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Transportation noise and incidence of hypertension

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A B S T R A C T

Background: The evidence on exposure to transportation noise and development of hypertension is inconclusive, mostly because of a lack of high quality studies of longitudinal design.

Objectives: This cohort study aimed at investigating the association between exposure to road traffic, railway or aircraft noise and incidence of hypertension. We also assessed effects of varying lengths of exposure as well as of multiple sources of exposure.

Methods: Based on the residential histories of a cohort of 4854 men and women from Stockholm County, we estimated the residential exposure to road traffic, railway and aircraft noise in 1, 5 and 10 year time-periods. Hypertension was assessed by blood pressure measurements, information from questionnaires and hospital diagnoses. Extensive information on potential confounders was available from repeated questionnaires and registers. Hazard Ratios (HR) and 95% confidence intervals (CI) of hypertension related to noise exposure was computed from Cox regression models.

Results: We observed a positive association between aircraft noise exposure and incidence of hypertension with a HR of 1.16 (95% CI 1.08–1.24) per 10 dB L_{den} 5 years preceding the event. No clear differences in risk were indicated between the three exposure time windows. Road traffic and railway noise were not associated with incidence of hypertension during any of the exposure periods. There appeared to be a particularly high risk of hypertension among persons exposed to both aircraft and road traffic noise ≥ 45 dB L_{den} with an HR of 1.39 (95% CI 1.14–1.70).

Conclusions: Exposure to aircraft noise may result in increased risk of hypertension.

1. Background

Transportation noise is a common environmental exposure leading to at least 1 million healthy life-years lost each year in Western Europe, attributed mostly to sleep disturbance and annoyance, but cardiovascular diseases also contribute substantially (WHO, 2011). Hypertension is a commonly studied outcome in relation to road traffic noise, however, fewer studies are available on aircraft and railway noise. Moreover, almost all of the available studies are of cross-sectional design. A recent systematic review, performed within the framework of the development of new WHO Environmental Noise Guidelines for the European Region found a statistically significant association between road traffic noise and prevalence of hypertension with relative risk (RR) of 1.05 and 95% confidence interval (CI) 1.02–1.08 per 10 dB L_{den} based on meta-analysis of 26 cross-sectional studies (van Kempen et al., 2018). This was, however, not confirmed in a cohort study, reporting an incidence rate ratio (IRR) of 0.97 (95% CI 0.90–1.05) per 10 dB L_{den} Sørensen et al., 2011). Aggregating data from nine cross-sectional studies, aircraft noise tended to be positively associated with hypertension (RR 1.05; 95% CI 0.95–1.17) (van Kempen et al., 2018). A longitudinal study of aircraft noise and incidence of hypertension showed a tendency of a positive association for men, RR 1.17 (95% CI 0.90–1.51), but not for women, RR 0.85 (95% CI 0.62–1.15) per 10 dB L_{den} (Eriksson et al. 2007, 2010). Considering railway noise, the WHO-review included four cross-sectional investigations together showing a not statistically significant association for prevalence of hypertension, RR 1.05 (95% CI 0.88–1.26). Furthermore, no clear association was found in the only cohort study on railway noise and incidence of hypertension, RR 0.96 (95% CI 0.88–1.04) per 10 dB L_{den} (Sørensen et al., 2011). Since the WHO review, some additional longitudinal studies on transportation noise and hypertension have been published showing diverging results (Carey 2016; Dimakopoulou 2017; Fuks 2017; Zeeb 2017 Héritier et al. 2017).

The relation between transportation noise and adverse cardiovascular effects is often explained by the general stress model, i.e. that noise subconsciously may activate the sympathetic nervous system as well as the endocrine system (Hypothalamic- Pituitary- Adrenal, HPA, axis), thereby increasing the allostatic load (Babisch, 2002). Moreover,

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higher saliva cortisol levels have been reported in noise-exposed subjects living near airports (Selander et al., 2009). However, detrimental effects of noise may also be mediated via sleep disturbances (Griefahn et al., 2008; Van Cauter et al., 2008). Due to differences in the characteristics of noise, such as temporality and frequency spectra, exposure to noise from different sources may have different health impact. For example, studies on annoyance have shown that aircraft noise is a stronger environmental stressor than road traffic and railway noise (Miedema and Oudshoorn, 2001). With regard to hypertension, few studies have simultaneously investigated source specific effects (Zeeb et al., 2017), and it is not clear if combined exposure to several sources of noise modifies the effect. Furthermore, the induction time for noise-induced hypertension is not known.

This cohort study aimed to assess impact of transportation noise from different sources including road traffic, railways and aircraft on incidence of hypertension among residents in five municipalities within Stockholm County, Sweden. We have previously reported on associations between aircraft noise and incidence of hypertension from this cohort (Eriksson et al., 2007, 2010), however, we now extend the analyses to incorporate results also for road traffic and railway noise using a newly developed methodology for exposure assessment. Furthermore, we also investigated the impact of different induction periods of transportation noise on the development of hypertension as well as assessed the effects of exposure to noise from multiple sources.

2. Material and methods

2.1. Study population

This cohort study is based on data from the Stockholm Diabetes Prevention Program, conducted in Stockholm County between 1992 and 2006 (Eriksson et al., 2008). The primary aim of the program was to study risk factors for type 2 diabetes as well as to implement and evaluate methods for diabetes prevention. The study participants were recruited from five suburban municipalities in Stockholm County between 1992 and 1998 and followed for an average of nine years.

As a result of the original design, approximately half of the study participants (52%) had a family history of diabetes (FHD). FHD was defined as known diabetes in at least one first-degree relative (mother, father, sister or brother), or in at least 2 s-degree relatives (grandparent, uncle or aunt). The other half of the population consisted of persons without FHD who were frequency-matched on age and sex to those with FHD. In total, 7949 participants without previously diagnosed diabetes took part in the baseline investigation. Between 2002 and 2006, all participants except those who had died or moved out of Stockholm County were invited to a follow-up investigation. Overall, the SDPP cohort includes 5712 subjects with data from both the baseline and follow-up investigations, corresponding to 72% of those who participated in the baseline survey (Fig. 1).

At both investigations, the participants filled out a questionnaire and took part in a clinical examination by trained staff. The examination included measurements of weight, height, waist circumference and blood pressure. From the baseline questionnaire, we obtained information on several individual characteristics, including age, sex, education, smoking status, alcohol intake, physical activity, dietary habits, psychological distress, shift work, insomnia and job strain. In addition, the follow-up questionnaire enquired about noise annoyance and noise sensitivity.

Some restrictions were made of the study population in order to create a cohort free of hypertension at baseline (Fig. 1). All participants who reported antihypertensive treatment (n = 136), hypertension diagnosed by a doctor before the baseline or had hypertension according to National Patient Register (n = 38) and/or those with a blood pressure equal to or exceeding 140/90 mmHg at the baseline examination (n = 503) were excluded, as well as subjects with missing data on both treatment and measured blood pressure (n = 34). Individuals with myocardial infarction (MI) and cardiac arrhythmias (CA) in the National Patient Register prior to the baseline investigation were also excluded since these conditions have common treatment strategies as hypertension (n = 25). Furthermore, those with MI or CA during the follow-up were excluded from the risk set at day of diagnoses (n = 62). Finally, at the stage of analyses, we excluded those with incomplete covariate information (n = 122). Thus, the final analytical sample included 4854 participants.

2.2. Exposure assessment

In order to assess the time-weighted average transportation noise exposure for our study participants, we obtained information on their residential address history from the Swedish Taxation Authority. This included information on each address where the participants had lived during the follow-up period, starting from 1990 and with precise dates of changes in residency. Based on residential address coordinates, we then assessed the exposure to noise from road traffic, railways and aircraft at the most exposed façade of each building. In this, we used a newly developed database incorporating information of relevance for calculation of noise exposure in Stockholm County which we obtained from several national, regional and local authorities. The database covers the time-period 1990–2010 and typically contains information on terrain, ground surface, building density, traffic flows on roads (> 1000 vehicles/24 h) and railway lines, speed limits as well as aircraft noise contours around the two major airports in Stockholm (Arlanda and Bromma).

For road traffic and railway noise, we modelled the 24-h A-weighted equivalent continuous sound level ($L_{Aeq,24h}$) based on the information in our database and a simplified version of the Nordic prediction method. The methodology used has been validated against the full Nordic prediction method modelled by commercially available software (Ögren and Barregard, 2016). Noise screen data were not included in noise exposure assessment because we generally lacked information on year of construction. Traffic flow data were available for the years 1990, 1995, 2000, 2005, 2010. Therefore, to obtain annual noise levels for the whole period 1990–2010 at each address point we made linear interpolation between the years with information. We then recalculated the L_{Aeq}-levels to L_{den} with penalties of (+5 dB) and (+10 dB) for evening and night events, assuming a 24 h traffic flow distribution of 75/20/5% for the day, evening and night period, respectively, for road traffic and the exact 24 h distribution for separate segments of the railway lines (Murphy and King, 2010).

Information on aircraft noise exposure was obtained as noise contours around Arlanda and Bromma for the years 1995, 2000, 2005 and 2010. For the year 1990, we assumed the same noise level as for 1995 since there were no major structural changes at either of the two airports during this time-period. The noise contour data ranged from 45 to 70 dB L_{den} around Arlanda and from 40 to 70 dB L_{den} around Bromma. By superimposing the noise contour data on a layer of buildings where our participants had lived, each address could be assigned a relevant noise level. For each address point we made linear interpolation between the years with noise data to obtain annual levels of aircraft noise exposure for the whole period 1990–2010.

2.3. Outcome definition

To identify cases of hypertension in our cohort, we combined self-reported information from the follow-up questionnaire, data on measured blood pressure from the clinical examination at the follow-up investigation and data on hospitalisation history from the National Patient Register, held by the National Board of Health and Welfare. Blood pressure at the clinical examination was measured once, in a sitting position after about 5 min rest, with a triple cuff hand aneroid sphygmomanometer. The cut-off for measured blood pressure was set in accordance with the World Health Organization definition of