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Aircraft, road and railway traffic noise as risk factors for heart failure and hypertensive heart disease—A case-control study based on secondary data

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ABSTRACT

Background: Several studies point to an elevated risk for cardiovascular diseases induced by traffic noise.
Aims: We examined the association between aircraft, road traffic and railway noise and heart failure or hypertensive heart disease (HHD) in a large case-control study.
Methods: The study population consisted of individuals that were insured by three large statutory health insurance funds in the Rhine-Main area of Germany. Based on insurance claims and prescription data, 104,145 cases of heart failure or HHD diagnosed 2006–10 were identified and compared with 654,172 control subjects. Address-specific exposure to aircraft, road and railway traffic noise in 2005 was estimated. Odds Ratios were calculated using logistic regression analysis, adjusted for age, sex, local proportion of persons receiving unemployment benefits, and individual socioeconomic status (available for 39% of the individuals).
Results: A statistically significant linear exposure-risk relationship with heart failure or hypertensive heart disease was found for aircraft traffic noise (1.6% risk increase per 10 dB increase in the 24-h continuous noise level; 95% CI 0.3–3.0%), road traffic noise (2.4% per 10 dB; 95% CI 1.6–3.2%), and railway noise (3.1% per 10 dB; 95% CI 2.2–4.1%). For individuals with 24-h continuous aircraft noise levels <40 dB and nightly maximum aircraft noise levels exceeding 50 dB six or more times, a significantly increased risk was observed. In general, risks of HHD were considerably higher than the risks of heart failure.
Conclusions: Regarding the high prevalence of traffic noise from various sources, even low risk increases for frequent diseases are relevant for the population as a whole.
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1. Introduction

Various stress reactions can be induced by traffic noise. Activation of the sympathetic system and of the hypothalamus-hypophysis-adrenal axis might account for consecutive cardiovascular diseases. Furthermore, nighttime traffic noise might lead to sleep disturbances (Halonen et al., 2012) which are known as a risk factor for cardiovascular diseases (Badran et al., 2015). According to the WHO, in Western Europe, at least one million disability-adjusted life years (DALY) are attributable to traffic noise-induced diseases (WHO, 2010).

Only few studies have examined the relationship between traffic noise and heart failure or hypertensive heart disease to date. The HYENA study examined the association between aircraft noise and self-reported cardiovascular disease in six European countries (Floud et al., 2013). The authors report a non-significantly elevated risk for heart failure (odds ratio OR = 1.05; 95% confidence interval CI 0.92–1.21), comparable to the risks for ischemic heart disease (OR = 1.06; 95% CI 0.91–1.22) and stroke (OR = 1.08; 95% CI 0.82–1.41). In a cross-sectional study including MRI examinations of 3827 subjects aged 45–84 years (Van Hee et al., 2009), living next to a main street (<50 m distance) was shown to be associated with a reduced left ventricular ejection fraction. However, this study could not differentiate between potential risks of air pollutants and the

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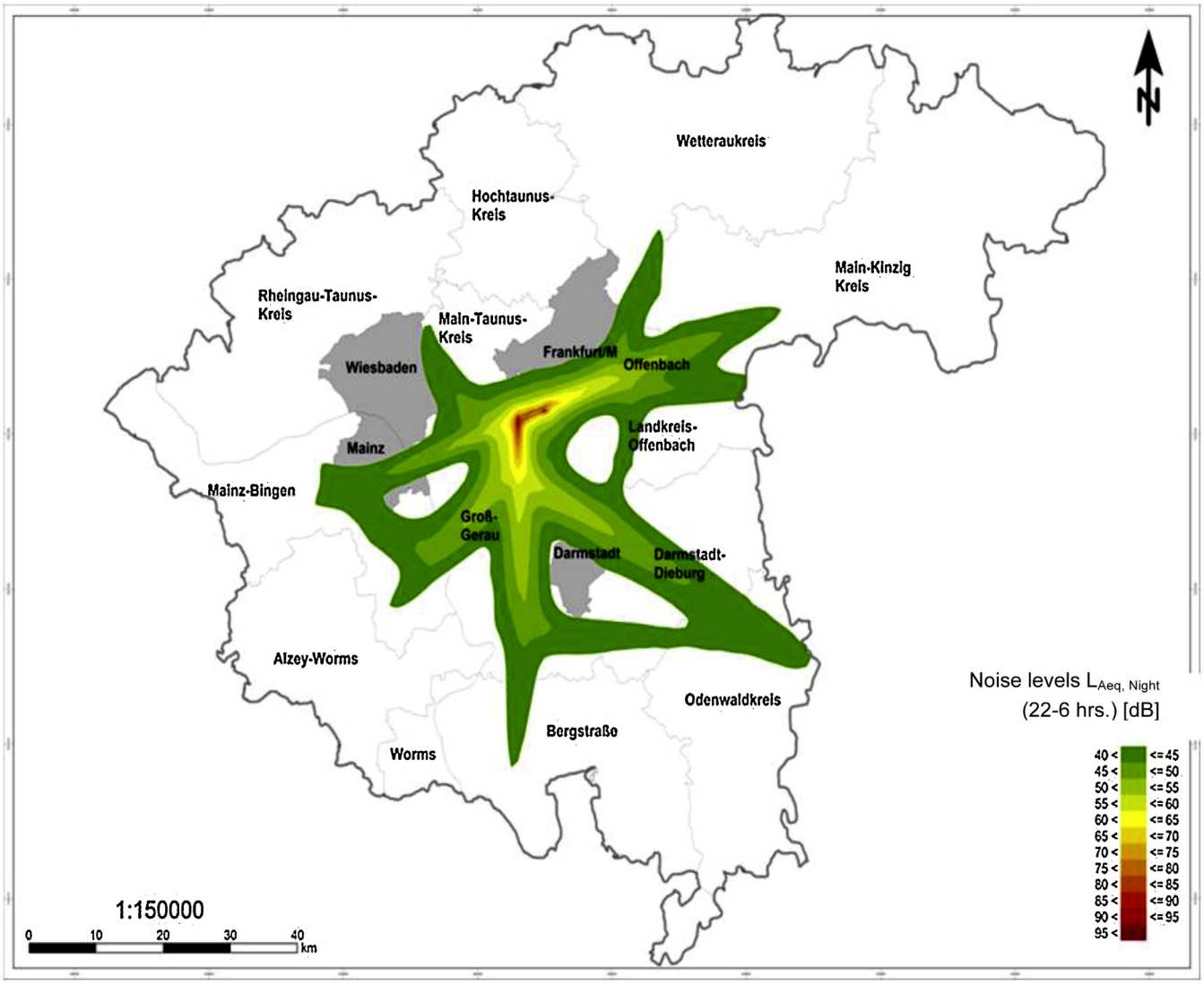

Fig. 1. Map of the study area (grey line), and contours of continuous sound levels for nighttime aircraft noise exposure in 2005. Areas with nighttime (22–06 h) sound levels below 40 dB are shaded white.

risks of road traffic noise. In a Dutch cohort study, Beelen et al. (2009) found a significantly elevated risk for heart failure (relative risk RR = 1.99; 95% CI 1.05–3.79) for a day-evening-night equivalent noise level (L_{DEN}) >65 dB, while the risk elevation for ischemic heart disease (including ischemic stroke) lacked statistical significance. Adjustment for air pollution did not substantially change the risk estimator for heart failure. Correia et al. (2013) conducted a large cross-sectional study in areas surrounding 89 airports in the USA. Hospitalizations for heart failure were non-significantly associated with a 10 dB increase in the 90th centile of noise. In their Canadian cohort study, Gan et al. (2012; Web Appendix, Table 2) revealed a non-significant relative risk of 1.15 (95% CI 0.89–1.48) per 10 dB increase in traffic noise (including road traffic noise, aircraft noise and railway noise). To date, no studies have examined risks for railway noise separately.

The aim of this secondary data-based case-control study is to examine the risk of heart failure or hypertensive heart disease separately for aircraft noise, road traffic noise and railway noise. Heart failure and hypertensive heart disease were combined, as there is large overlap between these two diagnoses. Moreover, hypertensive heart disease constitutes an important precursor of heart failure. An additional questionnaire-based survey was conducted among a sample of participants to evaluate the potential effect of important confounders (such as body mass index, smoking, alcohol consumption, and socioeconomic status) not documented in the health claims data.

2. Methods

2.1. Study population

The study population consisted of 1,026,670 people aged 40 years or more who were insured by three large statutory health insurance funds between 2005 and 2010, and encompassed about 23% of the population aged 40 or above in the study area. The study region was located around the Frankfurt airport and included the administrative region Darmstadt, the cities Mainz and Worms and the administrative districts Mainz-Bingen and Alzey-Worms (see Fig. 1).

2.2. Noise exposure assessment

The exposure to road traffic, railway and aircraft noise was estimated separately for each individual's address. The immis-

sion site was the outer surface of the house front exposed to the highest noise level for road and rail traffic and in the centre of the building for aircraft noise. Average and maximum sound levels caused by aircraft noise were determined in accordance with the guidelines for calculations of noise abatement zones (AzB) using historical radar data (Bundesregierung-Federal Government of Germany, 2008), and verified with data from local monitoring stations. Sources of the input data were the German flight safety operator (DFS) for aircraft noise calculations, official traffic counts for road traffic exposure estimation, and the Federal Railway Authority and the German Railway environmental department for rail traffic. The reduction in sound levels along the path of propagation between the source of the sound and the immission site was determined based on a digital landscape model that included both the landscape and the "footprints" of buildings, as well as data on the position of noise barriers and walls along roads and railway tracks. Information on building height was not included in the exposure assessment. The average sound levels for road and rail traffic noise were determined based on the methods for calculation (VBUS, VBUSCH) used for EU noise mapping (Bundesregierung, 2006; European Union, 2002). Furthermore, indoor sound levels were estimated for a subgroup with available questionnaire data. Indoor sound levels were assessed considering the orientation of the bedroom with respect to the traffic noise source and self-reported habits of opening windows in the summertime. Further information on all aspects of the acoustic calculations are given by Möhler et al. (2015).

2.3. Linkage of diagnostic data and individual traffic noise data

The participating health insurance funds provided pseudonymized health claims and prescription data to the Data Analysis Office in Dresden. Hospital (in-patient) and ambulatory diagnoses (ICD 10 codes) and prescription data according to the Anatomical Therapeutic Chemical/Defined Daily Dose Classification (ATC) were supplied by the health insurance funds for the years 2005 through 2010.

Linkage of traffic noise data and individuals' address data was conducted by the Data Linkage Office located at the Leibniz Institute for Prevention Research and Epidemiology-BIPS for two health insurance funds, one health insurance did its own linkage. Traffic noise data could be linked to address data for 95.1% of the included individuals with available address information (n = 907,736). Subsequently, diagnostic data and traffic noise data were joined at the Data Analysis Office. The main analyses were based on the 2005 traffic noise data. Only one of the three included statutory health insurance funds linked noise data to previous addresses. No address change was assumed for individuals insured by the two other health insurance funds: so noise levels in 2005 were related to the 2013 address information. For a detailed description of the study methods see Seidler et al. (2015).

2.4. Definition of cases with heart failure and hypertensive heart disease

Patients with a first diagnosis of heart failure and/or hypertensive heart disease (ICD 10 I50, I11, I13.0, or I13.2, based on Schubert et al., 2010) between 2006 and 2010 whose insurance data did not comprise a heart failure or hypertensive heart disease diagnosis four quarters (=12 months) before the first diagnosis in the studied time period were included as cases. Cases had to be 40 years or older at first diagnosis and had to have received a primary hospital discharge diagnosis or two ambulatory secure diagnoses of heart failure or hypertensive heart disease within 12 months (see Table 1). In cases of a secondary hospital discharge diagnosis or (only) one ambulatory secure diagnosis, an ATC-code C01, C03, C07 or C09 prescription was additionally required within a four quarter period to fulfill the case definition criteria. 104,145 individuals fulfilled these criteria and therefore qualified as cases. Subsets of these cases could be created according to diagnosis codes, with some individuals included in more than one subset. Of these, 70,012 heart failure cases (I50.-) were identified including 8945 individuals with I11.0 ("hypertensive heart failure") diagnoses but lacking the additional compulsory manifestation coding (I50.-); 50,681 cases were assigned an I11,- code (hypertensive heart disease) including 37,893 individuals with the code I11.9 (hypertensive heart disease without [congestive] heart failure); and 1054 cases of hypertensive heart and chronic kidney disease with heart failure were identified (I13.0 or I13.2 code). We conducted the main analyses for all cases combined and sub-analyses for the main sub-groups heart failure and hypertensive heart disease.

Table 1
Definition of heart failure/hypertensive heart disease.

ICD-10 classification	heart failure/hypertensive heart disease[a]
I50.-: Heart failure I11.-: Hypertensive heart disease	1.) 1x primary hospital discharge diagnosis I50.-, I11.-, I13.0 or I13.2 and/or 2.) 1x secondary discharge diagnosis I50.-, I11.-, I13.0 or I13.2 and 1x ATC-Code[b]: C01, C03, C07 or C09 within 4 quarter (before or after index quarter) and/or
I13.0: Hypertensive heart and chronic kidney disease with heart failure and with stage 1 through stage 4 chronic kidney disease, or unspecified chronic kidney disease	3.) 2x ambulatory secure diagnosis I50.-, I11.-, I13.0 or I13.2 ("g" = secure) within 4 quarter and/or
I13.2: Hypertensive heart and chronic kidney disease with heart failure and with stage 5 chronic kidney disease, or end stage renal disease	4.) 1x ambulatory secure diagnosis I50.-, I11.-, I13.0 or I13.2 ("g = secure) and 1x ATC-Code: C01, C03, C07 or C09 within 4 quarter (before or after index quarter)

[a] To fulfill the case definition, at least one of the criteria (1. and/or 2. and/or 3. and/or 4.) must be fulfilled.
[b] ATC code C01: cardiac therapy, C03: diuretics, C07: vasoprotectives, C09: agents acting on the renin-angiotensin system.

2.5. Definition of control subjects

Individuals without a stationary or ambulatory diagnosis of heart failure or hypertensive heart disease between 2005 and 2010 were included as control subjects in the analysis. To qualify as control subjects, individuals were required to be aged 40 years or older in 2010 and to have been insured for more than four quarters between 2005 and 2010. Overall, 654,172 individuals qualified as control subjects.

2.6. Potential confounders and statistics

The mean (SD) age at diagnosis of heart failure in cases was 71.8 (±11.5) years; the mean (SD) age of control subjects in 2008 (reference date) was 57.5 (±13.1) years, a difference calling for careful control in the analyses as described below. The characteristics of the cases and control subjects are given in Table 2.

Odds ratios (ORs) and 95% confidence intervals (95% CIs) were calculated by logistic regression analysis. The continuous sound levels for each traffic noise source were grouped in 5 dB categories. For the analysis of road and railway traffic noise, cases and control subjects with noise exposure of less than 40 dB were grouped into the reference category. For the analysis of aircraft noise, individuals exposed to a continuous sound pressure level below 40 dB with the nightly maximum level exceeding 50 dB six or more times (NAT 6) were grouped into a separate exposure category. Addition-

Table 2
Characteristics of cases with heart failure/hypertensive heart disease and control subjects.

	Cases		Control subjects	
	n	%	n	%
Total	104,145	100.0	654,172	100.0
Sex				
Males	46,081	44.2	290,704	44.4
Females	58,064	55.8	363,468	55.6
Age [yrs.]				
35–<45	1527	1.5	128,438	19.6
45–<50	2823	2.7	94,610	14.5
50–<55	4791	4.6	82,091	12.5
55–<60	7184	6.9	77,341	11.8
60–<65	9468	9.1	64,918	9.9
65–<70	14,621	14.0	71,212	10.9
70–<75	18,243	17.5	59,734	9.1
75–<80	16,874	16.2	35,670	5.5
80–<65	14,863	14.3	23,414	3.6
≥85	13,751	13.2	16,744	2.6
Statutory health insurance funds				
Health insurance 1	68,805	66.1	407,571	62.3
Health insurance 2	7926	7.6	47,869	7.3
Health insurance 3	27,414	26.3	198,732	30.4
Education				
Primary/secondary education, no vocational education	3846	3.7	60,352	9.2
Primary/secondary education with vocational education	7113	6.8	130,546	20.0
Graduated from high school, no vocational education	524	0.5	3602	0.6
Graduated from high school and vocational education	1209	1.2	12,222	1.9
College degree	1033	1.0	9006	1.4
University degree	416	0.4	11,123	1.7
Education unknown	90,004	86.4	427,321	65.3
Occupation according to Blossfeld				
AGR Agricultural occupations	141	0.1	2848	0.4
EMB Unskilled manual occupations	1826	1.8	33,048	5.1
QMB Skilled manual occupations	1780	1.7	31.168	4.8
TEC Technicians	278	0.3	4887	0.7
ING Engeneers	96	0.1	2154	0.3
EDI Simple services	3589	3.4	57,396	8.8
QDI Qualified services	472	0.5	11,168	1.7
SEMI Semiprofessionals	614	0.6	19,174	2.9
PROF Professionals	64	0.1	2160	0.3
EVB Simple commercial and administrative occupations	1081	1.0	23,394	3.6
QVB Qualified commercial and administrative occupations	2433	2.3	55,572	8.5
MAN Managers	275	0.3	5293	0.8
SONS Other	440	0.4	7001	1.1
Unknown	91,056	87.4	398,909	61.0
Local proportion of persons receiving unemployment benefits (SGBII; quintiles[a])				
≤6.7%	35,404	34.0	215,346	32.9
>6.7–≤7.5%	18,055	17.3	103,426	15.8
>7.5–≤8.7%	12,464	12.0	70,876	10.8
>8.7–≤12.7%	28,667	27.5	198,572	30.4
>12.7%	9555	9.2	65,952	10.1

[a] Calculation of quintiles: frequent duplication of SGB II-values led to an uneven distribution.

ally, the exposure-risk relationship was examined by linear or third degree polynomial models to the 24-h equivalent continuous sound levels. A linear model was considered as adequate when the difference between the linear AIC value (Akaike Information Criterion) and the polynomial AIC value was ≤5. For all continuous analyses, a starting point of 35 dB was chosen for noise in the range virtually indiscernible from background noise, below 40 dB. For all main analyses, we give the risk increase per 10 dB increase in 24-h continuous levels of traffic noise; odds ratios per 10 dB increase in 24-h continuous levels of traffic noise are written in italics (in Tables 3, 4, S3 and S4) if the linear model is not adequate (rounded AIC difference between linear and 3rd degree polynomial model >5) .

All statistical analyses were adjusted for sex and age. The main analyses were additionally conducted separately for men and women as well as for individuals <60 years and ≥60 years. Age was entered into the logistic regression model as a third degree polynomial (age included as a linear, as a quadratic, and as a cubic term). Furthermore, as an indicator for the regional socioeconomic status, we adjusted for the local proportion of people receiving long-term unemployment benefits. If possible, we additionally adjusted for individuals' socioeconomic status (education and job title). However, for 60.9% of the study subjects (84.2% of the cases, 57.1% of the control subjects; mainly retired persons and co-insured family members) neither individual education nor individual job title or individual behavioral risk factors were recorded in the health claims data. In a subgroup analysis, we therefore restricted our analysis to cases and control subjects with known individual socioeconomic status (education and/or job title). We furthermore conducted an additional questionnaire-based survey to assess unknown or residual confounding.

Table 3
Traffic noise (24 h sound levels $L_{Aeq,24h}$) and heart failure/hypertensive heart disease.

Exposure	Aircraft noise				Road traffic noise				Railway noise			
	Cases	Control subjects	OR	95%-CI	Cases	Control subjects	OR	95%-CI	Cases	Control subjects	OR	95%-CI
Heart failure and hypertensive heart disease (n = 104,145 cases)												
<40 dB, Max. <50 dB[#]	42,208	253,804	1.00	–	11,330	67,680	1.00	–	56,368	349,005	1.00	–
<40 dB, Max. ≥50 dB	6931	40,861	1.06	1.03–1.09								
≥40–<45 dB	30,463	197,474	1.01	0.99–1.03	22,990	137,420	1.03	1.01–1.06	12,698	82,525	1.02	1.00–1.04
≥45–<50 dB	16,604	106,497	1.07	1.04–1.09	25,147	157,094	1.02	1.00–1.05	16,524	104,006	1.07	1.05–1.09
≥50–<55 dB	6113	42,620	1.00	0.96–1.03	17,851	117,957	1.02	0.99–1.05	11,274	72,126	1.05	1.03–1.08
≥55–<60 dB	1802	12,744	1.03	0.98–1.09	11,291	71,948	1.04	1.01–1.08	4411	28,209	1.04	1.00–1.07
≥60–<65 dB	24	172	0.97	0.61–1.53	8329	54,341	1.07	1.03–1.10	1749	10,720	1.09	1.03–1.15
≥65–<70 dB	–	–	–	–	5610	37,141	1.09	1.05–1.13	692	4821	1.06	0.98–1.16
≥70 dB	–	–	–	–	1597	10,591	1.13	1.06–1.20	429	2760	1.17	1.04–1.30
Continuous (per 10 dB)§			1.016	1.003–1.030 p = 0.020			1.024	1.016–1.032 p < 0.001			1.031	1.022–1.041 p < 0.001
AIC difference between linear and 3rd degree polynomial model (AIC_{linear}-$AIC_{polynomial}$)			4.3				5.2				1.0	
Solely cases with heart failure (I50[a]; n = 70,012 cases)												
<40 dB, Max. <50 dB	29,654	253,804	1.00	–	8140	67,680	1.00	–	38,387	349,005	1.00	–
<40 dB, Max. ≥50 dB	4664	40,861	1.03	1.00–1.07								
≥40–<45 dB	19,886	197,474	0.96	0.93–0.98	15,523	137,420	0.97	0.95–1.01	8229	82,525	0.98	0.96–1.01
≥45–<50 dB	10,844	106,497	1.02	0.99–1.05	16,886	157,094	0.97	0.94–1.00	10,911	104,006	1.04	1.01–1.06
≥50–<55 dB	3852	42,620	0.92	0.89–0.96	11,648	117,957	0.95	0.92–0.98	7469	72,126	1.02	1.00–1.05
≥55–<60 dB	1094	12,744	0.93	0.87–1.00	7634	71,948	1.00	0.96–1.03	3036	28,209	1.04	0.99–1.08
≥60–<65 dB	18	172	1.12	0.67–1.88	5465	54,341	1.00	0.96–1.04	1228	10,720	1.11	1.04–1.19
≥65–<70 dB	–	–	–	–	3659	37,141	1.02	0.97–1.06	464	4821	1.07	0.96–1.18
≥70 dB	–	–	–	–	1057	10,591	1.08	1.01–1.17	288	2760	1.17	1.03–1.34
Continuous (per 10 dB)§			*0.974*	*0.958–0.990* *p = 0.001*			*1.011*	*1.001–1.021* *p = 0.030*			1.023	1.012–1.034 p < 0.001
AIC difference between linear and 3rd degree polynomial model (AIC_{linear}-$AIC_{polynomial}$)			6.6				25.9				–0.2	p < 0.001
Solely cases with hypertensive heart disease (I11.-; n = 50,681)												
<40 dB, Max. <50 dB	18,396	253,804	1.00	–	4762	67,680	1.00	–	26,391	349,005	1.00	–
<40 dB, Max. ≥50 dB	3415	40,861	1.16	1.11–1.20								
≥40–<45 dB	15,895	197,474	1.18	1.15–1.21	10,975	137,420	1.16	1.12–1.20	6665	82,525	1.12	1.08–1.15
≥45–<50 dB	8684	106,497	1.24	1.21–1.28	12,498	157,094	1.18	1.14–1.22	8452	104,006	1.15	1.12–1.18
≥50–<55 dB	3302	42,620	1.19	1.14–1.24	9065	117,957	1.18	1.13–1.22	5729	72,126	1.12	1.09–1.16
≥55–<60 dB	979	12,744	1.26	1.18–1.35	5486	71,948	1.17	1.12–1.22	2091	28,209	1.05	1.00–1.10
≥60–<65 dB	10	172	0.86	0.45–1.65	4246	54,341	1.24	1.19–1.30	819	10,720	1.09	1.01–1.17
≥65–<70 dB	–	–	–	–	2861	37,141	1.25	1.19–1.32	342	4821	1.09	0.97–1.22
≥70 dB	–	–	–	–	788	10,591	1.25	1.16–1.36	192	2760	1.07	0.92–1.24
Continuous (per 10 dB)[b]			*1.126*	*1.107–1.146* *p < 0.001*			*1.052*	*1.041–1.063* *p < 0.001*			*1.055*	*1.042–1.067* *p < 0.001*
AIC difference between linear and 3rd degree polynomial model (AIC_{linear}-$AIC_{polynomial}$)			40.4				37.4				74.9	

OR: Odds Ratio; adjusted for age, sex, education, and job title (when available), local proportion of persons receiving unemployment benefits; 95%-CI: 95%-confidence intervals.

[#] For road and railway traffic noise, the reference category included all individuals with 24 h continuous sound levels <40 dB (independent from the nightly maximum sound levels).

[a] N = 8945 individuals with ICD-10 code I11.0 were included in this category.

[b] ORs per 10 dB increase in 24-h continuous levels of traffic noise are written in italics if the linear model is not adequate (rounded AIC difference between linear and 3rd degree polynomial model >5).

Table 4
Traffic noise (24 h sound levels $L_{Aeq,24h}$) and heart failure/hypertensive heart disease, analysis restricted to persons for whom the individual socioeconomic status was known from the health insurance data (16% of cases, 43% of control subjects).

Exposure	Aircraft noise				Road traffic noise				Railway noise			
	Cases	Control subjects	OR	95%-CI	Cases	Control subjects	OR	95%-CI	Cases	Control subjects	OR	95%-CI
Heart failure and hypertensive heart disease (n = 16,495 cases)												
<40 dB, Max. <50 dB[#]	6121	108,159	1.00	–	1620	29,067	1.00	–	8625	150,724	1.00	–
<40 dB, Max. ≥50 dB	1077	17,808	1.10	1.03–1.18								
≥40–<45 dB	5067	85,301	1.08	1.04–1.13	3474	58,857	1.06	0.99–1.13	2130	35,244	1.08	1.02–1.13
≥45–<50 dB	2859	45,257	1.17	1.11–1.24	3917	67,308	1.05	0.99–1.12	2671	43,974	1.09	1.04–1.14
≥50–<55 dB	1056	18,363	1.07	0.99–1.15	3008	50,499	1.08	1.01–1.15	1916	30,680	1.11	1.05–1.17
≥55–<60 dB	311	5413	1.09	0.96–1.23	1787	30,749	1.04	0.97–1.12	686	11,926	1.04	0.95–1.13
≥60–<65 dB	4	80	1.04	0.37–2.92	1415	23,489	1.11	1.02–1.19	264	4552	1.04	0.91–1.18
≥65–<70 dB	–	–	–	–	1003	15,866	1.14	1.05–1.24	121	2070	1.07	0.89–1.30
≥70 dB	–	–	–	–	271	4546	1.11	0.97–1.28	82	1211	1.28	1.02–1.62
Continuous (per 10 dB)[a]			*1.061*	*1.029–1.094* *p <0.001*			1.029	1.010–1.048 p = 0.003			1.043	1.022–1.064 p < 0.001
AIC difference between linear and 3rd degree polynomial model (AIC_{linear}-$AIC_{polynomial}$)			10.8				−2.5				4.8	
Solely cases with heart failure (I50; n = 8440 cases)												
<40 dB, Max. <50 dB	3278	108,159	1.00	–	893	29,067	1.00	–	4463	150,724	1.00	–
<40 dB, Max. ≥50 dB	542	17,808	1.07	0.97–1.12								
≥40–<45 dB	2517	85,301	1.02	0.96–1.08	1808	58,857	1.00	0.92–1.09	1074	35,244	1.05	0.98–1.13
≥45–<50 dB	1430	45,257	1.12	1.04–1.20	2007	67,308	0.98	0.90–1.07	1347	43,974	1.05	0.99–1.13
≥50–<55 dB	524	18,363	1.01	0.91–1.12	1476	50,499	0.97	0.88–1.05	953	30,680	1.05	0.98–1.13
≥55–<60 dB	146	5413	0.97	0.82–1.16	907	30,749	0.97	0.87–1.07	356	11,926	1.01	0.90–1.13
≥60–<65 dB	3	80	1.64	0.50–5.33	708	23,489	1.01	0.91–1.12	145	4552	1.10	0.93–1.31
≥65–<70 dB	–	–	–	–	500	15,866	1.03	0.92–1.15	59	2070	1.00	0.76–1.30
≥70 dB	–	–	–	–	141	4546	1.06	0.88–1.27	43	1211	1.29	0.94–1.77
Continuous (per 10 dB)[a]			1.024	0.982–1.068 p = 0.268			1.005	0.979–1.030 p = 0.730			1.030	1.001–1.059 p = 0.040
AIC difference between linear and 3rd degree polynomial model (AIC_{linear}-$AIC_{polynomial}$)			1.1				−0.5				−3.1	
Solely cases with hypertensive heart disease (I11.-; n = 10,844 cases)												
<40 dB, Max. <50 dB	3734	108,159	1.00	–	985	29,067	1.00	–	5538	150,724	1.00	–
<40 dB, Max. ≥50 dB	707	17,808	1.16	1.06–1.26								
≥40–<45 dB	3503	85,301	1.22	1.16–1.28	2237	58,857	1.12	1.03–1.21	1448	35,244	1.13	1.06–1.20
≥45–<50 dB	1967	45,257	1.31	1.23–1.40	2601	67,308	1.13	1.05–1.22	1820	43,974	1.15	1.09–1.22
≥50–<55 dB	718	18,363	1.18	1.08–1.29	2019	50,499	1.17	1.08–1.26	1291	30,680	1.16	1.09–1.24
≥55–<60 dB	213	5413	1.23	1.06–1.43	1194	30,749	1.14	1.04–1.24	455	11,926	1.07	0.97–1.18
≥60–<65 dB	2	80	0.81	0.20–3.36	953	23,489	1.21	1.10–1.33	165	4552	1.01	0.86–1.18
≥65–<70 dB	–	–	–	–	668	15,866	1.24	1.12–1.37	83	2070	1.14	0.91–1.43
≥70 dB	–	–	–	–	187	4546	1.24	1.06–1.46	44	1211	1.05	0.78–1.43
Continuous (per 10 dB)[a]			*1.135*	*1.095–1.177* *p < 0.001*			1.051	1.028–1.074 p < 0.001			*1.056*	*1.030–1.082* *p < 0.001*
AIC difference between linear and 3rd degree polynomial model (AIC_{linear}-$AIC_{polynomial}$)			27.2				−0.6				20.3	

OR: Odds Ratio; adjusted for age, sex, education, and job title (when available), local proportion of persons receiving unemployment benefits; 95%-CI: 95%-confidence intervals.

[#] For road and railway traffic noise, the reference category included all individuals with 24 h continuous sound levels <40 dB (independent from the nightly maximum sound levels).

[a] ORs per 10 dB increase in 24-h continuous levels of traffic noise are written in italics if the linear model is not adequate (rounded AIC difference between linear and 3rd degree polynomial model > 5).

2.7. Questionnaire-based survey

A questionnaire-based survey was conducted among a sample of individuals with heart failure or hypertensive heart disease and corresponding controls (n = 6640) to obtain information on the potential confounders education, occupation, salary, smoking, height and weight, alcohol consumption, working night shifts, noise exposure at work, and physical activity level for the year 2005. Also, information concerning the location of the bedroom with respect to the nearest road and railway, as well as information on whether the bedroom windows were opened at night, was collected to estimate the interior sound level.

All cases with cardiovascular diseases and a random selection of eligible controls were selected by the Data Analysis Office for the questionnaire-based survey. After de-pseudonymization, the health insurance funds contacted all surviving subjects in the beginning of 2015 by letter. The response was 5.5%. Interview data of cases with heart failure/hypertensive heart disease and corresponding controls were used to estimate the distortion of results obtained from analyses of health claims data through undetected and residual confounding (primary aim of the survey) as well as to compare risk estimators of interior traffic noise sound levels with risk estimators of sound pressure levels from the house front (secondary aim of the survey).

3. Results

For all examined types of traffic noise (aircraft noise, road traffic noise and railway noise), the exposure-risk-relation was adequately reflected by a linear model. In Table 3, the results for the continuous (linear) 24-h sound levels as well as for the categorical analyses (for the entire case group, solely for cases with heart failure and solely for cases with hypertensive heart disease) are given separately for each of the three traffic noise sources. In general, results for the night time period 22–06 h were comparable with the results for the 24-h continuous noise levels (see Supplementary Tables S1–S2).

3.1. Aircraft noise and heart failure/hypertensive heart disease

With respect to the 24-h continuous noise levels ($L_{Aeq,24h}$), we found a risk increase of 1.6% (95% CI 0.3–3.0%) per 10 dB. In the categorical analysis, the OR was significantly elevated to 1.07 (95% CI 1.04–1.09) at 45 to <50 dB sound levels. Furthermore, a significantly increased risk (OR = 1.06; 95% CI 1.03–1.09) was observed for individuals with nightly maximum sound levels of >50 dB (NAT 6) and continuous noise levels <40 dB. Risk increases per 10 dB were somewhat higher in females than in men (2.1 vs. 1.1%; Supplementary Table S3), the corresponding interaction term between aircraft noise and sex did not reach statistical significance. Individuals <60 years showed higher risks than individuals ≥60 years (Supplementary Table S4).

When cases with heart failure (n = 70,012 cases) were analyzed separately, we no longer found a positive dose-response-relationship with aircraft noise exposure. Instead significant negative ORs were observed in several exposure categories (Table 3). Only in the highest exposure category of 60 to <65 dB sound levels, the OR was non-significantly elevated to 1.12 (95% CI 0.67–1.88). Restriction of the analysis to cases with hypertensive heart disease (n = 50,681 cases) led to a considerable elevation of aircraft noise risks. In the categorical analysis, the highest OR was found among individuals exposed to 55 to <60 dB (OR = 1.26; 95% CI 1.18–1.35).

3.2. Road traffic noise and heart failure

We found a risk increase of 2.4% (95% CI 1.6–3.2%) per 10 dB increase in 24-h continuous levels of road traffic noise. The categorical analysis showed a nearly monotonous risk increase, reaching statistical significance from 55 dB upwards. In the highest category of road traffic noise (≥70 dB), the OR was elevated to 1.13 (95% CI 1.06–1.20). We found comparable risk increases for females (2.2%) and males (2.4%; Supplementary Table S3) and slightly higher risk estimates for persons under 60 years of age than for persons aged 60 years or more (Supplementary Table S4). Individuals <60 years showed higher risks than individuals ≥60 years, particularly at noise levels ≥65 dB (Supplementary Table S4).

In all exposure categories, the risk increase was lower in the heart failure subgroup than in the hypertensive heart disease subgroup (Table 3).

3.3. Railway traffic noise and heart failure

In the entire case group, the highest risk increase of 3.1% (95% CI 2.2–4.1%) per 10 dB increase in 24-h continuous levels was observed for railway traffic noise. The ORs for the single sound level categories were mostly higher than the corresponding ORs for aircraft or road traffic noise. In the highest category of railway traffic noise (≥70 dB), we found an OR of 1.17 (95% CI 1.04–1.30). Risk increases were considerably higher for females than for males (4.7% vs. 1.0%; Supplementary Table S3).

For railway traffic noise, the risk increase was lower in the heart failure subgroup than in the hypertensive heart disease subgroup in all exposure categories <60 dB. However, in the highest exposure category of >70 dB, the OR was 1.17 (95% CI 1.03–1.34) in the heart failure subgroup, but only 1.07 (95% CI 0.92–1.24) in the hypertensive heart disease subgroup.

3.4. Bias from undetected or residual confounding?

To elucidate potential confounding by socioeconomic status, the analysis was restricted to persons for whom the individual socioeconomic information for 2005 was available from the health insurance data (Table 4). Aircraft noise-related risks for heart failure and hypertensive heart disease increased in most exposure categories for the entire case group as well as for the heart failure and hypertensive heart disease subgroups (Table 4, left column). We found the highest significant risk estimates in the exposure category 45 to <50 dB: the OR was 1.17 (95% CI 1.1–1.24) for the entire case group, 1.12 (95% CI 1.04–1.20) for the heart failure subgroup and 1.31 (95% CI 1.23–1.40) for the hypertensive heart failure subgroup. Restriction of the analysis to persons with known individual socioeconomic status (Table 4, middle column) had no substantial influence on the road traffic noise risks, while the railway traffic noise-related ORs for heart failure and hypertensive heart disease increased slightly (Table 4, right column): for the entire case group, restriction to persons with known individual socioeconomic status led to an OR of 1.28 (95% CI 1.02–1.62) in the highest exposure category of >70 dB, with a risk increase of 4.3% (95% CI 2.2–6.4%) per 10 dB increase in 24-h continuous levels of railway traffic noise.

A subset of 2639 persons with heart failure and/or hypertensive heart disease and 3890 corresponding control subjects without a diagnosis of heart failure or hypertensive heart disease were included in the analysis of the questionnaire-based survey. The heart failure and hypertensive heart disease risks of the health claims data analyses could be reproduced in the subset models by trend. Adjustment for tobacco and alcohol consumption, body-mass index, noise at work and physical activity as well as individual socioeconomic status ("fully adjusted model", no table) had no substantial influence on the risk estimates. However, this

questionnaire-based survey is open to selection bias. We therefore cannot exclude confounding by smoking, alcohol consumption, nutrition, or other lifestyle factors.

3.5. Comparison of interior and exterior sound levels on disease risks

When accounting for effects of interior sound pressure levels on disease risk in the questionnnaire-based survey (see Supplementary Tables S5–S7), we found considerably increased odds ratios of being diagnosed with heart failure/hypertensive heart disease for all traffic noise sources in comparison to exterior sound pressure noise levels: observed risk increases ranged from 1.4 to 4.3% per 10 dB rise in interior sound levels (starting point: 10 dB) compared with risks between −1.1 and 1.6% per 10 dB increase in exterior sound levels (starting point: 35 dB).

4. Discussion

This large case-control study based on health claims data and survey responses finds a relationship between aircraft, road traffic and railway noise exposure and the diagnosis of heart failure and hypertensive heart disease.

4.1. Strengths and limitations of this study

This study permits the first direct comparison of risk estimates for aircraft, road and railway traffic noise on the basis of a large data set of people insured by statutory health insurance funds. Traffic noise data was generated precisely for each address in the study area, using most recent international guidelines and multiple information sources. According to in-depth error estimations including stationary noise measurements, the standard error of our noise exposure estimates is between 3 and 5 dB (Möhler et al., 2015, 2016). Uncertainties of exposure measurement equally affect cases and control subjects; as a general rule, the resulting non-differential exposure misclassification might lead to a slight underestimation of risks.

For two of the three included health insurance funds (Insurance 1 and 2), previous addresses were unknown, so noise levels in 2005 had to be related to the 2013 address information. Therefore, exposure misclassification cannot be excluded for those individuals that moved between 2005 and 2013. To examine the consequences of this potential exposure misclassification, we re-analyzed the data of Insurance 3, ignoring previous address information. As a result of this sensitivity analysis, risk estimates did not substantially alter. Therefore, this sensitivity analysis speaks against biased results as a consequence of unknown moves.

The algorithm that we used for identifying new cases was based on both stationary and ambulant diagnoses. However, this approach could not entirely exclude prevalent cases from our case group.

We considered maximum nightly sound pressure levels separately from continuous sound pressure levels. This is in line with the hypothesis of sleep disturbance constituting an important pathophysiological link, particularly between aircraft traffic noise and disease risks. One important novel result of our study is the impact of the maximum aircraft noise levels at night on the cardiovascular system: significantly increased heart failure risks suggest that nightly maximum sound pressure levels exceeding 50 dB lead to increased disease risks from aircraft noise even if continuous sound pressure levels are below 40 dB. This finding is relevant for the general population living around airports, however, it requires validation in future studies.

Considerable efforts were made to test for undetected or residual confounding. First, we restricted our analysis to persons for whom the individual socioeconomic status in 2005 (education and/or job group) was known from the insurance data (16% of the cases, 43% of the control subjects). Generally the individual socioeconomic status is only known for working people; the low proportion of cases included in this sub-analysis is mainly explained by considerably higher mean age of cases. Exclusion of persons with unknown individual socioeconomic status led to an increase in aircraft and railway traffic noise risks, whereas road traffic noise risk did not change materially. Overall, these results indicate a rather limited effect of undetected socioeconomic confounding. Second, 6529 insured individuals including cases with heart failure/hypertensive heart disease and corresponding control subjects contributed primary data to our survey. As an overall result of this sub-analysis, the risk estimates for heart failure/hypertensive heart disease did not appear to be substantially and systematically biased by insufficient consideration of socioeconomic status and lifestyle factors. As a point in favour of the usability of the survey data, BMI and smoking were positively associated with heart failure/hypertensive heart disease in the subpopulation. The very low response of 5.5% might nevertheless have introduced substantial selection bias in this subgroup of survey participants.

We were not able to adjust for air pollution as potential confounder. However, in their systematic review on road traffic noise and aircraft noise, Vienneau et al. (2015) did not find air pollution adjustment to substantially attenuate the association between traffic noise exposure and ischemic heart disease. Based on this finding, we regard substantial undetected confounding by air pollution as rather improbable explanation of our results.

The risk estimates based on interior sound pressure levels were higher for aircraft, road, and railway traffic noise than risk estimates based on exterior sound pressure levels. In principle, this result is indicative of a causal effect between noise exposure and the development of heart failure: due to the individual living situation (e.g. orientation of rooms) and "living habits" (e.g. position/state of window) exterior sound pressure levels are not comparable with the real noise exposure "at the ear of the sleeper". Thus, interior sound pressure levels may better reflect the real noise exposure.

The insurees included in our study represent about 23% of the population aged 40 or above in the study area, so external validity of the results might be questioned. We found differences between the participating health insurance funds, particularly with respect to socioeconomic status of the insured clientele. However, no systematic differences in risks were found when data of the health insurance funds were analyzed separately. This supports the external validity of our results.

Secondary data based on health insurance claims have the great advantage of negligible selection bias with regard to exposure or case-control status. However, accuracy of diagnoses largely depends on the reliability of the physicians' ICD codings. To ensure a high diagnostic accuracy, we made use of secondary or ambulatory diagnoses only when additional confirming information (e.g. information about compatible prescriptions) was also documented.

Our study is based exclusively on noise estimations at the individuals' home address. We do not know which proportion of everyday life the included cases and control subjects spent at the registered address. Adjustment for noise at work had no substantial effect on the traffic noise-related risk estimators in the subset of responders to our additional questionnaire survey.

4.2. Risk estimates stratified for age and sex

In our study, risk estimates tended to be higher for persons under 60 years of age. This is in accordance with Greiser and Greiser (2015), who found decreasing risks of all cardiovascular diseases combined (including heart failure) with increasing age. In

contrast, Vienneau et al. (2015) found higher traffic noise-related risks for ischemic heart disease among older persons. However, age dependency of risks for heart failure and HHD might differ. Further potential explanations for our findings are a higher proportion of cases with HHD among younger persons and potentially less secure diagnoses (with more frequent comorbidities) among the older ones.

For railway traffic noise and aircraft noise, women showed a slightly higher risk of heart failure or HHD, whereas the risk estimates of men and women were comparable for road traffic noise. As a potential explanation for the observed sex differences in risks, the higher proportion of working people among men aged ≥40 years should be taken into consideration: the estimated 24-h residential noise exposure might better reflect the "de facto" noise exposure among women than among men.

4.3. Comparison of risk estimates for aircraft, road, and railway traffic noise

Considering heart failure risks per 10 dB increase in 24-h continuous sound pressure levels, in our main analysis, risk estimates tended to be more pronounced for railway traffic noise (3.1% per 10 dB) and road traffic noise (2.4% per 10 dB) in comparison to aircraft noise (1.6% per dB). However, the comparability of the aircraft noise results with the road traffic and railway noise is limited: In the linear model, persons with continuous noise levels <40 dB but with nightly maximum sound levels of >50 dB are included in the reference category. This might flatten the exposure-response-relation. Moreover, there are only very few people with an aircraft noise exposure above 60 dB (and there was no aircraft noise exposure above 62 dB at all in our study region in 2005). Overall, our results point to a particularly high risk of railway noise, especially for heart failure. We do not have an explanation for the decreased risk estimates for the association between aircraft noise and heart failure. As this negative association disappears when restricting the analysis to persons for whom the individual socioeconomic status was known (see Table 4), confounding by socioeconomic status in the main analysis may partly be responsible for this.

4.4. Comparison of study results with those obtained from former studies

Previous studies mostly focus on the relationship between traffic noise and hypertension, ischemic heart disease or stroke; studies specifically focusing on heart failure or hypertensive heart disease are rare. Hypertension is an important risk factor for hypertensive heart disease, but also for heart failure. Several studies examined the relationship between traffic noise – particularly road traffic noise and aircraft noise – and hypertension. A meta-analysis of 24 cross-sectional studies reveals a statistically significant risk increase of about 7% (95% CI 2–12%) per 10 dB road traffic noise (Van Kempen and Babisch, 2012). In their meta-analysis of five studies on the relationship between aircraft noise and hypertension, Babisch and van Kamp (2009) find a pooled risk increase of 13% (95% CI 0–2.8), a finding which Huang et al. (2015) confirm in their recently published meta-analysis. These authors nevertheless conclude that the evidence for a relationship between aircraft noise exposure and hypertension is still inconclusive because of limitations in study populations and exposure characterization as well as because of the potential for confounding. However, increased hypertension risks are not a necessary precondition for traffic noise-related increased risks of heart failure and hypertensive heart disease. Traffic noise could also affect the course of hypertension in persons who have acquired their hypertension independently from traffic noise.

For all three traffic noise exposures, risks for hypertensive heart disease are higher than the corresponding heart failure risks in our study. This is especially the case for aircraft noise exposure, for which we find particularly high risks for hypertensive heart disease. While the development of hypertensive heart disease is directly related to arterial hypertension, hypertension is only one – but by far not the only – pathophysiologic pathway to heart failure. There are other causes of heart failure (e.g. valvular heart disease, myocarditis, diabetes) whose association with traffic noise may differ from that of hypertension.

The HYENA study reported risk elevations for heart failure of comparable magnitude as for ischemic heart disease or stroke (Floud et al., 2013). In our case-control study, road traffic and aircraft noise-related risks, but not railway traffic noise-related risks were somewhat higher for heart failure/hypertensive heart disease than for myocardial infarction (Seidler et al., 2016). Current reviews on ischemic heart disease published by Vienneau et al. (2015) and Babisch (2014) report somewhat higher traffic noise-related risk estimates in comparison to our study on heart failure: the pooled analyses from Babisch (2014) suggest an increase in coronary heart disease risk of 8% per 10 dB increase in road traffic day-night equivalent noise level (L_{DN}), with a starting point of 52 dB. Vienneau et al. (2015) find a 4% increase in risk per 10 dB road traffic noise increase (L_{DEN}), with a starting point of 50 dB. For aircraft noise (L_{DEN}), these authors find an increase in risk of 6% per 10 dB. In contrast, the results of our study suggest risk estimates for heart failure between 1.6 and 3.1% per 10 dB increase of $L_{Aeq,24h}$ depending on the studied traffic noise sources. However, one has to keep in mind that we chose a starting point of 35 dB. According to the results of meta-analysis conducted by Vienneau et al. (2015), the estimated risk elevation for ischemic heart disease would be approximately 6.9% for a traffic noise level of 60 dB ($L_{Aeq,24h}$). Assuming a starting point of 35 dB as in our study, this would be equivalent to an increase of 2.7% per 10 dB, which is roughly compatible with our findings. Thus we conclude that our findings for traffic noise-related heart failure risks are of the same magnitude as the hitherto "best estimates" for ischemic heart disease risks.

We would like to underline that both Vienneau et al. (2015) and Babisch (2014) find that a linear model adequately reflects the dose-response relationship. Accordingly our study results do not suggest a threshold of effect despite the low starting point. Therefore, a "no-effect level" cannot be defined at this time,

4.5. Evaluating effect sizes—are low traffic noise-based increases in disease risks relevant for the population?

Risk estimates for noise exposure found in this and other studies are much lower than risks for "known" harmful factors such as tobacco consumption and increased body-mass for the development of cardiovascular diseases. However, lifestyle risks can be influenced by individual behavior. In contrast, protection against health consequences of traffic noise exposure is a governmental task. Therefore traffic noise-related risks and lifestyle risks are not directly comparable. Moreover, one has to keep in mind that a large part of the population is exposed to traffic noise, an exposure which is associated with (albeit low) increases in risks for cardiovascular diseases. Based on the population-based occurrence of traffic noise exposure as well as the prevalence of cardiovascular diseases, even low increases in risks have a considerable public health-relevance. Preventive measures should be intensified to reduce noise levels for aircraft, road traffic and railway noise. Since to date systematic reviews and our large case-control study do not find threshold levels for health-related effects, such preventive interventions should intend to minimize traffic noise exposure as much as reasonably achievable.

Conflict of interest

The authors declare they have no actual or potential competing financial interests.

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We complied with the comments of the Ethics Committee of the Medical Faculty, TU Dresden (AZ: EK328102012; 21 February 2013 and 22 April 2014). The Federal Commissioner for Data Protection and Freedom of Information (AZ: III-320/010#0011; reply of 11 June 2012) and the Data Protection Commissioners of the German states Hesse (AZ: 43.60-we; reply of 13 March 2012; amendments 7 February 2014) and Rhineland-Palatinate (AZ: 6.08.22.002; reply of 7 May 2012; amendments 4 February 2014) approved the study concept. These authorities confirmed, that the research project is, in principle, in accordance with data protection regulations.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.ijheh.2016.09.012.

References

Babisch, W., van Kamp, I., 2009. Exposure-response relationship of the association between aircraft noise and the risk of hypertension. Noise Health 11, 161–168.

Babisch, W., 2014. Updated exposure-response relationship between road traffic noise and coronary heart diseases: a meta-analysis. Noise Health 16, 1–9.

Badran, M., Yassin, B.A., Fox, N., Laher, I., Ayas, N., 2015. Epidemiology of sleep disturbances and cardiovascular consequences. Can. J. Cardiol. 31, 873–879.

Beelen, R., Hoek, G., Houthuijs, D., van den Brandt, P.A., Goldbohm, R.A., Fischer, P., Schouten, L.J., Armstrong, B., Brunekreef, B., 2009. The joint association of air pollution and noise from road traffic with cardiovascular mortality in a cohort study. Occup. Environ. Med. 66, 243–250.

Bundesregierung, 2006. Verordnung zur Durchführung des Bundes-Immissionsschutzgesetzes (Verordnung über die Lärmkartierung) vom 6. März 2006 (34. BImSchV (2006). Bundesanzeiger 154a, 6).

Bundesregierung, 2008. Erste Verordnung über die Datenerfassung und das Berechnungsverfahren für die Festsetzung von Lärmschutzbereichen vom 27. Dezember 2008 (1. FlugLSV). Anlage 2: Anleitung zur Berechnung von Lärmschutzbereichen (AzB). Bundesanzeiger 195a, 2.

Correia, A.W., Peters, J.L., Levy, J.I., Melly, S., Dominici, F., 2013. Residential exposure to aircraft noise and hospital admissions for cardiovascular diseases: multi-airport retrospective study. BMJ 347, f5561.

European Union, 2002. Directive 2002/49/EC of the European Parliament and of the Council of 25 June 2002 relating to the assessment and management of environmental noise—Declaration by the Commission in the Conciliation Committee on the Directive relating to the assessment and management of environmental noise, Available: http://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX:32002L0049 (accessed 23.07.16.).

Floud, S., Blangiardo, M., Clark, C., de Hoogh, K., Babisch, W., Houthuijs, D., Swart, W., Pershagen, G., Katsouyanni, K., Velonakis, M., Vigna-Taglianti, F., Cadum, E., Hansell, A.L., 2013. Exposure to aircraft and road traffic noise and associations with heart disease and stroke in six European countries: a cross-sectional study. Environ. Health 12, 89.

Gan, W.Q., Davies, H.W., Koehoorn, M., Brauer, M., 2012. Association of long-term exposure to community noise and traffic-related air pollution with coronary heart disease mortality. Am. J. Epidemiol. 175, 898–906.

Greiser, E., Greiser, C., 2015. Umgebungslärm und Gesundheit am Beispiel Bremen [Environmental noise and health with Bremen as an example]. Federal Institute of Environment http://www.umweltbundesamt.de/publikationen/umgebungslaerm-gesundheit-am-beispiel-bremen.

Halonen, J.I., Vahtera, J., Stansfeld, S., Yli-Tuomi, T., Salo, P., Pentti, J., Kivimäki, M., Lanki, T., 2012. Associations between nighttime traffic noise and sleep: the Finnish public sector study. Environ. Health Perspect. 120, 1391–1396, Erratum in: Environ. Health Perspect. 2013, 121, A147.

Huang, D., Song, X., Cui, Q., Tian, J., Wang, Q., Yang, K., 2015. Is there an association between aircraft noise exposure and the incidence of hypertension? A meta-analysis of 16784 participants. Noise Health 17, 93–97.

Möhler, U., Liepert, M., Mühlbacher, M., Beronius, A., Nunberger, M., Braunstein, G., Brillé, G., Schaal, J., Bartel, R., 2015. Erfassung der Verkehrsgeräuschexposition. Dokumentation zur Berechnung der akustischen Daten in NORAH. NORAH (Noise-related annoyance, cognition and health): Verkehrslärmwirkungen im Flughafenumfeld. Endbericht, Band 2, http://www.laermstudie.de/fileadmin/files/Laermstudie/Akustik_Wiss_Ergebnisbericht.pdf (accessed 29.08.16.).

Möhler, U., Liepert, M., Mühlbacher, M., Klatte, M., Vogelsang, B., Thomann, G., 2016. The acoustic basis of the NORAH field studies. 45th International Congress on Noise Control Engineering (INTER-NOISE 2016), http://www.norah-studie.de//de/publikationen.html?file=files/norah-studie.de/Downloads/Moehler_etal_2016_Acoustic%20basis_IN2016-288.pdf (last accessed 29.08.16.).

Schubert, I., Ihle, P., Köster, I., 2010. Interne Validierung von Diagnosen in GKV-Routinedaten: Konzeption mit Beispielen und Falldefinition. Das Gesundheitswesen 72, 316–322.

Seidler, A., Wagner, M., Schubert, M., Dröge, P., Hegewald, J., 2015. Sekundärdatenbasierte Fallkontrollstudie mit vertiefender Befragung. NORAH (Noise-related annoyance, cognition and health): Verkehrslärmwirkungen im Flughafenumfeld. Endbericht, Band 6, http://www.laermstudie.de/fileadmin/files/Laermstudie/Krankheitsrisiken_Wiss_Ergebnisbericht.pdf (accessed 23.07.16.).

Seidler, A., Wagner, M., Schubert, M., Dröge, P., Pons-Kühnemann, J., Swart, E., Zeeb, H., Hegewald, J., 2016. Myocardial infarction risk due to aircraft, road, and rail traffic noise. Dtsch. Arztebl. Int. 113, 407–414.

Van Hee, V.C., Adar, S.D., Szpiro, A.A., Barr, R.G., Diez Roux, A., Bluemke, D.A., Sheppard, L., Gill, E.A., Bahrami, H., Wassel, C., Sale, M.M., Siscovick, D.S., Rotter, J.I., Rich, S.S., Kaufman, J.D., 2009. Exposure to traffic and left ventricular mass and function: the Multi-Ethnic Study of Atherosclerosis. Am. J. Respir. Crit. Care Med. 179, 827–834.

Van Kempen, E., Babisch, W., 2012. The quantitative relationship between road traffic noise and hypertension: a meta-analysis. J. Hypertens. 30, 1075–1086.

Vienneau, D., Schindler, C., Perez, L., Probst-Hensch, N., Röösli, M., 2015. The relationship between transportation noise exposure and ischemic heart disease: a meta-analysis. Environ. Res. 138, 372–380.

WHO, 2010. Burden of disease from environmental noise: quantification of healthy life years lost in Europe. WHO: European Centre for Environment and Health, Bonn, http://www.euro.who.int/_data/assets/pdf_file/0008/136466/e94888.pdf (accessed 23.07.16.).