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Residential exposure to traffic noise and risk of incident atrial fibrillation: A cohort study

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ABSTRACT

Background: Studies have found long-term exposure to traffic noise to be associated with higher risk for hypertension, ischemic heart disease and stroke. We aimed to investigate the novel hypothesis that traffic noise increases the risk of atrial fibrillation (A-fib).

Methods: In a population-based cohort of 57,053 people aged 50–64 years at enrolment in 1993–1997, we identified 2692 cases of first-ever hospital admission of A-fib from enrolment to end of follow-up in 2011 using a nationwide registry. The mean follow-up time was 14.7 years. Present and historical residential addresses were identified for all cohort members from 1987 to 2011. For all addresses, exposure to road traffic and railway noise was estimated using the Nordic prediction method and exposure to air pollution was estimated using a validated dispersion model. We used Cox proportional hazard model for the analyses with adjustment for lifestyle, socioeconomic position and air pollution.

Results: A 10 dB higher 5-year time-weighted mean exposure to road traffic noise was associated with a 6% higher risk of A-fib (incidence rate ratio (IRR): 1.06; 95% confidence interval (95% CI): 1.00–1.12) in models adjusted for factors related to lifestyle and socioeconomic position. The association followed a monotonic exposure–response relationship. In analyses with adjustment for air pollution, NO_x or NO_2, there were no statistically significant associations between exposure to road traffic noise and risk of A-fib; IRR: 1.04; (95% CI: 0.96–1.11) and IRR: 1.01; (95% CI: 0.94–1.09), respectively. Exposure to railway noise was not associated with A-fib.

Conclusion: Exposure to residential road traffic noise may be associated with higher risk of A-fib, though associations were difficult to separate from exposure to air pollution.

1. Introduction

Traffic noise has become an almost inevitable environmental exposure with rising levels following urbanization. Exposure to traffic noise has generally been associated with cardiovascular disease, including hypertension (van Kempen and Babisch, 2012), ischemic heart disease (Vienneau et al., 2015) and stroke (Sorensen et al., 2011). No studies have investigated whether exposure to traffic noise is associated with atrial fibrillation (A-fib).

A-fib is the most common type of arrhythmia (Schnabel et al., 2015), and associated with both increased cardiovascular morbidity and mortality (Go et al., 2001). Although A-fib affects approximately 4% of the population over 50 years of age with rising prevalence, knowledge on the aetiology behind the development of A-fib is sparse (Andrade et al., 2014; Miyasaka et al., 2006).

Exposure to noise during the night is thought to be particularly hazardous, as night-time noise at normal urban levels (45–65 dB) is associated with reduced sleep quality and duration (Miedema and Vos, 2007). Noise also acts as a stressor, with hyperactivity of the autonomic nervous system and activation of the hypothalamus–pituitary–adrenal (HPA) axis, leading to a cascade of effects. This includes changes of atrial electrophysiology, which may be one pathway through which noise may initiate A-fib (Chen et al., 2014; Shen and Zipes, 2014). Another potential pathway is noise-induced effects on the immune system, as systemic inflammation in various studies has been associated with

Abbreviations: A-fib, atrial fibrillation; HPA, hypothalamus–pituitary–adrenal; dB, decibel; IRR, incidence rate ratio; L_{den}, equivalent continuous noise level day–evening–night.

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increased risk A-fib (Dewland et al., 2015; Issac et al., 2007). Both stress and disturbance of sleep may affect the immune system. Studies have found exposure to acute and chronic stressors, e.g. exams and stressful life events, to be associated with an impaired immune system (Picardi et al., 2009; Segerstrom and Miller, 2004) and release of cortisol, as a result of an active HPA axis, has been found to impair the function of glucocorticoid receptors and thereby contribute to systemic inflammation (Bellavance and Rivest, 2014; Silverman and Sternberg, 2012; Wolkow et al., 2015). Sleep is known to have a strong regulatory influence on the immune system (Ali and Orr, 2014; Gomez-Gonzalez et al., 2012), and disturbance of sleep has been associated with impairment of the immune system, including reduced post-vaccination antibody titers, changed numbers of circulating white blood cells and increased production of pro-inflammatory molecules (Aho et al., 2013; Besedovsky et al., 2012; Irwin et al., 2015; Lange et al., 2011). Lastly, release of cortisol may increase the glycogen concentration within atrial myocytes, which is suggested to be a risk factor for A-fib (Embi and Scherlag, 2014; Zhang et al., 2015).

The aim of the present study was to investigate the association between residential exposure to road traffic and railway noise and risk for incident A-fib in a large prospective cohort.

2. Methods

2.1. Study population

The study was based on the Diet, Cancer and Health study, into which 57,053 residents of Copenhagen or Aarhus aged 50–64 years were enrolled between 1993 and 1997 (Tjonneland et al., 2007). The participants had to be born in Denmark with no history of cancer at the time of enrolment. At enrolment, each participant completed self-administered, interviewer-checked, lifestyle questionnaires covering smoking habits, diet, alcohol consumption, physical activity and education. Height, weight, and waist circumference were measured by trained staff members according to standardized protocols. The study was conducted in accordance with the Helsinki Declaration and approved by the local Ethics Committees and written informed consent was obtained from all participants.

2.2. Identification of outcome

Cases who developed A-fib between baseline and death, emigration, or end of follow-up (31st December 2011) were identified by linking the unique personal identification number of each cohort member to the nationwide Danish National Patient Register (Lynge et al., 2011). Since 1977 patients diagnosed in-hospital have been registered in The Danish National Patient Register and coded using the International Classification of Diseases 8th (until 1994) and 10th (from 1994) Revision (ICD-8 and ICD-10) coding system. Since 1995 diagnoses from emergency rooms and outpatient visits have also been registered (Lynge et al., 2011). Cases were identified using ICD-8 codes 427.93 and 427.94 and ICD-10 code I48.9. We excluded participants with a diagnosis of A-fib before enrolment, and considered only the first hospitalization of A-fib.

2.3. Exposure assessment

Complete residential address history between 1st of July 1987 and event or end of follow-up at 31st December 2011 was available for 93% of the cohort members using the Danish civil registration system (Pedersen, 2011). Exposure to road traffic noise was calculated for the years 1990, 1995, 2000, 2005 and 2010 using SoundPLAN (http://www.soundplan.dk), which implements the joint Nordic prediction method for road traffic noise; a method which has been the standard method for noise calculation in Scandinavia since the introduction in 1981 (Bendtsen, 1999). Traffic noise for the year 1990 was used as a proxy for the period 1st of July 1987 to 30th of June 1992, the year 1995 was a proxy of the period 1st of July 1992 to 30th of June 1997, etc.

Equivalent noise levels were calculated for each address on the most exposed facade of the building using the following input variables; geographical coordinates and height (corresponding to floor level) of each address; road links with information on yearly average daily traffic, vehicle distribution (of light and heavy vehicles), travel speed and road type (motorway, express road, road wider than 6 m, road <6 m and >3 m, and other road); and building polygons for all buildings including information on building height. Information on building polygons was obtained from the Danish Geodata Agency, and is based on different sources including a laser scan of Denmark. First and second order reflections from buildings (3 dimensional) have been included in the modeling and reflection loss for buildings was set to 1 dB. No information was available on noise barriers. We obtained traffic counts for all Danish roads from a national road and traffic database (Jensen et al., 2009). This database is based on a number of different traffic data sources: 1) Collection of traffic data from the 140 Danish municipalities with most residents, covering 97.5% of the addresses included in the present study. Included roads typically have >1000 vehicles per day and are based on traffic counts as well as estimated/modeled numbers. Traffic data represents the period from 1995 to 1998; 2) Traffic data from a central database covering all the major state and county roads; 3) Traffic data for 1995–2000 for all major roads in the Greater Copenhagen Area; 4) traffic data for 1995 for all roads based on a simple method where estimated figures for distribution of traffic by road type and by urban/rural zone were applied to the road network and subsequently calibrated against known traffic data at county level. New roads were included in the calculations from the year they opened. Values below 40 dB were set to 40 dB, because we considered this as a lower limit of road traffic noise.

Exposure to railway noise was calculated for all addresses using SoundPLAN, with implementation of NORD2000. The input variables for the noise model were receptor point (geographical coordinate and height), railway links with information on annual average daily train lengths, train types, travel speed (obtained from BaneDanmark, which is operating and developing the Danish state railway network), and building polygons for all Danish buildings, including screening from buildings (as described for road traffic noise). The daily train lengths are given for 1997 and 2011. All noise barriers along the railway are included in the model.

In estimating noise we assumed that the terrain was flat, which is a reasonable assumption in Denmark, and that urban areas, roads, and areas with water were hard surfaces whereas all other areas were acoustically porous. Road traffic and railway noise were calculated as the equivalent continuous A-weighted sound pressure level (L_{Aeq}) at the most exposed facade of the dwelling at each address for the day (L_d; 07:00–19:00 h), evening (L_e; 19:00–22:00 h) and night (L_n; 22:00–07:00 h) and these where expressed as an indicator of the overall noise level during 24 h, as L_{den} (day, evening, night) by applying a 5-dB penalty for the evening and a 10-dB penalty for the night. L_{den} was used as estimator of road traffic and railway noise in all statistical analyses.

The noise impact from all Danish airports and airfields was determined from information about noise zones (5-dB categories) obtained from local authorities. Airport noise was transformed into digital maps and linked to each address by geocodes.

The concentration of traffic-related air pollution (nitrogen dioxide; NO_2 and nitrogen oxides; NO_x) was calculated using the dispersion model AirGIS, for each year (1987–2011) at each address at which the cohort members had lived. The Danish AirGIS modelling system calculates air pollution as the sum of regional background, urban background, and local street level calculated with the Operational Street Pollution Model (OSPM) (Berkowicz et al., 2008; Jensen et al., 2001; Kakosimos et al., 2010). Data of street configurations are the physical environment around the geocoded address, and the data are describing average height of building in the street, the width of the street from facade to