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Disease risks of traffic noise - a large case-control study based on secondary data

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ABSTRACT

Background: Several studies point to an elevated risk for cardiovascular disease induced by traffic noise. Evidence is limited with respect to depressive disorders and breast cancer.
Objective: To examine the association between aircraft noise, road traffic and railway traffic noise with cardiovascular diseases (myocardial infarction, stroke, heart failure or hypertensive heart disease), depressive episodes and breast cancer in a large case-control study.
Methods: The classification of cases (diagnosed 2006-2010) and control subjects was based on insurance claims and prescription data provided by three large statutory health insurance funds in the Rhine-Main area (administrative region Darmstadt, Rhine Hesse). For all insured individuals (1,026,670 people aged 40 years or more), address-specific exposure to aircraft, road and railway traffic noise in 2005 was estimated. Odds Ratios (OR) were calculated using logistic regression analysis, adjusted for age, sex, local proportion of persons receiving unemployment benefits, and individual socioeconomic status (when available).
Results: Overall, results of our case-control study suggest a relationship between traffic noise exposure and the pathogenesis of myocardial infarction, stroke, heart failure and unipolar depression.

Keywords: Traffic noise, case-control study, cardiovascular diseases, depression

1. INTRODUCTION

An examination of the risks for cardiovascular diseases (myocardial infarction, stroke and heart failure/hypertensive heart disease), depression, and breast cancer (women only) associated with traffic noise exposure (road, railway, and aircraft) was conducted using a case-control study of health insurance claim data (secondary data) among residents of the Rhine-Main area.

2. METHODS

The study area included the administrative region of Darmstadt, as well as Mainz and Worms, and the areas of Mainz-Bingen and Alzey-Worms. The study population consisted of persons aged 40 years and older that were insured by three statutory health insurances (n=1,026.658). This presented 23.3% of residents over 40 years in the Rhine-Main area. Newly diagnosed cases (diagnoses) of cardiovascular diseases, depression, and breast cancer between 2006 and 2010 were identified using claims and prescription data of participating health funds. In total, we identified 19.632 incident cases of myocardial infarction, 25.495 cases of stroke, 104.145 cases of heart failure/hypertensive heart disease, 77.295 cases of depression, and 6.643 women with breast cancer. 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. The control group consisted of individuals that had no diagnosis of the respective disease in the particular period.

Each person was linked to traffic noise exposure levels from 2005 according to their address. Disease risk estimates (odds ratio and 95% confidence intervals [CI]) were calculated using logistic regression and adjusted for age, sex, education and occupation (via job classification code), and local proportion of persons receiving unemployment benefits.

The continuous sound levels for each traffic noise source were grouped in 5 dB categories. Cases and controls with noise exposure of less than 40 dB represented the reference category; persons characterized by a continuous sound pressure level below 40 dB and a nightly maximum level of more than 50 dB were grouped in a separate exposure category. Additionally, the exposure-risk relationship was examined by applying linear or third degree polynomial models to the continuous permanent sound levels.

In addition to the analyses of health claims data, a questionnaire-based survey was conducted among a sample of persons with cardiovascular diseases and corresponding controls (n = 8.540) to obtain information on potential confounders which are not or not fully included in the health insurance data (i.e., education, job, salary, smoking, height, and weight, alcohol consumption, working night shifts, noise exposure at work, and physical activities). Also, information concerning the location of the bedroom with respect to the nearest road and railway, as well as information regarding if the bedroom windows were opened at night, was collected for estimating the interior sound level.

The primary aim of the questionnaire-based survey was to evaluate the effect of important confounders, especially those related to health behaviors (i.e., Body Mass Index, smoking, alcohol consumption) and socioeconomic status. Therefore, the risk estimates for cardiovascular diseases obtained from the analyses of health claims data (i.e. without consideration of important confounders) were compared with risk estimates obtained from the survey data (i.e. data that included important confounders). The secondary aim was to compare the relationship between cardiovascular diseases and interior traffic noise sound levels with sound pressure levels from the house facade.

3. RESULTS

3.1 Cardiovascular disease risks

For a detailed description of the study results see Seidler et al. (2016; myocardial infarction risks) and Seidler et al. (2015 [in German]; with detailed tables). Briefly, aircraft noise levels above 60 dB were associated with a (not statistically significant) increased risk for myocardial infarction (odds ratio = 1.42 [95% CI 0.62-3.25]) and stroke (odds Ratio = 1.62 [95% CI 0.79-3.34). Including only myocardial infarction patients that had died by 2014 in the analysis revealed a significant association for aircraft noise exposure levels above 60 dB and myocardial infarction (odds ratio = 2.70 [95% CI 1.08 – 6.74]). In contrast, stroke risks for the subgroup of deceased stroke patients did not differ substantially.

With respect to the 24-hour continuous noise levels, we found relationships between the risk for cardiovascular diseases and road traffic noise (especially myocardial infarction and heart failure/hypertensive heart disease) and railway noise (myocardial infarction, stroke, as well as heart failure/hypertensive heart disease) which were more pronounced than for aircraft noise: the risk of myocardial infarction (see Seidler et al. 2016), stroke, or heart failure/hypertensive heart disease was significantly increased at 50 dB noise exposure (in some cases 55 dB) and continuously increased with higher sound levels. However, it is worth mentioning that an increased risk for stroke of 14% was observed at railway noise exposure levels of 45 to <50 dB when the maximum sound level was at least 20 dB over the continuous noise level (emergence analysis).

In contrast to road traffic and railway noise, there was no positive linear relationship between continuous aircraft noise levels and the risk for myocardial infarction or stroke. On the other hand, a linear exposure-risk relationship was (also) found for aircraft noise exposure and heart failure/hypertensive heart disease; here the risk was significantly increased by 1.6% per 10 dB increase in continuous noise level (OR per 10 dB = 1.016 [95% CI 1.003-1.030]).

3.2 The effect of nightly maximum sound levels on cardiovascular diseases

One important novel result of our study is the impact of the maximum aircraft noise levels at night on the cardiovascular system. First, the lowest aircraft exposure class examined includes persons with continuous noise levels <40 dB but with nightly maximum sound levels of >50 dB. For this particular group, we observed increased risk estimates in many analyses. Thus, if these persons were included in the reference category, the risks of aircraft noise exposure would be underestimated. Second, for

stroke and heart failure/hypertensive heart disease, significantly increased odds ratios suggest that nightly maximum sound pressure levels of above 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, requires nevertheless validation in future studies.

3.3 Breast cancer

There was an association between aircraft noise at night and breast cancer diagnoses. This observation reached statistical significance for the time period between 11 pm and 5 am in the highly exposed categories (OR =2.98 [95% CI 1.31-6.79]), however with few observed cases. There was no significant association between road and railway traffic noise and the risk of developing breast cancer.

3.4 Depression

There was a significant relationship between unipolar depression and all three noise sources (aircraft, road, and railway traffic noise). The risk for having a diagnosis of unipolar depression steeply increased in aircraft noise with a 17% risk increase at 50-55 dB 24-hour continuous noise levels; the risk increase was slightly weaker in road traffic noise and railway noise. However, we observed lower risk estimates for depression in categories with higher aircraft noise exposure as well as higher railway noise (comparable to an inversed U-curve), and a linear noise exposure-risk relationship did not constitute an appropriate model for aircraft noise and railway noise (but for road traffic noise).

3.5 The effects of sex and health insurance providers

Generally, we did not observe any systematic difference between males and females for all studied diseases when analyzed separately. Also, no systematic differences were found when data of a single health insurance provider were analyzed separately. This indicates that the results are externally valid, since the participating health insurance providers are characterized by different clienteles.

3.6 Simultaneous analysis of traffic noise sources (aircraft, road, railway traffic noise)

To ensure the comparability between each traffic noise source and to obtain information regarding the “isolated” effects of each traffic noise source, an additional analysis was conducted with all three traffic noise parameters included simultaneously in a logistic regression model for each disease. The exposure-response-relationship for each single noise source did not substantially differ when the model was adjusted for the other two noise sources.

3.7 Noise history: longtime unchanged physical address and “cumulative noise years”

The complex „reconstruction“ of historical noise exposure data for the years from 1996 to 2010 permitted the consideration of “constant” physical addresses and “cumulative noise years”. “Cumulative noise years” were calculated by using the “total noise dose” for a defined time period. Including only persons with a physical address that remained “constant” over many years in the analyses, tended to result in higher risk estimates for several diseases. In the highest category of continuous sound pressure due to aircraft noise (≥60 dB), risk estimates for stroke were increased when occupancy was at least 5 years; living 10 years or more at the same physical address increased risk estimates further but were not statistically significant.

Analyses of „cumulative noise years“ for the last 5 years before disease diagnosis, respectively before 2008 for controls showed continuously increased risk estimates for heart failure/hypertensive heart disease with significant results for the highest quartile. For breast cancer diagnoses, risk estimates were prominently (but not significantly) increased, with an odds ratio of 3.96 observed when only women living 5 years or longer at the same physical address were included in the analysis. No remarkable effect was observed for cumulative noise-years of aircraft, road traffic and railway traffic noise. For depression, significantly increased odds ratios were found in the highest noise-years categories (3rd and 4th quartile) for aircraft noise.

3.8 Bias from undetected or residual confounding? Results of the interview

To evaluate the possible distortion of the results by confounders that were not or only partially contained in the health claims data, 8,540 insured persons were questioned about their education, job, income, tobacco and alcohol consumption, weight and height, shift work, noise exposure at work, and physical activities. Contacted persons were a subset of the insurant pool provided by the health insurance providers, comprising 639 persons with an incident myocardial infarction, 612 with an

incident stroke, and 3,138 persons with incident heart failure or hypertensive heart disease (including persons with more than one diagnosis). 8,517 of the contacted persons represented controls for at least one case group. Only insured persons that were alive at the time of the interview could be included in the survey. The exclusion of the deceased led to considerably altered risk estimates in the analysis of claims data for myocardial infarction and stroke – diseases with a high mortality, indicating considerable selection bias. This may have been further aggravated by low response rates of 6% on average and the associated differential response behavior. In contrast, the selection of surviving persons did not lead to a substantial change in risk estimates for heart failure or hypertensive heart disease. With respect to the analyses of health claims data, odds ratios for heart failure or hypertensive heart disease diagnoses did not differ between persons participating in the interview and the whole study group. For this reason, interview data of cases with heart failure or hypertensive heart disease (but not myocardial infarction and stroke) 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).

Results show that odds ratios did not considerably differ from those based on the health claims data analyses when the models were adjusted for the individual socioeconomic status (Winkler index), tobacco and alcohol consumption, body-mass index, noise at work and physical activity. Thus, the results for heart failure/hypertensive heart disease do not appear to be substantially biased by insufficient consideration of socioeconomic status and lifestyle factors.

3.9 Subset analysis of insured persons with information on socioeconomic status

To further investigate effects of possible confounding by socioeconomic state on results obtained for myocardial infarction, stroke, unipolar depression and breast cancer, an additional subset analysis was performed only for those persons where information on individual socioeconomic status (education, job) was available in health funds data. Results of analyses suggest no considerable change in risk estimates assuming no substantial bias by insufficient controlling of socioeconomic status.

3.10 Comparison of interior and exterior sound levels on disease risks

When accounting for interior sound pressure levels on disease risk (secondary study aim), we found considerable increased odds ratios of being diagnosed with heart failure or hypertensive heart disease for all traffic noise sources in comparison to exterior sound pressure noise levels.

4. DISCUSSION

Overall, results of our case-control study based on health claims data and interview data suggest a relationship between traffic noise exposure and the pathogenesis of myocardial infarction, stroke, heart failure/hypertensive heart disease and unipolar depression. No significant association between continuous sound pressure levels and breast cancer diagnosis was found for all three traffic noise sources. However, a significant increased risk for breast cancer was found for nightly aircraft noise exposure between 11pm and 5 am.

4.1 Disease risks per 10 dB increase in continuous sound levels

The highest disease risks were observed for unipolar depression and significant results were obtained for all three traffic noise sources. For cardiovascular diseases, noise-related effects on myocardial infarction, stroke and heart failure/hypertensive heart disease were more pronounced for road and railway traffic noise in comparison to aircraft noise. The highest risk increase per 10 dB road traffic noise was 4.1% for unipolar depression, 2.8% for myocardial infarction, 2.4% for heart failure/hypertensive heart disease and 1.7% for stroke. For railway noise, the highest increase in risk was found for depression (with an inappropriate linear fit), 3.1% for heart failure/hypertensive heart disease, 2.3% for myocardial infarction and 1.8% for stroke per 10 dB increase in sound pressure level. For aircraft noise, the highest increase in risk was found for unipolar depression (with an inappropriate linear fit) and heart failure/hypertensive heart disease (1.6%).

4.2 Comparison of study results with those obtained from current reviews

Current reviews of Babisch (2014) and Vienneau et al. (2015) obtained slightly higher traffic noise-related risk estimates compared to our study results: the pooled analyses from Babisch (2014) suggest an increase in risk of 8% per 10 dB increase in road traffic noise. Vienneau et al. (2015) found a 4% increase in risk per 10 dB road traffic noise increase (L_{DEN}). For aircraft noise (L_{DEN}), the authors

found an increase in risk of 6%. In contrast, results of our study suggest risk estimates below 4% per 10 dB increase for all studied traffic noise sources on cardiovascular diseases. However, one has to keep in mind that we chose a starting point of 35 dB, which is considerably lower than the starting points chosen by Babisch (2014) and Vienneau et al. (2015). Therefore, the risk increases per 10 dB found in our study cannot be directly compared with the risk increases reported by Babisch (2014) and Vienneau et al. (2015). Further, our results for the relationship between unipolar depression and traffic noise are generally in accordance with the current level of knowledge, although a systematic review on this topic is still missing.

4.3 Importance of noise exposure duration

In accordance with previous studies (e.g. Huss et al. 2010, Floud et al. 2013), our results suggest the importance of considering the duration of noise exposure. Thereby, the calculation of "cumulative noise years" represents a promising approach which has not been applied in noise traffic research so far. However, the increases in risk have to be interpreted in a conservative way, because it is not possible to distinguish between a "real" effect of prolonged noise exposure over multiple years and a selection bias ("cause and effect-bias").

4.4 Exclusion of undetected or residual confounding

Considerable effort was made to test for undetected or residual confounding: 8.540 insured persons from the secondary data-based case-control study participated in the survey. For heart failure/hypertensive heart disease, the largest case group, risk estimates obtained from data analyses of survey participants were comparable to those obtained from the analyses of health claims data. Thus, substantial distortion of risk estimates obtained from health claims data analyses appears doubtful for heart failure or hypertensive heart disease in terms of the socioeconomic status and lifestyle factors collected in the survey.

4.5 The effect of interior noise levels on heart failure/hypertensive heart disease

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. Generally, this result suggests a causal effect between noise exposure and the development of heart failure or hypertensive heart disease. 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 level may better reflect the real noise exposure. Nevertheless, the interpretation is hindered by the fact that interior sound pressure levels might not represent the cause but the effect of noise exposure: Meaning, a relatively low interior sound pressure level might be the result of a traffic noise disturbed person sleeping with the windows closed at all times (though the constraint of closing the window may possibly lower the sleep quality).

4.6 Methodological strengths of the secondary data-based case-control study

As far as we know, this study permitted the first direct comparison of risk estimates for aircraft, road and railway traffic noise on the basis of a large data set of persons insured by statutory health insurance providers. Aircraft noise data were precisely generated for each physical address in the NORAH study. We at best included only newly diagnosed cases for the diseases myocardial infarction, stroke, heart failure/hypertensive heart disease, breast cancer and unipolar depression considering stationary and ambulant diagnoses. Further, we found empirical support for the biological importance of the separate consideration of maximum sound pressure levels. In particular, future research on health effects of noise should consider the possible insufficient representation of continuous sound pressure levels and should include separate consideration of maximum sound pressure levels.

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

Overall, risk estimates for road and railway traffic noise tended to be higher for myocardial infarction, stroke and heart failure/hypertensive heart disease compared to risk estimates for aircraft noise. However, uncertainties of risk estimations are higher in aircraft noise, as considerably less people were exposed to aircraft noise levels of 55 dB or more: only about 2% were exposed to aircraft noise levels of 55 dB or more, but 7% were exposed to railway noise levels and 26% to road traffic noise levels of 55 dB or more. Statistically significant increases in depression risks from aircraft noise were already found in lower to moderately noise exposed classes (in terms of an inverse U-curve); this result requires cautious interpretation.

5. CONCLUSIONS

In accordance with the literature, risk estimates for noise exposure found in this study are much lower as compared to risks for "known" harmful parameters such as tobacco consumption and increased body-mass on the development of cardiovascular diseases. However, a large part of the population is exposed to traffic noise which is -in accordance with this study – associated with (albeit low) increases in risks for cardiovascular diseases and unipolar depression. Based on the population-based occurrence of traffic noise exposure as well as the studied cardiovascular diseases even low increases in risks have relevance for the general population.

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