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Traffic noise and hypertension – results from a large case-control study

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ABSTRACT

Background: Environmental traffic noise is a potential cause of hypertension. We aimed to study the association between hypertension as recorded in health insurance claims data and the exposure to three sources of traffic noise (aircraft, road and rail).

Methods: This large case-control study was conducted among persons aged 40 and above in 2010 and living in the region around Frankfurt airport in Germany. Individual residential noise exposure for the index year 2005 was assessed using standard noise algorithms. Cases were all newly diagnosed cases of hypertension recorded in three large health insurances databases in the period 2006–2010. Controls had no hypertension diagnosis. Categorical and continuous analyses were conducted with binary logistic regression models adjusted for sex, age and residential area-based socioeconomic information.

Results: The main analysis included 137,577 cases and 355,591 controls. There were no associations with any of the traffic noise sources. Odds ratios (OR) per 10 dB noise increase were 0.99 (95% confidence interval: 0.98;1.01) for aircraft noise, and 1.00 (0.99;1.01) both for road and railway noise. Similarly, nighttime noise levels showed no associations with hypertension. Odds ratios were increased for the subgroup of newly diagnosed hypertension cases with a subsequent diagnosis of hypertensive heart disease: per 10 dB aircraft noise there was a 13.9% OR increase (6.0% for road traffic, 5.4% for rail traffic). Increases were also noted when we analyzed cases with a longer exposure-outcome time window.

Conclusion: Our results are suggestive of an association of noise exposure with clinically more severe hypertension diagnoses, but not with uncomplicated hypertension. The absence of individual confounder data, however, adds to the risk of bias. The results contribute to evidence on traffic noise as a cardiovascular risk factor.

1. Introduction

Traffic noise is a recognized environmental risk factor. Cardiovascular diseases, and particularly hypertension, have received the most attention as clinical outcomes possibly associated with diverse sources of traffic noise. Due to the high public interest in large-scale infrastructural developments such as airports, and the concomitant burdens on nearby communities, aircraft noise has been at the center of epidemiologic research (Eriksson et al., 2014; Evrard et al., 2015, 2016). However, noise associated with road and railway traffic is even more ubiquitous in many countries and particularly road traffic noise has been researched intensely over the past years (Babisch, 2014; Babisch et al., 2014; Meline et al., 2015; Recio et al., 2016b).

In Germany, the prevalence of hypertension in adults aged 18–79 years is about 30% for women, and 33% for men, with highest values of above 70% in the oldest age group (65–79 years) (Neuhauser and Sarganas, 2015). A cumulative hypertension incidence of 26.2% over a 12-year period was reported recently (Diederichs and Neuhauser, 2017). A possible relationship between noise and hypertension is often explained by a chronic stress response to noise, involving the sympathetic nervous system as well as endocrine responses (Babisch et al., 2014; Recio et al., 2016a).

Epidemiological studies on aircraft noise and hypertension have yielded inconsistent results. The cross-sectional "Hypertension and Exposure to Noise around Airports (HYENA)" study investigated associations between aircraft and traffic noise exposure and hypertension in 4861 people residing in the vicinity of six large European airports (Jarup et al., 2008). A 10 dB increase in nighttime aircraft

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noise levels increased the odds of hypertension by 14% (95%CI: 1.2;28.6%), while for average daytime road traffic noise exposure the OR per 10 dB increase was elevated by 9.7% (95%CI:0.3;20.1%). Sex-specific analyses of the HYENA data indicated significant increases in odds ratios with increasing road traffic noise for men but not for women, while for nighttime aircraft noise no sex differences were apparent. The absolute blood pressure increase due to nighttime aircraft noise measured among study participants was in the order of 6 mmHg systolic and 7 mmHg diastolic pressure. In Sweden, Eriksson (Eriksson et al., 2010) found no overall association between aircraft noise exposure and incident hypertension in a cohort of residents near airports; in subgroup analyses, non-smoking men, but not women showed a statistically significant relative risk increase of 21% (95%CI 5;39%) per 5 dB. Looking at traffic noise overall including aircraft noise, the "Residential Environment and Coronary heart Disease (RECORD)" study indicated increased hypertension risks associated only with combined road, rail and air traffic noise at the workplace, but not in the residential setting (Meline et al., 2015). A 2009 review estimated a pooled 13% risk increase per 10 dB (95%CI 0;28%) of aircraft noise based on five studies, but also noted limited validity of measurements taken in several of the included studies (Babisch and Kamp, 2009). A recent meta-analysis summarized data from three cross-sectional studies and one cohort study and computed an OR of 1.63 for hypertension among residents with aircraft noise exposure (Huang et al., 2015). This increase was significant only for men.

Overall the evidence appears inconclusive, but with a tendency towards a positive association between hypertension and aircraft traffic noise. Looking at road traffic only, van Kempen and Babisch (van Kempen and Babisch, 2012) reported a statistically significant 3.4% increased OR per 5 dB based on the pooling of 24 studies.

We therefore investigated the association between physician-diagnosed hypertension as recorded in health insurance claims data and the exposure to three sources of traffic noise in a large case-control study, deriving study participants from a database of about 1 million persons with individual health and residential noise information. The current analysis is embedded in the NORAH (noise-related annoyance, cognition and health) case-control study conducted in the region around Frankfurt airport in Germany.

2. Materials and methods

We analyzed case-control data from health insurance claims databases for persons residing in the vicinity of Frankfurt airport, using individual residential noise data for three sources of noise: air, rail and road traffic. The detailed study design and methods are available from (Seidler et al., 2017, 2016a, 2016b). We provide a summary overview of the general methods and a detailed description of specific approaches to hypertension.

2.1. Study population

The study population from which cases and controls were identified included 1,026,670 persons aged 40 years or above in 2010 who were members of one of three large statutory health insurance funds in the period 2005–2010, representing some 23% of all residents of this age group in the study region situated around Frankfurt airport. The study region included all administrative areas with substantial aircraft noise exposure of the population.

2.2. Cases of hypertension

Requirements for the classification as a newly diagnosed case were a recorded main or secondary hospital discharge diagnosis of hypertension (ICD 10: I10.-), or two confirmed I10.- diagnoses in the ambulatory setting within a time period of 12 months. A further requirement was that no hypertension diagnosis was recorded in the 12 months prior to the first diagnosis date during the study period. Thus, 137,577 individuals qualified as cases based on this definition.

Using a predefined restrictive case definition, we additionally analyzed all persons with a confirmed hypertension diagnosis as above who also had a further diagnosis of hypertensive heart disease (I 11.-) during the study period (n =7031).

2.3. Controls

Control subjects were all persons in the claims database without any new or prevalent hypertension diagnosis as described above during the study period. They had to be aged 40 years or above in 2010 and have an insurance period of at least 12 months in one of the participating health insurance funds. 355,591 persons served as controls for the main analysis.

2.4. Exposure assessment

Extensive steps to map aircraft, rail and road noise data to individual residential addresses were undertaken. Immission sites per noise source were selected (aircraft: center of building; road, rail: main exposed outer surface of residence) and average and maximum noise levels for the index year 2005 calculated based on most appropriate data sources. These were historical radar data and input data provided by German Flight Safety Operator (DFS) for aircraft noise and traffic count or operation data from relevant official sources (traffic count data; German Railway Operator and Federal Railway Authority). Noise models included sound propagation scenarios from source to immission site as well as data on noise barriers and walls along car traffic and railway routes, covering day- and nighttime exposures. All calculations were done using national or EU algorithms for noise mapping. Aircraft noise data were compared for consistency with measurements from local monitoring stations (Möhler, 2016). A graphical overview of aircraft noise exposure and the geographical study area has been published earlier (Seidler et al., 2017).

Several independent databases were designed to assure that no single institution had access to personal identifiers (addresses) together with sensitive health claims data. The data linkage office in Bremen merged address-specific noise and address data from health insurances (one insurance fund performed this step independently). Address data were then replaced by study ID, and the merged data set forwarded to the data analysis office in Dresden where claims information from the three health insurances was linked to the noise data via the common study ID. All procedures followed a strict data protection protocol approved beforehand by federal and state authorities.

2.5. Data analysis and adjustment for confounding

We initially performed extensive descriptive analyses of the large case-control data set, using 2005 as the index year for exposure, and the period 2005–2010 as analysis window for relevant health and confounder information. We then built logistic regression models to calculate OR and 95%CI with hypertension as outcome and noise exposure per 5 dB category as explanatory variable, using the 24 h average noise exposure (LpAeq. 24 h) category < 40 dB as reference. 24 h average noise exposure was also modelled as a continuous variable and presented as OR per 10 dB increase in noise exposure. Separate models were run to study nighttime (22–6 h) traffic noise. We included sex (male, female) and age (in 5-year categories, starting at 40 years) in the models. Data on education and job title were available for a proportion of cases (32.1%, n =44,188) and controls (50.9%, n =180,881) in the health insurance funds database. We included this information in the final regression model, along with an area-based measure of socioeconomic status (SES) (= proportion of persons on long-term unemployment benefit) available for all study participants. A separate analysis was restricted to all persons for whom SES informa-