

Löst nächtlicher Fluglärm die Sterblichkeit aus? Eine Fall-Crossover-Studie zu 24 886 kardiovaskulären Todesfällen

Apolline Saucy , Beat Schäffer , Louise Tangermann , Danielle Vienneau , Jean-Marc Wunderli , Martin Rösli 

European Heart Journal , ehaa957, <https://doi.org/10.1093/eurheartj/ehaa957>

Veröffentlicht: 30. November 2020 Artikel Geschichte 

Abstrakt

Ziele

Es ist unklar, ob nächtliche Lärmereignisse, auch von Flugzeugen, einen kardiovaskulären Tod auslösen könnten. In dieser Studie untersuchen wir die möglichen akuten Auswirkungen von Fluglärm auf die Mortalität und die spezifische Rolle verschiedener nächtlicher Expositionsfenster anhand eines Fall-Crossover-Studiendesigns.

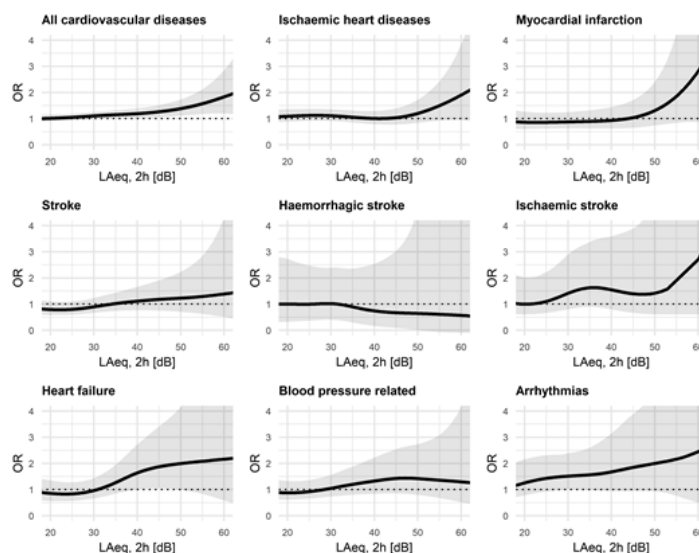
Methoden und Ergebnisse

Wir haben zwischen 2000 und 2015 24 886 Todesfälle durch Herz-Kreislauf-Erkrankungen (CVD) aus der Schweizerischen Nationalkohorte am Flughafen Zürich ausgewählt. Bei nächtlichen Todesfällen waren die Expositionsniveaus 2 Stunden vor dem Tod für alle Ursachen von CVD signifikant mit der Mortalität assoziiert [OR = 1,44 (1,03–2,04) für die höchste Expositionsguppe ($L_{Aeq} > 50$ dB gegenüber < 20 dB)]. Die konsistentesten Assoziationen wurden für ischämische Herzerkrankungen, Myokardinfarkt, Herzinsuffizienz und Arrhythmie beobachtet. Die Assoziationen waren bei Frauen ($P = 0,02$) und bei Menschen, die in Gebieten mit geringem Straßen- und Eisenbahnhintergrundgeräusch ($P = 0,01$) und in Gebäuden, die vor 1970 (gebaut $P = 0,36$) leben, stärker ausgeprägt. Wir haben einen bevölkerungsbezogenen Anteil von 3% in unserer Studienpopulation berechnet.

Fazit

Unsere Ergebnisse legen nahe, dass nächtlicher Fluglärm eine akute kardiovaskuläre Mortalität auslösen kann. Die Assoziation ähnelte der zuvor für die langfristige Lärmbelastung von Flugzeugen beobachteten.

Grafische Zusammenfassung



Keywords: Fluglärm , kardiovaskuläre Mortalität , Fallkreuzung , akute Effekte

Thema: Herzrhythmusstörung, Herzinfarkt, Herz-Kreislauf-Erkrankungen, Herzinsuffizienz, Flugzeug, auslösende Faktoren, Mortalität, Lärm, kardiovaskulärer Tod, Nachtzeit, Lärmbelastung

Themenbereich: Klinische Forschung

PDF

Hilfe

Einführung

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 (NAT_{55}) 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 (NO_2), maximum temperature and precipitation data at home locations for all cases and control event dates (2 days averages). We used modelled daily NO_2 at 100 m × 100 m spatial resolution, available from 2005 onwards for Switzerland.²⁹ For the earlier years, we calculated individual NO_2 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 NO_2 , 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 NAT_{55}) 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 NAT_{55} 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).

Results

In unserem Untersuchungsgebiet in der Nähe des ZRH identifizierten wir zwischen 2000 und 2015 24 886 Todesfälle bei Erwachsenen aufgrund kardiovaskulärer Ursachen, von denen 7641 nachts und 17 245 tagsüber auftraten (*Tabelle 1*). Der mittlere L_{Aeq} lag für die verschiedenen Belichtungszeitfenster im Bereich von 17,6 bis 45,2 dB. Im Durchschnitt waren alle drei Geräuschmetriken im Abendzeitfenster am höchsten (19: 00–23: 00) und in der Kernnacht am niedrigsten (23: 30–06: 00). L_{Amax} korrelierte stark mit L_{Aeq} (Pearson-Korrelationskoeffizient = 0,91, $P < 0,001$). Eine detailliertere Beschreibung der Fluglärm-Expositionsmetriken finden Sie an anderer Stelle.²⁶

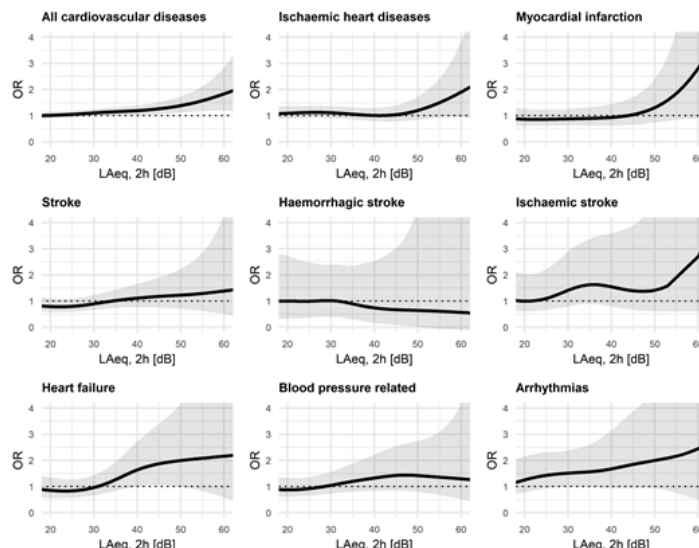
Tabelle 1 Populationsmerkmale untersuchen

| Bevölkerungsmerkmale | Frauen (N = 13 269) | Männer (N = 11 617) |
|--|-----------------------|-----------------------|
| Zeitpunkt des Todes, n (%) | | |
| Tagsüber | 9108 (70) | 8137 (70) |
| Nachtzeit | 4161 (30) | 3480 (30) |
| Alter, Mittelwert (Standardabweichung) | 84 (9) | 78 (12) |
| Bildungsniveau, n (%) | | |

| | | |
|-----------------------------|-----------|-----------|
| Obligatorisch oder weniger | 6660 (50) | 2170 (18) |
| Sekundarstufe | 5756 (43) | 6597 (57) |
| Tertiär | 521 (4) | 2629 (23) |
| Zivilstatus, <i>n</i> (%) | | |
| Single | 1154 (9) | 923 (8) |
| Verheiratet | 2421 (18) | 6865 (59) |
| Geschieden | 1197 (9) | 1022 (9) |
| Verwitwet | 8497 (64) | 2807 (24) |
| Todesursache, <i>n</i> (%) | | |
| Ischämische Herzkrankheiten | 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) |

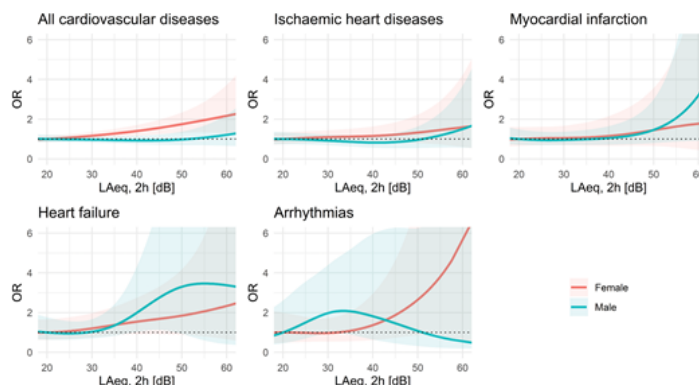
We found an association between 2 h aircraft noise exposure preceding the time of a cardiovascular death during the night (*Figure 1*). Indication of an association was specifically observed for IHD, myocardial infarction, heart failure, and arrhythmias. The odds of night-time cardiovascular mortality (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](#)).

Figure 1



Wahrscheinlichkeit der Nachtsterblichkeit in Bezug auf 2h-L_{Aeq} Werte.

Figur 2



Wahrscheinlichkeit der Nachtmortalität in Bezug auf 2h-L_{Aeq} Werte, geschichtet nach Geschlecht (Referenz = 20 dB).

Tabelle 2 Assoziationen zwischen nächtlicher Mortalität aufgrund kardiovaskulärer Ursachen und Lärmbelastungsgruppen 2 h vor dem Tod (2h-L_{Aeq})

| Belichtungsgruppen | Alle Herz-Kreislauf-Erkrankungen | | | Ischämische Herzkrankheiten | | | Herzinfarkt | | | Herzinsuff | |
|--------------------|----------------------------------|-------------|--------------------|-----------------------------|------|-------------|--------------------|------|-------------|-------------|------------|
| | n | ODER | 95% CI | n | ODER | 95% CI | n | ODER | 95% CI | n | OD |
| <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 | 1.1 |
| 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 | 2.0 |
| 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 | 2.0 |
| >50 dB | 246 | 1.44 | (1.03–2.04) | 117 | 1.64 | (0.96–2.79) | 35 | 1.62 | (0.62–4.25) | 16 | 3.0 |
| Trend | P for trend = 0.01 | | | P for trend = 0.18 | | | P for trend = 0.57 | | | P for trend | |

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

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 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} und die Sterblichkeit wurde durch den Zivilstatus modifiziert, obwohl sie nicht signifikant war ($P = 0,39$), wobei die höchste Sterblichkeitswahrscheinlichkeit für geschiedene Personen beobachtet wurde, während die Ehe das geringste Sterblichkeitsrisiko für Männer aufwies und ebenso hoch war wie für geschiedene Frauen. Grafische Darstellungen dieser Effektmodifikationen finden Sie im [ergänzenden Online-Material, Abbildung S4](#).

Tisch 3 Effektmodifikation der Assoziation zwischen 2h-L_{Aeq} und nächtlicher kardiovaskulärer Mortalität, geschichtet nach Geschlecht

| Covariates | Alle | | | Frauen ^a | | | Männer | | | P |
|----------------------------|-------|-------------------------|-------------|---------------------|-------------------------|-------|--------|-------------------------|-----------|---|
| | Fälle | ODER (95% CI) | P-int | Fälle | ODER (95% CI) | P-int | Fälle | ODER (95% CI) | P | |
| Hintergrundgeräusche | | | 0,01 | | | 0,19 | | | 0, | |
| Niedrig (Tertil 1) | 2683 | 1,08 (1,02–1,15) | | 1497 | 1,15 (1,06–1,25) | | 1186 | 1,00 (0,91–1,09) | | |
| Mitte (Tertil 2) | 2516 | 1,06 (1,00–1,13) | | 1337 | 1,14 (1,04–1,24) | | 1179 | 0,98 (0,90–1,08) | | |
| Hoch (Tertil 3) | 2442 | 1,04 (0,98–1,11) | | 1327 | 1,11 (1,03–1,22) | | 1115 | 0,96 (0,87–1,05) | | |
| Bauzeit | | | 0,36 | | | 0,93 | | | 0, | |
| Vor 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) | | |
| Nach 1990 | 656 | 0,89 (0,71–1,12) | | 329 | 1,03 (0,74–1,43) | | 327 | 0,79 (0,57–1,09) | | |
| Staatsangehörigkeit | | | 0,07 | | | 0,93 | | | 0, | |
| schweizerisch | 6727 | 1,06 (0,99–1,12) | | 3759 | 1,14 (1,05–1,24) | | 2968 | 0,96 (0,87–1,06) | | |
| Rest von Europa | 415 | 1,37 (1,06–1,78) | | 161 | 1,11 (0,76–1,62) | | 254 | 1,66 (1,14–2,41) | | |
| Andere oder unbekannt | 499 | 0,90 (0,70–1,16) | | 241 | 1,07 (0,77–1,50) | | 258 | 0,74 (0,51–1,08) | | |
| Bildung | | | 0,36 | | | 0,77 | | | 0, | |
| Obligatorisch oder weniger | 2756 | 1,09 (0,99–1,20) | | 2085 | 1,16 (1,04–1,29) | | 671 | 0,94 (0,78–1,13) | | |
| Sekundarstufe | 3756 | 1,07 (0,98–1,17) | | 1814 | 1,09 (0,96–1,24) | | 1942 | 1,05 (0,93–1,19) | | |
| Tertiär | 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, | |
| 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, | |
| 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. |
| ≤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) |

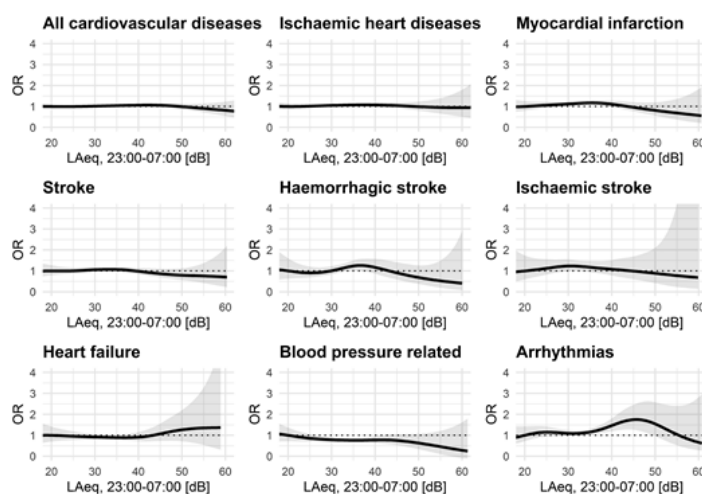
Statistisch signifikante Ergebnisse bei Alpha = 5% sind fett markiert.

ein Der Zusammenhang zwischen L_{Aeq} und der Mortalität aller Herz-Kreislauf-Erkrankungen ist bei Frauen stärker als bei Männern (P -Wechselwirkung = 0,02).

b SSEP, Schweizer Nachbarschaftsindex der sozioökonomischen Position.²⁴

Bei Todesfällen am Tag wurde kein konsistenter Risikoanstieg beobachtet (Abbildung 3), obwohl bei Arrhythmien das morgendliche Expositionsfenster (06:00–07:00) kritisch sein könnte (ergänzendes Online-Material, Abbildung S5).

Figur 3



Wahrscheinlichkeit der Tagesmortalität in Bezug auf die Nacht- L_{Aeq} -Werte (23:00–07:00) der vorhergehenden Nacht.

Diskussion

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 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 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.

Acknowledgements

We thank the Swiss Federal Statistical Office for providing mortality and census data and for the support that made the Swiss National Cohort and this study possible. The members of the Swiss National Cohort Study Group are Matthias Egger, Adrian Spoerri, Marcel Zwahlen, Milo Puhan, Matthias Bopp, Martin Rösli, Michel Oris, and Murielle Bochud. Meteorological information was obtained from MeteoSwiss. Calculations were performed at sciCORE (<http://scicore.unibas.ch/>) scientific computing centre at the University of Basel. We thank Martin Bissegger of Zürich Airport for the permission to use the lists of movements and aircraft noise exposure data and Christian Schindler for his insights on statistical methods.

Funding

This work was supported by the Swiss National Science Foundation (grant no. 324730_173330).

Conflict of interest: The authors declare no conflict of interest. All authors report grants from Swiss National Science Foundation, during the conduct of the study. J.-M.W. and B.S. declare doing calculations for and providing maps to Zürich Airport, outside the submitted work.

References

- 1 European Environment Agency (EEA). *Environmental Noise in Europe (2020)*. EEA, Copenhagen, Denmark; 2020.
- 2 WHO, Regional Office for Europe. *Environmental Noise Guidelines for the European Region*. Copenhagen, Denmark: World Health Organization; 2018.
[Google Scholar](#)
- 3 Van Kempen E Casas M Pershagen G Foraster M. WHO environmental noise guidelines for the European Region: a systematic review on environmental noise and cardiovascular and metabolic effects: a summary. *Int J Environ Res Public Health* 2018;**15**:379.
[Google Scholar](#)
- 4 Vienneau D Eze IC Probst-Hensch N Rösli M. Association between transportation noise and cardio-metabolic diseases: an update of the WHO meta-analysis. 2019.
- 5 Weihofen VM Hegewald J Euler U Schlattmann P Zeeb H Seidler A. Aircraft noise and the risk of stroke: a systematic review and meta-analysis. *Dtsch Arztebl Int* 2019;**116**:237–244.
[Google Scholar](#) [PubMed](#)
- 6 Pyko A Andersson N Eriksson C de Faire U Lind T Mitkovskaya N Ögren M Östenson C-G Pedersen NL Rizzuto D Wallas AK Pershagen G. Long-term transportation noise exposure and incidence of ischaemic heart disease and stroke: a cohort study. *Occup Environ Med* 2019;**76**:201–207.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 7 Dimakopoulou K Koutentakis K Papageorgiou I Kasdagli MI Haralabidis AS Sourtzi P Samoli E Houthuijs D Swart W Hansell AL Katsouyanni K. Is aircraft noise exposure associated with cardiovascular disease and hypertension? Results from a cohort study in Athens, Greece. *Occup Environ Med* 2017;**74**:830–837.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 8 Héritier H Vienneau D Foraster M Eze IC Schaffner E de Hoogh K Thiesse L Rudzik F Habermacher M Köpfler M Pieren R Brink M Cajochen C Wunderli JM Probst-Hensch N Rösli M. A systematic analysis of mutual effects of transportation noise and air pollution exposure on myocardial infarction mortality: a nationwide cohort study in Switzerland. *Eur Heart J* 2019;**40**:598–603.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 9 Seidler A Wagner M Schubert M Dröge P Römer K Pons-Kühnemann J Swart E Zeeb H Hegewald J. Aircraft, road and railway traffic noise as risk factors for heart failure and hypertensive heart disease—a case-control study based on secondary data. *Int J Hyg Environ Health* 2016;**219**:749–758.
[Google Scholar](#) [Crossref](#) [PubMed](#)

- 10 Münzel T Kröller-Schön S Oelze M Gori T Schmidt FP Steven S Hahad O Rössli M Wunderli J-M Daiber A Sørensen M. Adverse cardiovascular effects of traffic noise with a focus on nighttime noise and the new WHO noise guidelines. *Annu Rev Public Health* 2020;41:309–328.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 11 Héritier H Vienneau D Foraster M Eze IC Schaffner E Thiesse L Ruzdik F Habermacher M Köpfl M Pieren R Schmidt-Trucksäss A Brink M Cajochen C Wunderli JM Probst-Hensch N Rössli M. Diurnal variability of transportation noise exposure and cardiovascular mortality: a nationwide cohort study from Switzerland. *Int J Hyg Environ Health* 2018;221:556–563.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 12 Jarup L Babisch W Houthuijs D Pershagen G Katsouyanni K Cadum E Dudley M-L Savigny P Seiffert I Swart W Breugelmans O Bluhm G Selander J Haralabidis A Dimakopoulou K Sourtzi P Velonakis M Vigna-Taglianti F. Hypertension and exposure to noise near airports: the HYENA study. *Environ Health Perspect* 2008;116:329–333.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 13 Vienneau D Schindler C Perez L Probst-Hensch N Rössli M. The relationship between transportation noise exposure and ischemic heart disease: a meta-analysis. *Environ Res* 2015;138:372–380.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 14 Schmidt FP Basner M Kroger G Weck S Schnorbus B Muttray A Sariyar M Binder H Gori T Warnholtz A Munzel T. Effect of nighttime aircraft noise exposure on endothelial function and stress hormone release in healthy adults. *Eur Heart J* 2013;34:3508–314a.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 15 Haralabidis AS Dimakopoulou K Vigna-Taglianti F Giampaolo M Borgini A Dudley ML Pershagen G Bluhm G Houthuijs D Babisch W Velonakis M Katsouyanni K Jarup L Consortium H ; for the HYENA Consortium. Acute effects of night-time noise exposure on blood pressure in populations living near airports. *Eur Heart J* 2008;29:658–664.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 16 Munzel T, Gori T, Babisch W, Basner M. Cardiovascular effects of environmental noise exposure. *Eur Heart J* 2014;35:829–836.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 17 Rössli M Brink M Ruzdik F Cajochen C Ragettli MS Flückiger B Pieren R Vienneau D Wunderli J-M. Associations of various nighttime noise exposure indicators with objective sleep efficiency and self-reported sleep quality: a field study. *Int J Environ Res Public Health* 2019;16:3790.
[Google Scholar](#)
- 18 Basner M Müller U Elmenhorst E-M Kluge G Griefahn B. Aircraft noise effects on sleep: a systematic comparison of EEG awakenings and automatically detected cardiac activations. *Physiol Meas* 2008;29:1089–1103.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 19 Janes H Sheppard L Lumley T. Case-crossover analyses of air pollution exposure data: referent selection strategies and their implications for bias. *Epidemiology* 2005;16:717–726.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 20 Maclure M Mittleman MA. Should we use a case-crossover design? *Annu Rev Public Health* 2000;21:193–221.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 21 Carracedo-Martínez E Taracido M Tobias A Saez M Figueiras A. Case-crossover analysis of air pollution health effects: a systematic review of methodology and application. *Environ Health Perspect* 2010;118:1173–1182.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 22 Nawrot TS Perez L Künzli N Munters E Nemery B. Public health importance of triggers of myocardial infarction: a comparative risk assessment. *Lancet* 2011;377:732–740.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 23 Spoerri A Zwahlen M Egger M Bopp M. The Swiss National Cohort: a unique database for national and international researchers. *Int J Public Health* 2010;55:239–242.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 24 Panczak R Galobardes B Voorpostel M Spoerri A Zwahlen M Egger M ; for the Swiss National Cohort and the Swiss Household Panel. A Swiss neighbourhood index of socioeconomic position: development and association with mortality. *J Epidemiol Community Health*

2012;66:1129–1136.

[Google Scholar](#) [Crossref](#) [PubMed](#)

- 25 Schäffer B Thomann G Huber P Brink M Plüss S Hofmann R. Zurich aircraft noise index: an index for the assessment and analysis of the effects of aircraft noise on the population. *Acta Acust United Acust* 2012;98:505–519.
[Google Scholar](#) [Crossref](#)
- 26 Saucy A Schäffer B Tangermann L Vienneau D Wunderli J-M Rössli M. Individual aircraft noise exposure assessment for a case-crossover study in Switzerland. *Int J Environ Res Public Health* 2020;17:3011.
[Google Scholar](#) [Crossref](#)
- 27 Pietrzko S Bütikofer R Plüss S Thomann G, FLULA2, Ein Verfahren zur Berechnung und Darstellung der Fluglärmbelastung. In: *Technische Programm-Dokumentation. Version 4*. Swiss Federal Laboratories for Materials Science and Technology (Empa), Dübendorf, Switzerland; 2010.
[Google Scholar](#)
- 28 Karipidis I Vienneau D Habermacher M Köpfl M Brink M Probst-Hensch N Rössli M Wunderli J-M. Reconstruction of historical noise exposure data for environmental epidemiology in Switzerland within the SiRENE project. *Noise Mapping* 2014;1.
[Google Scholar](#)
- 29 De Hoogh K Saucy A Shtein A Schwartz J West E A Strassmann A Puhan M Rössli M Stafoggia M Kloog I. Predicting Fine-Scale Daily NO₂ for 2005–2016 Incorporating OMI Satellite Data Across Switzerland. *Environmental Science & Technology* 2019;53:10279–10287.
[Google Scholar](#)
- 30 Federal Office of Meteorology and Climatology. *Daily Precipitation and Temperature*. MeteoSwiss, Bern, Switzerland; 2017.
- 31 Navidi W. Bidirectional case-crossover designs for exposures with time trends. *J Biometrics* 1998;54:596–605.
[Google Scholar](#) [Crossref](#)
- 32 Patino CM Ferreira JC. Test for trend: evaluating dose-response effects in association studies. *J Bras Pneumol* 2016;42:240–240.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 33 Perez L Kunzli N. From measures of effects to measures of potential impact. *Int J Public Health* 2009;54:45–48.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 34 Correia AW Peters JL Levy JI Melly S Dominici F. Residential exposure to aircraft noise and hospital admissions for cardiovascular diseases: multi-airport retrospective study. *BMJ* 2013;347:f5561–f5561.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 35 Héritier H Vienneau D Foraster M Eze IC Schaffner E Thiesse L Rudzik F Habermacher M Köpfl M Pieren R Brink M Cajochen C Wunderli JM Probst-Hensch N Rössli M; for the S.N.C. study group. Transportation noise exposure and cardiovascular mortality: a nationwide cohort study from Switzerland. *Eur J Epidemiol* 2017;32:307–315.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 36 Hansell AL Blangiardo M Fortunato L Floud S de Hoogh K Fecht D Ghosh RE Laszlo HE Pearson C Beale L Beevers S Gulliver J Best N Richardson S Elliott P. Aircraft noise and cardiovascular disease near Heathrow airport in London: small area study. *BMJ* 2013;347:f5432–f5432.
[Google Scholar](#) [Crossref](#) [PubMed](#)
- 37 Locher B Piquerez A Habermacher M Ragetti M Rössli M Brink M Cajochen C Vienneau D Foraster M Müller U Wunderli J. Differences between outdoor and indoor sound levels for open, tilted, and closed windows. *Int J Environ Res Public Health* 2018;15:149.
[Google Scholar](#) [Crossref](#)
- 38 World Health Organization. *Environment and Health Risks: A Review of the Influence and Effects of Social Inequalities*. WHO Regional Office for Europe, Copenhagen, Denmark; 2010.
- 39 Dudas K Lappas G Stewart S Rosengren A. Trends in out-of-hospital deaths due to coronary heart disease in Sweden (1991 to 2006). *Circulation* 2011;123:46–52.
[Google Scholar](#) [Crossref](#) [PubMed](#)

Griefahn B Marks A Robens S. Experiments on the time frame of temporally limited traffic curfews to prevent noise induced sleep disturbances. *Somnologie* 2008;12:140–148.

[Google Scholar](#) [Crossref](#)

© The Author(s) 2020. Published by Oxford University Press on behalf of the European Society of Cardiology.

Dies ist ein Open Access-Artikel, der unter den Bedingungen der Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/4.0/>) verbreitet wird und die nichtkommerzielle Wiederverwendung, Verteilung und Reproduktion in jedem Medium, sofern das Originalwerk ordnungsgemäß zitiert wird. Für die kommerzielle Wiederverwendung wenden Sie sich bitte an journals.permissions@oup.com

Zusatzdaten

[ehaa957_Supplementary_Data](#) - PDF-Datei