

Associations of road traffic noise, blood pressure and heart rate in three harmonized European cohorts

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Summary

Exposure to road traffic noise may increase risk of hypertension. Ambient air pollution may be a confounder in this relationship, as both noise and air pollution originate from traffic and both may be associated with hypertension. We investigated associations of road traffic noise with blood pressure and heart rate, while taking into account exposure to ambient air pollution, in three European cohorts using a harmonized approach. Data were obtained from LifeLines (the Netherlands), EPIC-Oxford (UK) and HUNT (Norway). Road traffic noise exposure was assessed using a European noise model based on Common Noise Assessment Methods in Europe (CNOSSOS-EU). Exposure to particulate matter with a diameter $\leq 10 \mu\text{m}$ (PM_{10}) and nitrogen dioxide (NO_2) was estimated using a European land use regression model. Both systolic and diastolic blood pressure and heart rate measurements were conducted by trained medical professionals within each participating cohort. Federated analysis of harmonized data for the three cohorts will be conducted at the individual level using the DataSHIELD approach. Data from 123,366 participants, with a mean age of 47.6 years, were available for the current study. Median annual average day-evening-night levels of road traffic noise (L_{den}) were 54.7 dB(A) (LifeLines), 54.8 dB(A) (EPIC-Oxford), and 49.4 dB(A) (HUNT). Results from pooled linear regression analyses adjusted for sex, age, and ambient air pollution will be presented at the conference. Pooling harmonized data from multiple European cohorts allows the use of large sample sizes with a wide noise exposure range. Findings from this study will contribute to the knowledge about harmful effects of road traffic noise on cardiovascular health.

1. Introduction

Environmental noise poses a major public health problem. In Western-Europe, each year at least one million healthy life years are lost due to traffic related noise. It is estimated that these losses are mostly due to ischemic heart disease, cognitive

impairment of children, sleep disturbance, tinnitus, and annoyance [1].

A meta-analysis of 24 studies concluded that road traffic noise was positively associated with hypertension. A pooled odds ratio of 1.034 (95% confidence interval 1.011-1.056) per 5 decibel (A) was reported. This indicates that each increase of 5 decibel was associated with a 3.4% increased risk for hypertension [2]. Hypertension is a major risk factor for cardiovascular diseases and one of the

leading risk factors for global disease burden [3], making it a relevant public health problem. Relations of noise and heart rate have also been investigated, but results were inconsistent. Positive associations [4,5] but also no [6,7] or negative associations [8] of noise and heart rate have been found.

Noise is believed to provoke a stress response. Noise may directly or indirectly activate sympathetic and endocrine systems, followed by increases in heart rate, blood pressure and release of stress hormones. Sleep disturbance may also disrupt secretion of stress hormones, affecting metabolism and the cardiovascular system [9]. These mechanisms may explain associations between noise and hypertension. Exposure to ambient air pollution, especially particulate matter (PM), is also associated with cardiovascular diseases. As road traffic is the common source for both noise and air pollution, it is important to distinguish between the two. Until recently, previous studies did not take into account the simultaneous effects of traffic-related noise and air pollution. A recent systematic review identified nine studies that assessed the confounding effects of noise and air pollution on cardiovascular health outcomes [10]. Based on these studies, the authors concluded that noise and air pollution probably have an independent effect on cardiovascular diseases. However, due to the heterogeneity of the studies that were assessed and also due to the scarceness of the current literature, a final conclusion cannot yet be drawn. Additional research is needed where effects of traffic-related noise and air pollution on cardiovascular health are investigated simultaneously. We will investigate associations of road traffic noise with blood pressure and heart rate, while taking into account exposure to ambient air pollution with data from three European cohorts. Our study is undertaken within the Biobank Standardisation and Harmonisation for Research Excellence in the European Union (BioSHaRE-EU) project, using a harmonized approach to both exposures and health data.

2. Methods

Data were obtained from three European cohorts: LifeLines (the Netherlands) [11], EPIC-Oxford (United Kingdom) [12] and HUNT (Norway) [13]. Cohort characteristics are summarized in Table I. All participants provided written informed consent, and study protocols were approved by the local ethical committees.

Road traffic noise exposure at individual home addresses was assessed using a European noise model based on Common Noise Assessment Methods in Europe (CNOSSOS-EU) [14]. The CNOSSOS-EU noise modelling framework was developed as a common methodology for noise modelling across Europe. National versions of CNOSSOS-EU were developed within the BioSHaRE project to estimate road traffic noise in the geographical areas of LifeLines, EPIC-Oxford and HUNT. These national versions were based on less detailed inputs because high resolution input data at national or large regional level is either unavailable, expensive or would be too computationally intensive to process. The noise exposure model that was developed attempts to solve these problems by using data available at the required national scale, often of a lower resolution, to generate modelled noise values at an acceptable level of accuracy. The CNOSSOS-EU framework contains empirically derived equations to determine the initial noise level based on traffic flow and sound attenuation based on known environmental factors and physical processes. Input data used for the model included road networks, hourly flow of vehicles and their average speeds, land cover types (especially urban fabric vs. areas of vegetation), and meteorology.

Full details of the BioSHaRE/CNOSSOS-EU noise modelling are currently being prepared in a manuscript [15]. Five A-weighted indicators of road traffic noise were estimated for each cohort: L_{day} (12-hour day time period from 07:00 to 19:00 hour); L_{evening} (4-hour evening time period from 19:00 to 23:00 hours); L_{night} (8-hour night time period from 23:00 to 07:00 hours); L_{aeq16} (16-hour day and evening time period from 07:00 to 23:00 hours);

and L_{den} (day-evening-night time period of 24 hours). Hourly noise estimates (L_{aeq} 0-23 hours) are also available. L_{den} and L_{night} are used in the current study.

Exposure to particulate matter with a diameter $\leq 10 \mu m$ (PM_{10}) and nitrogen dioxide (NO_2) at individual home addresses was estimated using a European land use regression model [16]. This model covers Western Europe and incorporates Geographic Information System (GIS)-derived land use data, road network data and other topographic data, and satellite-derived estimates of ground level concentrations for PM_{10} and NO_2 .

Systolic and diastolic blood pressure and heart rate measurements were conducted by trained medical professionals within each participating cohort. Covariates reported in questionnaires were harmonized according to the DataSHaPER methodology [17,18].

Federated analyses of harmonized data for the three cohorts were conducted at the individual level using the DataSHIELD approach. DataSHIELD enables analysis of multiple studies by analyzing individual-level harmonized data from each cohort, without physically pooling of data. Cohort data stays behind the firewalls of the cohort's host computers. As a result, individual data can be simultaneously analyzed without transferring data externally. DataSHIELD offers a solution to practical, ethical, and legal issues associated with data sharing and analysis [19,20]. Pooled individual-level linear regression analyses will be performed to investigate the associations of (1) road traffic noise and systolic blood pressure; (2) road traffic noise and diastolic blood pressure; and (3) road traffic noise and heart rate. Associations with 24 hour (L_{den}) and night time (L_{night}) noise will be investigated, and regression models will be adjusted for cohort, age, sex, and PM_{10} , and/or NO_2 .

3. Results

Data from 123,366 participants, with a mean age of 47.6 years, were available for this study. The (preliminary) pooled and cohort specific population characteristics are summarized in Table II. Median annual average day-evening-night levels of road traffic noise (L_{den}) were 54.7 dB(A) (LifeLines), 54.8 dB(A) (EPIC-Oxford), and 49.4 dB(A) (HUNT), resulting in a pooled median of 52.8 dB(A). Distribution of pooled L_{den} is shown in Figure I. Median levels of PM_{10} varied between 11.1 (HUNT) and 23.6 (LifeLines) $\mu g/m^3$, and between 11.8 (HUNT) and 25.4 (EPIC-Oxford) $\mu g/m^3$ for NO_2 . Correlations between road traffic noise and PM_{10} and road traffic noise and NO_2 were low in EPIC-Oxford ($r=0.07$ and 0.11 respectively) and HUNT ($r=0.06$ and -0.04 respectively), and moderate in LifeLines ($r=0.40$ and 0.46 respectively; all p-values were $<.001$). Mean levels of systolic blood pressure varied between 124.4 and 130.6 mmHg, with highest mean level observed in HUNT. Pooled mean systolic blood pressure (SBP) was 126.9 mmHg. Mean levels of diastolic blood pressure (DBP) varied between 73.3 mmHg (LifeLines) and 77.1 mmHg (EPIC-Oxford), and the mean pooled level was 73.9 mmHg. Mean heart rate levels varied little across the cohorts, and the pooled mean level was 71.0 beats per minute (bpm). Prevalence of hypertension based on self-report data and medication use were 19.8% and 22.3%, respectively. Results from pooled linear regression analyses adjusted for sex, age, and ambient air pollution will be presented at the conference.

Table I. Cohort characteristics.

	<i>LifeLines</i>	<i>EPIC-Oxford</i>	<i>HUNT</i>
Region	Three Northern provinces from the Netherlands	United Kingdom	Nord-Trøndelag region, Norway
Measurement period	2007-2013	1993-1997	2006-2008
Participants	General population	General population and vegetarians, vegans	General population
Sample size at baseline	167,729	57,446	50,807

Table II. Pooled and cohort specific population characteristics. *All results presented here are preliminary, as analyses are in progress.*

	<i>Pooled</i>	<i>LifeLines</i>	<i>EPIC-Oxford</i>	<i>HUNT</i>
N	123,366	61,888	16,231	45,247
Age (years); mean (SD)	47.6 (14.2)	43.8 (12.3)	48.8 (14.2)	52.4 (16.1)
Females, %	59	58	77	55
SBP (mmHg); mean (SD)	126.9 (17.5)	124.4 (14.9)	126.0 (19.0)	130.6 (18.7)
DBP (mmHg); mean (SD)	73.9 (10.4)	73.3 (9.3)	77.1 (11.0)	73.4 (11.3)
HR (bpm); mean (SD)	71.0 (11.1)	71.7 (10.9)	72.1 (10.8)	69.6 (11.5)
Hypertension ¹ , %	19.8	20.1	11.9	NA
Antihypertensive medication, %	22.3	22.9	NA	21.9
L _{den} (dB(A)); median (IQR)	52.8 (4.6)	54.7 (4.4)	54.8 (3.4)	49.4 (6.0)
L _{night} (dB(A)); median (IQR)	43.5 (4.3)	45.2 (4.4)	45.3 (3.4)	40.2 (5.0)
PM ₁₀ (µg/m ³); median (IQR)	18.8 (2.4)	23.6 (2.3)	22.0 (3.0)	11.1 (1.6)
NO ₂ (µg/m ³); median (IQR)	18.0 (8.3)	20.6 (8.8)	25.4 (9.1)	11.8 (5.3)

Abbreviations: SD=standard deviation; SBP=systolic blood pressure; DBP=diastolic blood pressure; HR=heart rate; bpm=beats per minute; L_{den}=24 hour noise estimate; dB(A)=decibels A; L_{night}=night-time noise estimate; IQR=interquartile range; PM₁₀= particulate matter with a diameter ≤10 µm; NO₂=nitrogen dioxide; NA=not available for cohort. Hypertension¹: self-reported history of hypertension

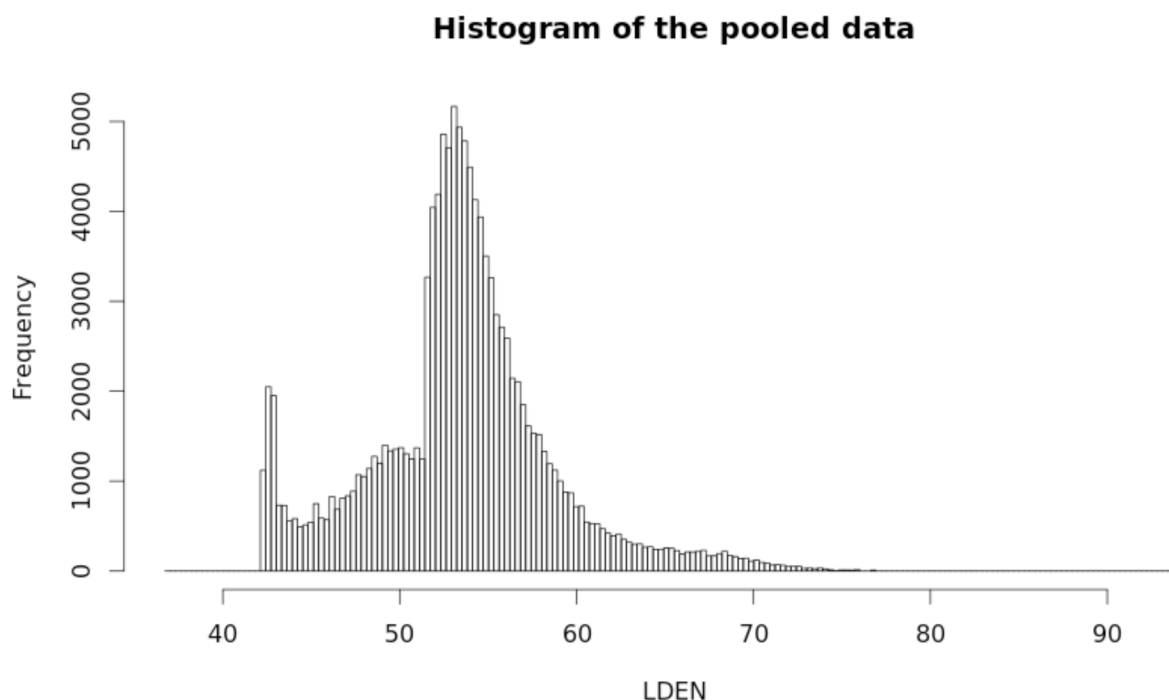


Figure I. Histogram of pooled 24 hour road traffic noise levels (dB(A)) in LifeLines, EPIC-Oxford and HUNT.

4. Discussion and conclusions

Several strengths and limitations should be taken into consideration when interpreting the results. Firstly, although exposure ranges of road traffic noise and ambient air pollution in our study were quite large, median levels were relatively low, if compared to other European cohorts (e.g. [21]). Our conclusions will therefore be limited to low exposure levels. Secondly, because of selection for ‘health conscious’ persons, participants in EPIC-Oxford may be more healthy than participants in the other cohorts. Selection of such a specific group might underestimate the results. Furthermore, although blood pressure and heart rate data were available for the cohorts, examination methods differed. Differences in measurement procedures could have resulted in variations in outcome data for the cohorts. Pooling harmonized data from multiple European cohorts allows the use of large sample sizes with a wide noise exposure range, but we have to be aware of heterogeneity between cohorts. Findings

from this study will contribute to the knowledge about harmful effects of road traffic noise on cardiovascular health.

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