Environment International 85 (2015) 238–243

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

Exposure to long-term air pollution and road traffic noise in relation to cholesterol: A cross-sectional study

Mette Sørensen [a,*], Dorrit Hjortebjerg [a], Kirsten T. Eriksen [a], Matthias Ketzel [b], Anne Tjønneland [a], Kim Overvad [c,d], Ole Raaschou-Nielsen [a,b]

[a] Diet, Genes and Environment, Danish Cancer Society Research Centre, Copenhagen, Denmark
[b] Department of Environmental Science, Aarhus University, Roskilde, Denmark
[c] Department of Public Health, Section for Epidemiology, Aarhus University, Aarhus, Denmark
[d] Department of Cardiology, Aalborg University Hospital, Aalborg, Denmark

ARTICLE INFO

Article history:
Received 26 June 2015
Received in revised form 18 September 2015
Accepted 20 September 2015
Available online xxxx

Keywords:
Air pollution
Road traffic noise
Cholesterol
Epidemiology
Cross-sectional

ABSTRACT

Background: Exposure to traffic noise and air pollution have both been associated with cardiovascular disease, though the mechanisms behind are not yet clear.
Objectives: We aimed to investigate whether the two exposures were associated with levels of cholesterol in a cross-sectional design.
Methods: In 1993–1997, 39,863 participants aged 50–64 year and living in the Greater Copenhagen area were enrolled in a population-based cohort study. For each participant, non-fasting total cholesterol was determined in whole blood samples on the day of enrolment. Residential addresses 5-years preceding enrolment were identified in a national register and road traffic noise (L_{den}) were modeled for all addresses. For air pollution, nitrogen dioxide (NO_2) was modeled at all addresses using a dispersion model and $PM_{2.5}$ was modeled at all enrolment addresses using a land-use regression model. Analyses were done using linear regression with adjustment for potential confounders as well as mutual adjustment for the three exposures.
Results: Baseline residential exposure to the interquartile range of road traffic noise, NO_2 and $PM_{2.5}$ was associated with a 0.58 mg/dl (95% confidence interval: −0.09; 1.25), a 0.68 mg/dl (0.22; 1.16) and a 0.78 mg/dl (0.22; 1.34) higher level of total cholesterol in single pollutant models, respectively. In two pollutant models with adjustment for noise in air pollution models and vice versa, the association between air pollution and cholesterol remained for both air pollution variables (NO_2: 0.72 (0.11; 1.34); $PM_{2.5}$: 0.70 (0.12; 1.28) mg/dl), whereas there was no association for noise (−0.08 mg/dl). In three-pollutant models (NO_2, $PM_{2.5}$ and road traffic noise), estimates for NO_2 and $PM_{2.5}$ were slightly diminished (NO_2: 0.58 (−0.05; 1.22); $PM_{2.5}$: 0.57 (−0.02; 1.17) mg/dl).
Conclusions: Air pollution and possibly also road traffic noise may be associated with slightly higher levels of cholesterol, though associations for the two exposures were difficult to separate.

1. Introduction

Epidemiological studies have consistently found both long-term exposure to ambient air pollution and road traffic noise to be associated with cardiovascular disease (Babisch, 2014; Brook et al., 2010). As road traffic is an important source of both noise and ambient air pollution the two exposures are correlated, making it difficult to separate their effects. Some of the few studies including both exposures in relation to cardiovascular disease suggest an effect of both road traffic noise and air pollution in mutually adjusted models (Babisch et al., 2014; Dratva et al., 2012; Vienneau et al., 2015), whereas other studies indicate associations mainly with one of the pollutants (Beelen et al., 2009; Sorensen et al., 2014).

Many of the biological mechanisms believed to link exposure to risk for cardiovascular disease are similar for road traffic noise and ambient air pollution, including increased sympathetic tone, effects on the immune system, endothelial dysfunction and atherosclerosis (Bauer et al., 2010; Hoffmann et al., 2009; Kalsch et al., 2014; Kunzli et al., 2005; Kunzli et al., 2010; Schmidt et al., 2013). Interestingly, one study found that in mutually adjusted models both long-term exposure to air pollution and traffic noise was associated with subclinical atherosclerosis (Kalsch et al., 2014). A known risk factor for atherosclerosis and cardiovascular disease is cholesterol (Prospective Studies Collaboration et al., 2007). The few studies that have investigated the association between air pollution and levels of total cholesterol and

Abbreviations: CI, confidence interval; NO_2, nitrogen dioxide.

* Corresponding author at: Diet, Genes and Environment, Danish Cancer Society Research Center, Strandboulevarden 49, 2100 Copenhagen Ø, Denmark.
E-mail address: mettes@cancer.dk (M. Sørensen).

http://dx.doi.org/10.1016/j.envint.2015.09.021
0160-4120/

other blood lipids, indicate that air pollution may influence the level of blood lipids, though results are not consistent (Chuang et al., 2010; Chuang et al., 2011; Poursafa et al., 2014; Yeatts et al., 2007). For road traffic noise, one study indicated a positive association with level of cholesterol (Babisch et al., 1988), whereas another study found no association (Babisch et al., 1993). Also, traffic noise is believed to increase risk for cardiovascular disease through disturbance of sleep, and most (Ekstedt et al., 2004; Gangwisch et al., 2010; Wan Mahmood et al., 2013) but not all (Petrov et al., 2013) studies on sleep and blood lipids indicate that short and disturbed sleep is associated with higher levels of cholesterol.

We aimed to investigate whether long-term residential exposure to road traffic noise and ambient air pollution were associated with higher levels of total cholesterol in a large cohort, and whether a potential association between noise and cholesterol was influenced by adjustment for air pollution and vice versa.

2. Methods

2.1. Study population

The study was based on the Diet, Cancer and Health cohort where 160,725 randomly chosen subjects without a history of cancer, between 50 and 64 years of age and living in the Copenhagen or Aarhus area, were invited to participate between 1993 and 1997 (Tjonneland et al., 2007). All in all 57,053 subjects accepted the invitation and were enrolled into the cohort, and of these 39,863 participants lived in the Greater Copenhagen. Participation was based on written informed consent. The study was conducted in accordance with the Helsinki Declaration and approved by the local ethical committees. At enrolment, 30 ml non-fasting blood sample was drawn from each participant and each participant completed self-administered questionnaires including questions on disease and lifestyle habits, such as diet, tobacco smoking, alcohol intake and sport during leisure time. Information on hypercholesterolemia at enrolment was assessed by questions on whether the participants had ever been diagnosed with hypercholesterolemia and whether they received or had ever received medication for hypercholesterolemia. Height, weight and waist circumference were measured by trained personnel according to standardized protocols. We excluded cohort members not living in Greater Copenhagen at baseline, with cancer before baseline, without cholesterol measurement, with incomplete address/exposure history, with missing covariates information and/or who received medication for hypercholesterolemia.

2.2. Measurement of total cholesterol

Non-fasting total cholesterol (mmol/L) was determined in whole blood on the day of enrolment at the time of blood sampling using a Lipotrend® C device with Lipotrend test strips (Boehringer Mannheim). Lipotrend® C is a reflectance photometer for the determination of total cholesterol from capillary blood or plasma in the range of 100–500 mg/dl (2.6–13.0 mmol/L). The sample was applied onto the test strip by means of a plastic capillary. Cholesterol determination was automatically begun upon correct insertion of the test strip into the instrument. The optical system measures the color intensity of the test strip's reaction zone at two different times, and the difference is converted into the cholesterol concentration using the Lipotrend® C software and shown in the display. For our analyses, the SI unit (mmol/L) was converted to mg/dl by multiplying by 38.7.

2.3. Exposure assessment

The level of road traffic noise was modeled at each address at which the cohort members had lived from five years preceding enrolment until enrolment by use of SoundPLAN (see http://www.soundplan.dk/). This noise calculation program implements the joint Nordic prediction method for road traffic noise, which has been the standard method for noise calculation in Scandinavia during many years (Bendtsen, 1999). The input variables for the noise model were: point for noise estimation (including height); road links with information on annual average daily traffic, vehicle distribution (of light and heavy vehicles), travel speed and road type (motorway, express road, road wider than 6 m, road less than 6 m and more than 3 m, and other road); and building polygons including height for all buildings, obtained from the Danish Geodata Agency. Information on road lines as well as traffic counts for all Danish roads with more than 1000 vehicles per day was obtained from a national road and traffic database at the Danish Centre for Environment and Energy, Aarhus University (Jensen et al., 2009a). We assumed a flat terrain, 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. No information was available on noise barriers and road surface. Road traffic noise was calculated as equivalent continuous A-weighted sound pressure levels at the most exposed facade of the dwelling for day, evening and night, and expressed as L_{den}. Values below 40 dB were set to 40 dB, considered as a lower limit of road traffic noise.

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 with building heights 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.

Annual ambient exposure to NO_2 was calculated for all residential addresses 5 years preceding enrolment using the Danish AirGIS dispersion modeling system (see http://envs.au.dk/videnudveksling/luft/model/airgis/). AirGIS performs calculation of air pollution at an address location as the sum of: 1) local air pollution from the nearest street traffic, calculated from traffic (intensity and type), emission factors for the car fleet, street and building geometry and meteorology; 2) urban background, calculated from data on urban vehicle emission density, city dimensions and typical building heights; and 3) regional background, estimated from trends at rural monitoring stations and from national vehicle emissions (Berkowicz et al., 2008). Input data on traffic were the same as described for noise modeling. The AirGIS system model have been successfully validated and applied in several studies (Andersen et al., 2012; Jensen et al., 2009b; Ketzel et al., 2011; Ketzel et al., 2012).

Exposure to $PM_{2.5}$ was determined at all residential baseline addresses for cohort participants living in Greater Copenhagen, using a land-use regression model, as part of the ESCAPE study (Eeftens et al., 2012). Briefly, a three-step procedure was used. First, $PM_{2.5}$ was measured during different seasons at different locations in the study area. Second, a land-use regression model was developed for $PM_{2.5}$, with the yearly mean concentration as the dependent variable and an extensive list of geographical attributes as possible predictors, such as road network and population density. Finally, the model was used to assess exposure at the baseline address of each cohort member.

2.4. Statistical analyses

The present study is a cross-sectional study and we used generalized linear models for the analyses. For each person, exposure to road traffic noise and both air pollutants were modeled at baseline address as well as a time-weighted 5-year means preceding enrolment date for noise and NO_2, taking the complete migration history in this period into account. We investigated the association between the three exposures and level of total cholesterol at enrolment, including exposures as continuous variables (per interquartile range). We adjusted for potential