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Residential exposure to traffic noise and risk for non-hodgkin lymphoma among adults



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ABSTRACT

Exposure to traffic noise may result in stress and sleep disturbances, which have been associated with impairment of the immune system. People with weakened immune systems are known to have a higher risk for non-Hodgkin lymphoma (NHL). We aimed to determine whether traffic noise was associated with risk for NHL in a nationwide case-control study. We identified 2753 cases aged 30–84 years with a primary diagnosis of NHL in Denmark between 1992 and 2010. For each case we selected two random population controls, matched on sex and year of birth. Road traffic and railway noise were calculated, and airport noise was estimated for all present and historical residential addresses of cases and controls from 1987 to 2010. Associations between traffic noise and risk for NHL were estimated using conditional logistic regression, adjusted for disposable income, education, cohabiting status and comorbidity. We found that a 5-year time-weighted mean of road traffic noise above 65 dB was associated with an 18% higher risk for NHL (95% confidence interval (CI) 1.01–1.37) when compared to road traffic noise below 55 dB, whereas for exposure between 55 and 65 dB no association was found (odds ratio: 0.98; 95% CI: 0.88–1.08). In analyses of NHL subtypes, we found no association between road traffic noise and risk for T-cell lymphoma, whereas increased risks for B-cell lymphoma and unspecified lymphomas were observed at exposures above 65 dB. In conclusion, our nationwide study may indicate that high exposure to traffic noise is associated with higher NHL risk.

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1. Introduction

Non-Hodgkin lymphoma (NHL) is a diverse group of malignancies originating from lymphoid tissue. Though incidence rates have more than doubled since 1960, with stabilization around year 2000 (Sandin et al., 2006), the etiology of most types of NHL remains largely unknown. One well-established risk factor is suppression of the immune system (Shankland et al., 2012), and several studies have shown an increased risk of NHL among HIV patients, organ recipients receiving immunosuppressive drugs and patients with autoimmune diseases (Fallah et al., 2014; Grulich et al., 2007). In addition, a growing number of studies have indicated that genetic variants related to the immune system, including immune recognition and regulation, is associated with increased risk for NHL (Carvalho et al., 2012; Cerhan et al., 2014; Nieters et al., 2011). Also, circadian genes have been associated with NHL, possibly through their influence on immune regulation

(Hoffman et al., 2009), and some epidemiological studies have indicated that night-shift workers are at higher risk for NHL (Lahti et al., 2008; Parent et al., 2012). Due to the apparent strong relationship between the immune system and development of NHL, it is conceivable that also more moderate disturbances of the immune system may increase the risk for NHL.

Exposure to traffic noise has been associated with cardiovascular disease and diabetes (Babisch, 2014; Sorensen et al., 2013a). One of the proposed mechanisms is disturbance of sleep, as nighttime exposure to traffic noise at normal urban levels has been associated with sleep disturbances, including short sleep duration and reduced sleep quality (Basner et al., 2011; Miedema and Vos, 2007). Sleep is known to have a strong regulatory influence on the immune system (Ali and Orr, 2014; Gomez-Gonzalez et al., 2012), and deprivation of sleep has been associated with impairment of the immune system, e.g. reduced post-vaccination antibody titers have been demonstrated following sleep restriction at the night after vaccination (Lange et al., 2003; Spiegel et al., 2002). Sleep deprivation has also been associated with various aberrations in the immune system, including changed numbers of circulating white blood cells and increased production of pro-inflammatory

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molecules (Aho et al., 2013; Irwin et al., 2006, 2008).

Another potential adverse health effect of noise is that traffic noise can trigger a stress response, causing hyperactivity of the sympathetic autonomic nervous system, activation of the hypothalamus-pituitary-adrenal axis and increased levels of cortisol (Munzel et al., 2014; Selander et al., 2009a). It is also possible that noise-induced stress may affect the immune system, as some studies have suggested that exposure to various acute and chronic stressors, e.g. exams and stressful life events, is associated with an impaired immune system (Segerstrom and Miller, 2004).

In the present nationwide case-control study, we aimed to assess the association between exposure to traffic noise and risk for NHL within the adult population of Denmark.

2. Methods

2.1. Study population

Cases were identified using the Cancer Registry (Gjerstorff, 2011). Eligible cases were Danes between 30 and 84 years of age with a primary diagnosis of NHL between 1992 and 2010. NHL cancer diagnoses were identified as morphology codes 9590-9596 and 9670-9729 according to the third edition of International Classification of Diseases for Oncology (ICD-O-3) (World Health Organization, 2000). We further required that cases were born in Denmark, had no previous diagnosis of cancer (except non-melanoma skin cancer), and lived at a geocodable address in Denmark at the time of diagnosis.

For each case two random controls, matched on sex and year of birth, were selected from the Civil Registration System. Controls which were dead, emigrated or diagnosed with cancer (except non-melanoma skin cancer) before diagnosis date of their matched case were excluded and substitute controls, matched on sex and year of birth, and under risk at age at diagnosis of the matched case, were sampled from surplus controls from the present study as well as from random population controls sampled for other ongoing studies. All controls in the final sample were required to be alive, free of cancer (except non-melanoma skin cancer), born in Denmark and living at a geocodable address in Denmark at time of diagnosis of matched case.

2.2. Socioeconomic position and comorbidity

Following information on individual socioeconomic position (SEP) was obtained by linkage to Statistics Denmark (Baadsgaard and Quitzau, 2011): highest attained education, cohabiting status, and individually disposable income determined as household income after taxation and interest per person, adjusted for number of household persons and divided into quintiles based on Danish background population (age-standardized). Education was assessed at date of diagnosis, whereas the other SEP variables were assessed one year before diagnosis.

Information on comorbidity was obtained from the Danish National Patient Registry, which holds information on all non-psychiatric hospital admissions since 1977 and on all outpatient and psychiatric hospital contacts since 1995 (Lyngé et al., 2011). The data include medical diagnoses (ICD-8 until 1993 and ICD-10 from 1994 onwards), surgical procedures, and date of activity. Using this we generated Charlson Comorbidity index (0, 1 and ≥ 2) for all based on all relevant diagnosis until one year before index date.

2.3. Exposure

Residential address history for all cases and controls between

1987 and index date was collected using the Civil Registration System. Road traffic noise exposure was calculated at the most exposed facade for all present and historical addresses using SoundPLAN, a software implementing the joint Nordic prediction method for road traffic noise (Bendtsen, 1999). Input variables were: geographical coordinate, road links with information on annual average daily traffic, vehicle distribution, travel speed and road type; and building polygons for all Danish buildings. We obtained traffic counts for all Danish roads with more than 1000 vehicles per day from a national database (Jensen et al., 2009). We assumed flat terrain, and that urban areas, roads and areas with water were hard surfaces and all other areas acoustically porous. No information was available on noise barriers. Road traffic noise was expressed as L_{den} (day, evening and night).

Exposure to railway noise was calculated for all addresses using SoundPLAN, with implementation of NORD2000. Input variables were geographical coordinate, railway links with information on annual average daily train lengths, train types, travel speed; building polygons and noise barriers along the railway. Railway traffic noise was expressed as L_{den} at the most exposed facade. The noise impact from all Danish airports and airfields was determined from information about noise zones (5 dB categories) obtained from local authorities and transformed into digital maps and linked to each address.

Ambient air pollution (NO_2) was calculated for each year at each address using AirGIS, which calculates a sum of local, urban and regional contributions (Berkowicz et al., 2008). Input data included traffic data as described above, emission factors, street and building geometry, and meteorological data (Jensen et al., 2009; Ketznel et al., 2011). We used NO_2 as a proxy of traffic-related air pollution as it has been shown to correlate closely with particulate matter, including ultrafine particles and PM_{10} in Danish streets (Ketznel et al., 2003).

2.4. Statistical analyzes

We used conditional logistic regression. For each person, exposure to road traffic noise was modeled as time-weighted 1- and 5-years means preceding index date, taking the complete migration history in these periods into account. The assumption of linearity of road traffic noise was evaluated visually and by linear spline models with boundaries placed at quartiles for cases. We found that the association between road traffic noise and NHL deviated statistically significant from linearity and, therefore, all analyzes were performed using following categories: < 55 , 55–65 and ≥ 65 dB. The reference was set to < 55 dB, as this level is often used in studies of traffic noise and health as a reference cut-point (Babisch, 2014), and the 65 dB cut-point was set to select a group of highly exposed persons ($\approx 90\%$ percentile). For airport and railway noise we used noise exposure at index date address in categories for airport noise of above and below 55 dB and for railway noise in categories of: 0 dB, 0–55 dB and ≥ 55 dB.

We calculated odds ratios (OR) and 95% confidence intervals (CI) for risk for NHL associated with exposure to traffic noise, with adjustment for age and sex (by design), education (basic or high school; vocational education; higher education; unknown), disposable income (quintiles), cohabitation status (married/cohabiting, single, widow/widower or divorced) and Charlson comorbidity index (0, 1 or ≥ 2). In further analyzes, we performed additional adjustment for air pollution. As a *post-hoc* sensitivity analysis, we divided the highest exposure category (above 65 dB) into two categories: 65–70 dB and > 70 dB, and calculated adjusted ORs. ORs were estimated for NHL subtypes: B-cell lymphoma (ICD-O-3: 9670–9699, 9728), T-cell lymphomas (ICD-O-3: 9700–9719, 9729) and unspecified lymphomas (ICD-O-3: 9590-9596, 9727). We used SAS version 9.2 (SAS Institute, North Carolina, USA).

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