association for arrhythmias, as well as a general older age at death among females. Pyko *et al.*⁶ suggested that higher risk of mortality among females may be due to possible higher susceptibility to stress response, as shown in higher salivary cortisol in response to noise exposure. The association between aircraft noise and mortality was more pronounced in areas with little railway and road traffic background noise. Single flight events are indeed more likely to be perceived and to cause potential physiological response in a quiet environment as compared to a noisy environment with possible noise masking. We also observed higher effect size for people living in older buildings, where sound insulation is likely to be less efficient than in more recent constructions. This may explain part of the observed higher risk in people with lower socio-economic status usually living in less costly residential buildings. Our findings thus suggest that such social characteristics can affect the individual risk of mortality associated to aircraft noise exposure. Social inequities may therefore not only represent differences in environmental exposures levels as previously described³⁸ but also affect the risk of mortality from environmental factors.

A particular strength of this study is the high precision aircraft noise modelling accounting for single, specific flight events, yielding individual aircraft noise exposure estimates with high spatial and

temporal accuracy for each death case. Nevertheless, some exposure misclassification is unavoidable, for instance due to inaccurate location (errors in the address history, residents not at home) or imprecision from the aircraft calculations (individual flights slightly deviating from the usual air routes). A strength of the case-crossover design is, however, that health risks are estimated based on the exposure difference between case and control events for the same person and the same location, thus minimizing bias from errors in exposure modelling. The choice of exposure time window can also be critical. In this study, we a priori considered a 2-h exposure window for night-time deaths, as described for other triggers of cardiovascular mortality.²² We cannot exclude that the time between first symptoms onset and death might exceed this delay in some cases, especially for patients dying in hospital or patients living alone with possible imprecision in time of death. The proportion of deaths occurring at home vs. in hospital is unknown in our study population, but evidence from other countries suggests that this number is rather limited.³⁹ In both cases, misclassification is expected to be independent from exposure levels and health outcomes, resulting in an overall underestimation of the risk estimates.

Due to the night-time flight restriction in place at ZRH, the observed night-time aircraft noise levels were rather low. Further research should investigate if similar results can be reproduced in other

locations, also including higher night-time exposure levels and individual sleeping patterns whenever available. We found only weak indication that night-time aircraft noise exposure (i.e. mainly early morning exposure), might also affect mortality occurring the next day. This may indicate that acute physiological reaction from aircraft noise is restricted to a few hours and does not persist into the following day. Longer averaged exposure intervals are also less susceptible to show important day-to-day variations. Furthermore, we did not consider daytime noise exposure for the analysis of daytime deaths because we suspected substantial exposure misclassification when people are not at home during the day. Thus, future studies with appropriate design are needed to clarify whether aircraft noise during the day may also trigger cardiovascular deaths. Better understanding of the effects of noise characteristics and timing on health outcomes could have important implications on noise regulations, such as night-time road speed reduction or air traffic regulations.⁴⁰

Conclusions

Our findings suggest that night-time aircraft noise events may trigger cardiovascular deaths, which would explain 3% of all cases of death from cardiovascular cause in our population living in the vicinity of an international airport if this association was causal. Our study suggests that night-time aircraft noise exposure may be of particular importance in relation to IHD and heart failure, as also found for long-term exposures.

Data availability

The data underlying this article cannot be shared publicly due to the sensitivity of individual data used in the study. The data will be shared on reasonable request to the corresponding author.

Supplementary material

Supplementary material is available at *European Heart Journal* online.

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