Vascular diseases and night-time aircraft noise – the Cologne-Bonn Airport Study

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ABSTRACT

A retrospective case-control study was conducted to investigate the impact of aircraft noise on cardiovascular and vascular diseases in the vicinity of a Cologne-Bonn International Airport, an aiport with unlimited night-time air traffic. The study region comprised the City of Cologne, and two counties adjacent to the airport. Residency-specific environmental noise data and data from 8 compulsory sickness funds (531.172 persons, aged 40 years and older, covering 53.4% of the population of the study region) were used including data on prescriptions of therapeutic drugs and hospital discharge diagnoses. Target diseases were a combination of specific cardiovascular diseases (myocardial infarction(MI), coronary heart disease (CHD), heart failure, stroke), MI, CHD, stroke, atherosclerosis of peripheral arteries, dementia, and kidney failure. There were dose-dependent risk increases for all diagnostic entities in persons not entitled to reimbursement for noise protection, indicating a protective effect of noise protection measures. ORs larger than 2.0 were detected in dementia and in chronic kidney failure. In general, this study demonstrated decreasing risks with increasing age. Risk increases in all analyses were larger in women than in men.

INTRODUCTION

Hypertension has been linked to aircraft noise in numerous publications since 1976 [1, 2, 3, 4, 5, 6, 7, 8, 9, 10]. Hypertension has been ascertained as self-reported [2, 3, 4], as determined by a standardised epidemiological measurement [2, 7, 8, 9, 10], or by the prescription of antihypertensive drugs [5, 6]. In addition, a multi-centre study in several European countries indicated a correlation between aircraft noise and increased blood pressure in elementary school children [11]. The scientific evidence linking aircraft noise, especially during the night, with hypertension seems to be sufficient to discuss a causal relationship.

In addition there are several publications linking aircraft noise to cardiovascular diseases. One publication based on self-reported items linked medical treatment for cardiac symptoms as well as the intake of cardiac drugs and a “pathological heart shape” to aircraft noise [2]. Swiss epidemiologists were able to link the Swiss national death index individually to the data of the 2000 population census and to address-specific aircraft noise data [12]. This study demonstrated an increasing risk of myocardial death with increasing aircraft noise. This effect was more pronounced in deceased persons who had lived for longer periods at the same address. However, there were no significant effects for either stroke mortality or coronary heart disease mortality. In a prolonged follow-up of the Swiss national cohort these results could be confirmed [13]. In a follow-up of 4,712 participants of the HYENA study from 2004 to 2006, 24-hour aircraft noise was linked to the occurrence of heart disease and stroke combined [14]. The observed effect had a significantly higher in a subset of participants with more than 19 years of residence at the same address. Two recent large studies demonstrated the effects of aircraft noise on cardiovascular endpoints. Hansell and co-authors [15] analysed the impact of day-time and night-time aircraft noise in a population of more than 3.5 million people living in the vicinity of Heathrow Airport. They used National Health Service hospital discharge data provided for census tracts with an average of 297 persons and census super tracts with an average of 1,510 persons for the mortality analyses. When comparing regions with low day-time aircraft noise exposure (< 51 dB(A)) to regions with high exposure (> 63 dB(A)), they found increased ORs for stroke and coronary heart disease and for a combination of cardiovascular diseases. Similar effects were found for the respective mortality rates. No relevant differences could be established between day-time and night-time aircraft noise effects. Correia and co-authors [16] analysed the effects of day-night sound levels (L_{dn}) in Medicare insurees (65 years and older) living in the vicinity of 89 US airports (study population 6 million people). They used Medicare hospital discharge diagnoses and respective noise data on the post-code level of aggregation, where on average, a post-code comprised 168 census blocks with several hundred persons each. They found a significant increase for all cardiovascular discharge diagnoses combined, leading to an estimated attributable fraction of 2.3% of all cardiovascular discharge diagnoses. Seidler and co-authors [17] investigated the impact of aircraft noise in the vicinity of Frankfurt-Main airport, based oninsurees of three major compulsory sickness funds. The results demonstrated a significant linear exposure-risk relationship with heart failure or hypertensive heart disease.

Of all publications on aircraft noise and cardiovascular diseases, only three [6, 9, 16] used night-time aircraft noise as a risk factor, whereas all others used some type of 24-hour energy-weighted aircraft noise parameter with penalties for night-time aircraft noise and, in some instances, aircraft noise during evening periods.

The objective of our study was to investigate the impact of aircraft noise, especially of night-time aircraft noise, on cardiovascular and psychiatric diseases.

METHODS

We conducted a retrospective case-control study on the impact of night-time aircraft noise on vascular diseases and on psychiatric illnesses in the vicinity of the Cologne-Bonn Airport, based on hospital discharge diagnoses for vascular diseases (specific major cardiovascular diseases, stroke, dementia, and chronic kidney failure) and out-patient prescriptions of therapeutic drugs of persons insured by eight compulsory sickness funds. In this publication we restrict our report on vascular diseases. For the extraction of the respective discharge diagnoses from the sickness fund data, ICD-9 and ICD-10 codes were used (Table 1). We

decided to include codes for Alzheimer's disease in the definition of dementia cases because a cross-tabulation of discharge diagnoses for vascular dementia by diagnoses of Alzheimer's disease during the course of the insurance periods displayed major overlaps: in 55% of the diagnoses of Alzheimer's disease, there were concurrent diagnoses of vascular dementia. The Cologne-Bonn Airport has unlimited night-time aircraft traffic rights, which are mostly used for air cargo flights. We used residency-specific A weighted equivalent (Aeq) sound pressure levels (L), denoted as L_{Aeq}, for the time period of 11 p.m. to 1 a.m., as this period of the night rendered the highest excess risk for all of the diagnostic entities analysed (data not shown).

Study area and population

The study region includes the city of Cologne and two counties adjacent to the airport (Rhein-Sieg-Kreis and Rheinisch-Bergischer Kreis), with a total population of 1,840,908 (average population of 2004). Eight compulsory sickness funds contributed individual data on both out-patient prescriptions of therapeutic drugs and the hospital discharge diagnoses of 1,081,446 persons (= 53.4% of the total population of the study region), resulting in a total of 3,495,146 person-years of data. The privacy commissioners of all sickness funds approved the design of the study before data were provided. For the analyses of this publication, the data of persons older than 39 years were used (531,172 persons, corresponding to 1,983,357 person-years).

Environmental noise data

Aircraft noise data were calculated based on aircraft movements from 6 months of 2004, with most of the air traffic using the official German procedure AzB 99 [18]. Calculations were performed for day-time traffic (6 a.m. – 10 p.m.) and night-time traffic (10 p.m.-6 a.m.) and for two time periods within the night with the most traffic (11 p.m.-1 a.m., 3 - 5 a.m.) for LAeq from 40 dB(A). Road and railroad traffic noise data were provided by the Northrhine-Westfalian Environmental Office [19], comprised of day-time and night-time LAeq from 40 dB(A) upwards. To derive residency-specific noise data, terrain contours and residency profiles were used. The results of these calculations were linked to all of the 376,223 residential addresses within our study region using their respective Gauß-Krüger coordinates.

Health data

For each of the 1,081,646 insured person with a main residence within the study region, the sickness funds provided anonymous data on insurance period, age, gender, out-patient prescriptions of medication and date of prescriptions, up to six discharge diagnoses per hospital admission, and the calendar dates of the hospital stay. Of these, all persons aged 40 and older (N = 531,172) were included in the analyses. All of the sickness funds were provided with a database containing 376,223 residential addresses of the study region and a character string containing all environmental noise data and region-specific confounder data (percentage of persons receiving social welfare support per community borough and percentage of nursing beds per population aged 65 and over within communities) and data on the possibility for reimbursement of costs for bedroom window noise protection. The sickness funds linked these data to the addresses of insured persons and returned the character string with an anonymous identifier to link to other data of the same person.

Definition of disease outcomes

The disease entities used for the analyses included cardiovascular diseases (coronary heart disease, myocardial infarction, stroke, and a combination of myocardial infarction, heart failure, coronary heart disease, stroke), dementia and chronic kidney failure, as coded according to ICD-9 and ICD-10 (Table 1).

Data on potential confounders

Specific out-patient prescriptions were included into the analyses as proxy information for cardiovascular risk factors: antihypertensive drugs were used as a proxy for arterial hypertension, other cardiac drugs for other conditions leading to cardiovascular diseases, lipid lowering drugs for hyperlipidemia, insulin and oral anti-diabetic agents for diabetes mellitus, antidepressants for depression. Depression has been implicated as a potential risk factor for coronary heart disease [20]. We used region-specific percentages of persons on social welfare support as a proxy for social inequality. As it was deemed possible that nursing homes with an increased prevalence of cardiovascular diseases could lead to biased estimates, the percentage of nursing beds per community population aged 65 and over was included as a potential confounder in all analyses. As the preliminary analyses showed a strong effect of age on increasing disease risk, the interaction term age*L_{Aeq} was included.

Statistical analyses

Multivariate logistic analyses were conducted using the SAS version 9.2 procedure Proc Phreg [21] exclusively. Cases were defined as persons with at least one of the respective hospital discharge diagnoses during the insurance period. Controls were defined as persons without the respective discharge diagnosis. All environmental risk factors were treated as continuous variables. To calculate the effect of interactions (age*aircraft noise), the procedure proposed by Hosmer and Lemeshow [22] was used. We defined the dependent variables to be all cardiovascular diseases combined, coronary heart disease, myocardial infarction, heart failure, stroke, and a combination of only the latter four, peripheral artery disease, dementia, and kidney failure. The latter two diagnostic entities were included because hypertension acts as a major risk factor for both. ICD-10 as well as ICD-9 codes for these diagnoses (Table 1) were extracted from sickness fund records. As the analyses of the regional aircraft noise distribution for the four time periods showed that the regions covered were not completely identical , the following procedure was applied to prohibit the dilution of effects; e.g., when L_{Aeq} values of the night-time 11 p.m.-1 a.m. period were below 40, cases and controls were omitted from the analyses if the L_{Aeq} values of other time windows were above 39 dB(A). As the analyses on all four time windows of aircraft noise indicated that risk increases were most pronounced for night-time aircraft noise from 11 p.m. to 1 a.m., the results from only these analyses are presented. The risk effects were calculated as the risk increase per 10 dB(A) increase of L_{Aeq}. The Cologne-Bonn Airport administration provided addresses where inhabitants were entitled to the reimbursement of costs for noise-protection windows in bedrooms. We used this indicator to differentiate between the part of the population entitled to reimbursement and that without such a possibility.

RESULTS

Exposure to aircraft noise

Less than 20% of the study population was exposed to night-time aircraft noise , and 1.4% (N=4,011 men and 3,115 women) were exposed to noise levels above 54 dB(A). Of the total population, 5.7% were entitled to reimbursement for noise protection of bedroom windows (N=29,795, aged 40 and older,(Figure 1). Regional distribution of aircraft noise is shown in Figure 2.

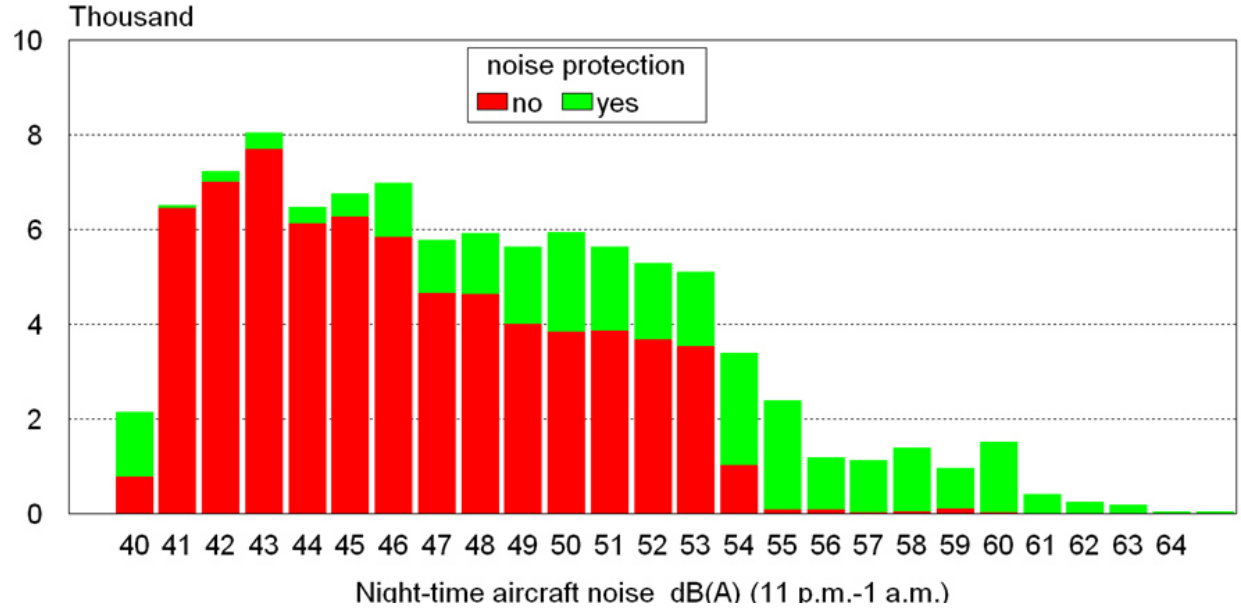

Figure 1. Persons entitled to reimbursement for noise protection of bedroom windows.

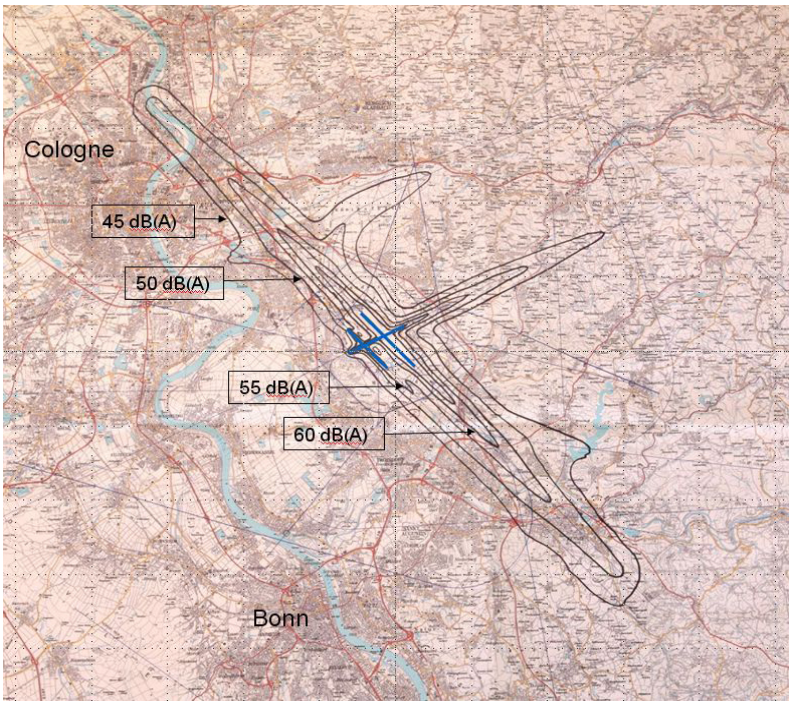

Figure 2. Isophones of night-time aircraft noise at Cologne-Bonn Airport

Disease risks

In all of the diseases under investigation, the prevalences were considerably smaller in women than in men (Table 1). The distribution of confounding factors showed only moderately lower prevalences in women (data not shown). However, the odds ratios (ORs) in women were in the same range or higher as in men, except for stroke and for dementia (Table 2). In all of the diagnostic entities investigated, there was a marked decrease of excess risks with increasing age. Maximum risk increases with odds ratios above 1.7 were found in both dementia as in chronic kidney failure.

When comparing odds ratios for total populations and sub-populations with and without the financing of noise protection for bedroom windows, a considerable difference emerged: in all diagnostic entities, the ORs derived for the sub-population without such financing were larger than in the other group whenever the ORs were statistically significant. None of the odds ratios for the subgroup of persons entitled to reimbursement for noise protection of bedroom windows were statistically significant.

Table 1. Baseline characteristics of the study population

Population

Age	**Total**	**< 40**	**40-49**	**50-59**	**60-69**	**70-79**	**80-89**
Persons	1 081 446	550 274	158 071	114 813	124 076	78 964	41 172
Person years	3 495 146	1 511 789	510 749	400 784	49 2036	359 863	174 070
% females	44.6	43.8	48.4	48.5	46.9	36.9	41.4

Prevalence of diseases (%)

			Age groups (% of respective population)					
Diagnoses	**Gender**	**All ages**	**< 40**	**40-49**	**50-59**	**60-69**	**70-79**	**80-89**
Specific cardiovasc. diagnoses*	Men	40 309 (5.3)	675 (0.2)	1 814 (1.6)	4 035 (5.2)	9 582 (11.0)	12 860 (22.1)	8 448 (31.2)
	Women	17 317 (3.6)	301 (0.1)	766 (1.0)	1 976 (3.6)	4 042 (6.9)	4 597 (15.8)	4 389 (25.8)
CHD[1]	Men	31 719 (4.1)	420 (0.1)	1 411 (1.3)	3 250 (4.1)	7 834 (8.9)	10 286 (17.4)	6 461 (23.0)
	Women	12 550 (2.6)	132 (0.1)	498 (0.7)	1 504 (2.7)	3 158 (5.4)	3 445 (11.8)	3 025 (17.8)
Myocardial infarction[2]	Men	9 620 (1.1)	105 (<0.1)	(0.4)	1 136 (1.2)	2 486 (2.5)	3 121 (5.0)	1 806 (6.3)
	Women	2 806 (0.6)	29 (<0.1)	118 (0.2)	270 (0.5)	602 (1.0)	802 (2.7)	794 (4.7)
Stroke[3]	Men	11 60 (1.8)	202 (0.1)	357 (0.4)	926 (1.2)	2 526 (2.9)	4 213 (7.3)	3 379 (13.0)
	Women	5 505 (1.3)	141 (0.1)	254 (0.3)	495 (0.9)	1 089 (1.9)	1 548 (5.3)	1 978 (11.6)

Dementia[4]	Men	6 453 (0.9)	9 (<0.1)	43 (<0.1)	106 (0.1)	410 (0.5)	1 678 (3.0)	2 680 (10.4)
	Women	3 252 (0.7)	13 (<0.1)	13 (<0.1)	33 (0.1)	171 (0.3)	695 (2.4)	1 599 (9.4)
Chronic kidney failure[5]	Men	9 647 (1.6)	291 (0.1)	286 (0.4)	569 (1.0)	1 728 (2.6)	3 177 (6.4)	2 634 (10.9)
	Women	3 727 (0.8)	170 (0.1)	147 (0.2)	241 (0.4)	638 (1.1)	912 (3.1)	1 232 (7.2)

* =Coronary heart disease (CHD), myocardial infarction, heart failure, stroke

[1] ICD-10: I20, I24, I25.0-I25.5, I25.8, I25.9 / ICD-9: 411, 413 - [2] ICD10: I21-I24, I25.2 / ICD-9: 410, 413

[3] ICD-10: I60, I61, I63-I67, I69 / ICD-9: 431-436 - [4] ICD-10: F00-F03, G30 / ICD-9: 290, 331.0, 331.2

[5] ICD-10: N17-N19 / ICD-9: 583, 584.6-584.9, 585-587, 590.8, 593.9

Table 2. Increase of risk per 10 dB(A) increase of night-time aircraft noise (above 39 dB(A))
(Odds ratio & 95% confidence intervals; significant odds ratios in **bold**)

	Specific cardiovascular diseases*			**Myocardial infarction**			**Coronary heart disease**		
Age	All	Males	Females	All	Males	Females	All	Males	Females
40	**1.22 (1.14-1.31)**	**1.16 (1.07-1.26)**	**1.27 (1.11-1.44)**	**1.37 (1.16-1.63)**	**1.25 (1.03-1.51)**	**1.60 (1.09-2.36)**	**1.30 (1.18-1.43)**	**1.22 (1.09-1.36)**	**1.33 (1.11-1.59)**
44	**1.20 (1.11-1.28)**	**1.14 (1.05-1.24)**	**1.23 (1.08-1.41)**	**1.31 (1.11-1.55)**	**1.20 (1.00-1.45)**	**1.50 (1.02-2.21)**	**1.25 (1.14-1.38)**	**1.19 (1.06-1.33)**	**1.29 (1.08-1.53)**
50	**1.16 (1.08-1.24)**	**1.11 (1.03-1.21)**	**1.18 (1.04-1.35)**	**1.22 (1.03-1.44)**	1.14 (0.95-1.37)	1.37 (0.94-2.00)	**1.19 (1.09-1.31)**	**1.14 (1.02-1.28)**	**1.22 (1.03-1.45)**
54	**1.13 (1.05-1.21)**	**1.10 (1.01-1.19)**	**1.15 (1.02-1.31)**	1.16 (0.98-1.37)	1.10 (0.92-1.32)	1.29 (0.89-1.87)	**1.15 (1.05-1.26)**	**1.11 (1.00-1.24)**	1.18 (0.99-1.40)
60	**1.09 (1.02-1.17)**	1.07 (0.99-1.16)	1.11 (0.98-1.26)	1.08 (0.92-1.27)	1.04 (0.87-1.25)	1.17 (0.81-1.69)	**1.09 (1.00-1.20)**	1.07 (0.96-1.19)	1.12 (0.95-1.32)
64	**1.07 (1.00-1.14)**	1.05 (0.97-1.14)	1.08 (0.95-1.22)	1.03 (0.87-1.20)	1.01 (0.84-1.20)	1.10 (0.77-1.58)	1.06 (0.97-1.16)	1.04 (0.94-1.16)	1.08 (0.91-1.28)
70	1.03 (0.97-1.10)	1.03 (0.95-1.11)	1.04 (0.92-1.17)	0.95 (0.82-1.11)	0.95 (0.80-1.14)	1.01 (0.70-1.43)	1.00 (0.92-1.10)	1.01 (0.91-1.11)	1.03 (0.87-1.21)
74	1.01 (0.95-1.08)	1.01 (0.94-1.09)	1.01 (0.90-1.14)	0.91 (0.78-1.06)	0.92 (0.77-1.09)	0.94 (0.66-1.34)	0.97 (0.89-1.06)	0.98 (0.88-1.09)	0.99 (0.84-1.17)
80	0.98 (0.91-1.04)	0.99 (0.92-1.07)	0.97 (0.86-1.09)	0.84 (0.73-0.98)	0.87 (0.74-1.03)	0.86 (0.61-1.22)	0.92 (0.85-1.00)	0.94 (0.85-1.04)	0.94 (0.80-1.10)
84	0.95 (0.89-1.02)	0.97 (0.90-1.05)	0.95 (0.84-1.06)	0.80 (0.69-0.93)	0.84 (0.71-0.99)	0.81 (0.58-1.14)	0.89 (0.82-0.97)	0.92 (0.83-1.01)	0.91 (0.78-1.06)

* Myocardial infarction, heart failure, coronary heart disease, stroke

Table 2. continued. Increase of risk per 10 dB(A) increase of night-time aircraft noise (above 39 dB(A))
(Odds ratio & 95% confidence intervals; significant odds ratios in **bold**)

	Stroke			**Dementia**			**Chronic kidney failure**		
Age	All	Males	Females	All	Males	Females	All	Males	Females
40	**1.31 (1.11-1.56)**	**1.25 (1.01-1.53)**	1.31 (0.96-1.77)	**1.75 (1.37-2.23)**	**1.74 (1.31-2.31)**	**1.65 (1.05-2.61)**	**1.76 (1.22-2.53)**	**1.27 (1.02-1.58)**	**1.64 (1.16-2.31)**
44	**1.28 (1.08-1.51)**	**1.22 (1.00-1.50)**	1.26 (0.93-1.70)	**1.70 (1.34-2.16)**	**1.69 (1.28-2.25)**	**1.61 (1.02-2.53)**	**1.72 (1.20-2.47)**	**1.25 (1.01-1.55)**	**1.58 (1.12-2.22)**
50	**1.22 (1.03-1.44)**	1.19 (0.98-1.46)	1.19 (0.89-1.60)	**1.62 (1.28-2.06)**	**1.63 (1.23-2.15)**	1.54 (0.99-2.41)	**1.66 (1.16-2.37)**	1.22 (0.99-1.51)	**1.49 (1.06-2.09)**
54	**1.19 (1.01-1.40)**	1.17 (0.96-1.43)	1.15 (0.86-1.54)	**1.58 (1.25-1.99)**	**1.58 (1.20-2.09)**	1.50 (0.96-2.34)	**1.62 (1.13-2.31)**	1.21 (0.98-1.49)	**1.43 (1.03-2.00)**
60	1.14 (0.97-1.33)	1.14 (0.94-1.39)	1.09 (0.81-1.45)	**1.51 (1.20-1.90)**	**1.52 (1.16-2.00)**	1.44 (0.93-2.24)	**1.56 (1.10-2.22)**	1.18 (0.96-1.45)	1.35 (0.97-1.88)
64	1.10 (0.94-1.29)	1.12 (0.93-1.36)	1.05 (0.79-1.39)	**1.46 (1.16-1.84)**	**1.48 (1.13-1.94)**	1.41 (0.91-2.17)	**1.52 (1.07-2.17)**	1.17 (0.95-1.43)	1.30 (0.94-1.80)
70	1.06 (0.90-1.24)	1.09 (0.90-1.32)	0.99 (0.75-1.31)	**1.40 (1.12-1.76)**	**1.42 (1.09-1.85)**	1.35 (0.88-2.07)	**1.47 (1.04-2.08)**	1.14 (0.93-1.40)	1.23 (0.89-1.70)
74	1.03 (0.88-1.20)	1.08 (0.89-1.30)	0.95 (0.72-1.26)	**1.36 (1.09-1.70)**	**1.38 (1.06-1.80)**	1.31 (0.86-2.01)	**1.43 (1.01-2.03)**	1.13 (0.92-1.37)	1.18 (0.86-1.63)
80	0.98 (0.84-1.14)	1.05 (0.87-1.26)	0.90 (0.69-1.19)	**1.30 (1.04-1.62)**	**1.33 (1.02-1.72)**	1.26 (0.83-1.92)	1.38 (0.98-1.95)	1.10 (0.91-1.34)	1.12 (0.82-1.53)
84	0.95 (0.82-1.11)	1.03 (0.86-1.24)	0.87 (0.67-1.14)	**1.26 (1.01-1.57)**	**1.29 (1.00-1.67)**	1.23 (0.81-1.86)	1.35 (0.96-1.90)	1.09 (0.90-1.32)	1.08 (0.79-1.47)

DISCUSSION

Strengths and weaknesses of the study

Our study is based on individual data on exposure to environmental noise, on individual hospital discharge diagnoses, and on individual data on important confounders, including potentially preventive measures. Moreover, the database is sufficiently large to allow for detailed analyses by gender. To our knowledge, our study is the first to show a declining risk increase with increasing age, more so in men than in women. A major weakness of our database is the lack of information on the duration of exposure to environmental noise. In addition, we did not have the possibility to determine the exact incidence of cardiovascular diseases. However, we did calculate incidences based on 12- and 24-month latency periods (data not shown). As we could not preclude that e.g., in the case of myocardial infarction events that occurred prior to the insurance period of individuals reported by the sickness funds, we refrained from conducting logistic regressions with incidences as dependent variables. In addition, our aircraft noise exposure data are based on ideal aircraft routes. In practice, aircraft guides allow pilots upon request to diverge from those routes, primarily because such divergences save aircraft fuel. This divergence, in consequence, leads to exposure of a larger portion of the population than calculated and hence to an underestimation of risk increases.

General discussion

The markedly lower ORs in the subpopulation with reimbursement for noise reduction windows indicate a potential preventive effect. None of the other studies published thus far included noise prevention measures in their analyses. As it must be assumed that not all of the people who are entitled to reimbursement for noise-protection windows in bedrooms will apply for it and that some people who are not entitled may obtain such windows at their own cost, the ORs found in our study may underestimate the preventive effects of noise protection. The declining ORs with increasing age, which we found in all of our analyses, can possibly be explained by the increase of hearing loss with increasing age. In a study on aircraft noise, Rosenlund and co-authors [3] found markedly lower ORs in men and women with hearing loss compared to persons without hearing loss, but the analyses in dichotomised age groups (<=55 vs. 56+) hinted at slightly larger ORs for older people. In a German study on the effects of night-time road traffic noise on hypertension [23] ORs in persons older than 60 years were much lower and not significant compared to ORs in younger persons aged 20-60. The first publication on aircraft noise and subsequent cardiovascular diseases [2] presented declining risks for “taking cardiovascular drugs” and for “medical treatment for heart trouble” with increasing age; this study reported statistically significant risks in women but not in men. Several publications [24, 25, 26] have shown an increase in hearing loss with increasing age and a considerably larger proportion of males with hearing loss than females. This observation might be an explanation for both our findings of decreasing ORs with increasing age and of larger ORs in women belonging to the sub-population without reimbursement for noise protection windows in all age groups because overall the prevalence of hearing loss in men appears to be 70% larger than in women. There is ample evidence from numerous studies linking aircraft noise to arterial hypertension. The underlying patho-physiologic mechanism seems to be linked to the hypothalamic-pituitary-adrenal axis, which when stimulated by any type of stress effect or sleep disruption, leads to increased excretion of cortisol and/or adrenalin by the adrenal cortex [27, 28, 29]. This link has been shown to exist in populations exposed to road traffic noise [30] or to aircraft noise [31]. A recent study in patients with or at risk for coronary heart disease exposed to night-time aircraft noise confirmed this mechanism

and added evidence of endothelial damage after only two nights of exposure to aircraft noise [32].

Previous studies

The results of our study ought to be discussed in contrast to recent important studies of comparable magnitude to elucidate results that differ from our results. The Swiss mortality study [12] presented an increased mortality for only myocardial infarction but not for cardiovascular diseases combined or for stroke. A major difference from our study was the determination of noise parameters, which in the Swiss study was not conducted in a residency-specific manner but rather in squares of 200x200 meters. This method possibly presents a bias in exposure parameters, which might lead to an imprecision of effects. The authors of the Heathrow Airport study [15], despite including data on more than 3.5 million persons, could not use individual exposure data but had to resort to census output areas (with an average of 297 persons) for morbidity and to census super output areas (with an average of 1,510 persons) for analyses of mortality. This certainly could lead to imprecise noise calculations. The confounders in this study were regional, but not individual variables. The comparison of the US multi-airport study [16] with our study is hampered by the fact that the US study was restricted to persons aged 65 and older. The areas for which noise data were available were on the ZIP code level, which results in up to several thousand persons with identical noise data. Nevertheless, the results of this study showed a significant increase in hospital admissions for cardiovascular disease linked to night-time aircraft noise but not to day-time aircraft noise. In our study, we found a smaller but also significant risk increase for day-time aircraft noise. However, in our study, the determination of day-time effects was hampered by at least partial night-time exposure of the same population. This prohibits a calculation of “pure” day-time effects. In summary, biases in exposure variables are likely to lead to underestimating the overall effects. The decline of excess risks with increasing age could only be detected when including an appropriate interaction term (age*noise parameter) into the model, which goes a step farther than merely adjusting for age. Gender differences might have been detected when stratifying analyses by gender instead of adjusting for gender.

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