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Road traffic noise, air pollution and incident cardiovascular disease: A joint analysis of the HUNT, EPIC-Oxford and UK Biobank cohorts

Yutong Cai[a,b,*], Susan Hodgson[a], Marta Blangiardo[a], John Gulliver[a], David Morley[a], Daniela Fecht[a], Danielle Vienneau[c,d], Kees de Hoogh[c,d], Tim Key[e], Kristian Hveem[f], Paul Elliott[a,g], Anna L. Hansell[a,g]

[a] MRC-PHE Centre for Environment and Health, Department of Epidemiology and Biostatistics, School of Public Health, Imperial College London, London, United Kingdom
[b] MRC-PHE Centre for Environment and Health, Department of Analytical, Environmental and Forensic Sciences, School of Population Health and Environmental Sciences, King's College London, London, United Kingdom
[c] Swiss Tropical and Public Health Institute, Basel, Switzerland
[d] University of Basel, Basel, Switzerland
[e] Cancer Epidemiology Unit, Nuffield Department of Population Health, University of Oxford, Oxford, United Kingdom
[f] Department of Public Health and Nursing, Norwegian University of Science and Technology, Trondheim, Norway
[g] Directorate of Public Health and Primary Care, Imperial College Healthcare NHS Trust, London, United Kingdom

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ABSTRACT

Background: This study aimed to investigate the effects of long-term exposure to road traffic noise and air pollution on incident cardiovascular disease (CVD) in three large cohorts: HUNT, EPIC-Oxford and UK Biobank.
Methods: In pooled complete-case sample of the three cohorts from Norway and the United Kingdom (N = 355,732), 21,081 incident all CVD cases including 5259 ischemic heart disease (IHD) and 2871 cerebrovascular cases were ascertained between baseline (1993–2010) and end of follow-up (2008–2013) through medical record linkage. Annual mean 24-hour weighted road traffic noise (Lden) and air pollution (particulate matter with aerodynamic diameter ≤ 10 μm [PM10], ≤2.5 μm [PM2.5] and nitrogen dioxide [NO2]) exposure at baseline address was modelled using a simplified version of the Common Noise Assessment Methods in Europe (CNOSSOS-EU) and European-wide Land Use Regression models. Individual-level covariate data were harmonised and physically pooled across the three cohorts. Analysis was via Cox proportional hazard model with mutual adjustments for both noise and air pollution and potential confounders.
Results: No significant associations were found between annual mean Lden and incident CVD, IHD or cerebrovascular disease in the overall population except that the association with incident IHD was significant among current-smokers. In the fully adjusted models including adjustment for Lden, an interquartile range (IQR) higher PM10 (4.1 μg/m3) or PM2.5 (1.4 μg/m3) was associated with a 5.8% (95%CI: 2.5%–9.3%) and 3.7% (95%CI: 0.2%–7.4%) higher risk for all incident CVD respectively. No significant associations were found between NO2 and any of the CVD outcomes.
Conclusions: We found suggestive evidence of a possible association between road traffic noise and incident IHD, consistent with current literature. Long-term particulate air pollution exposure, even at concentrations below current European air quality standards, was significantly associated with incident CVD.

1. Introduction

Traffic noise and air pollution are the leading environmental risk factors for health in Europe. A study using World Health Organisation (WHO) 2009 exposure response functions (WHO, 2009) estimated that noise from road, rail and air traffic in western European countries was associated with 400–1500 disability-adjusted life years (DALYs) per one million Europeans, the second highest ranking after particulate air pollution (Hanninen et al., 2014).

As an environmental stressor, traffic noise is hypothesized to exert adverse health effects via both direct (e.g. sleep disturbance) and indirect (e.g. annoyance) pathways (Babisch, 2014). In acute response to noise, via activations of the hypothalamus-pituitary-adrenal (HPA) axis and the sympathetic-adrenal-medulla axis, stress hormones such as

* Corresponding author at: MRC-PHE Centre for Environment and Health, Department of Epidemiology and Biostatistics, School of Public Health, Imperial College London, St Mary's Campus, Norfolk Place, London W2 1PG, United Kingdom.
E-mail address: yutong.cai@imperial.ac.uk (Y. Cai).

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adrenalin and cortisol are released (Munzel et al., 2017; Recio et al., 2016). In the long term, these adaptive physiological responses may result in adverse pathophysiologic changes including to blood pressure, lipids and glucose, which ultimately may contribute to manifest cardiovascular disease (CVD) (Cai et al., 2017).

Beyond findings on annoyance and sleep disruption, the evidence base for cardiovascular effects of long-term traffic noise exposure, particularly those of road transport sources, has been substantially strengthened in the past decade. Of note, most evidence to date has linked noise exposure to hypertension (van Kempen and Babisch, 2012) and ischemic heart disease (IHD) (Babisch, 2014; Vienneau et al., 2015), although for the latter more evidence is needed to strengthen our understanding of the dose-response relationship and to identify susceptible subgroups. Evidence on associations with other CVD outcomes such as stroke (Halonen et al., 2015; Sorensen et al., 2011), atherosclerosis (Kalsch et al., 2014), atrial fibrillation (Monrad et al., 2016), heart failure (Heritier et al., 2017; Sorensen et al., 2017) and arterial stiffness (Foraster et al., 2017) is emerging but more studies are needed given the paucity of available data.

There is also a growing number of studies reporting associations between long-term air pollution exposures and cardiovascular morbidity and mortality (Newby et al., 2015), for which the main hypothesized underlying mechanism is via oxidative stress (Kelly and Fussell, 2017). Additionally, the combined contribution of road traffic noise and air pollution to CVD outcomes merits more investigation (Stansfeld, 2015) since both exposures share a common source and some similar mechanistic pathways leading to CVD (Babisch, 2014; Brook et al., 2010).

In this study, analysing harmonised noise, air pollution and health data from three large cohort studies, we aimed to separately investigate the associations between long-term residential road traffic noise, ambient air pollution and incident CVD, IHD and stroke, taking into account effects of potential confounders and co-adjustment of both road traffic noise and air pollution in the main model. We also examined possible effect modification of these associations for a range of a priori selected variables in this very large study sample.

2. Methods

2.1. Study populations

Three population-based cohorts, HUNT (Helseundersøkelsen i Nord-Trøndelag) (Krokstad et al., 2013), EPIC-Oxford (European Prospective Investigation into Cancer and Nutrition) (Davey et al., 2003) and UK Biobank (Sudlow et al., 2015), were included in this study as part of the BioSHaRE-EU (Biobank Standardisation and Harmonisation for Research Excellence) project.

The HUNT study is a population-based health survey conducted in the whole county of Nord-Trøndelag in central Norway, targeting all residents aged ≥20 years. We used data from the second survey (HUNT2) undertaken in 1995–1997, during which 65,232 residents participated and provided data (Krokstad et al., 2013). EPIC-Oxford is a component of the EPIC study (Riboli and Kaaks, 1997). During 1993–1999, 57,446 participants aged ≥20 years living throughout the United Kingdom (UK) were recruited through general practices or via postal methods and completed baseline assessments (Davey et al., 2003). UK Biobank, established during 2006–2010, recruited 502,649 participants aged 40–69 years across the UK (Sudlow et al., 2015). Informed consent and ethical approvals were obtained from all three cohorts.

2.2. Incident CVD outcomes

In each cohort, the same censoring date was set for both non-fatal and fatal incident CVD outcomes (Table 1). Since baseline recruitment, follow-up of first CVD event was based on linkages to both hospital admission records and mortality registries using the unique National Health Service (NHS) number in the UK and Personal Identification Number (PIN) in Norway. Registry of hospital admission started in England on 1st April 1997 and therefore participants from England who were recruited into EPIC-Oxford prior to this date did not have hospital admission records between recruitment and 1st April 1997. These participants (n = 7314) were therefore excluded from analyses.

International Classification of Diseases (ICD) codes revisions 9 and 10 were used in the registries in both UK and Norway. The outcomes examined in this study were all CVD (ICD-9:390–459; ICD-10: I00–I99), IHD (ICD-9: 410–414; ICD-10: I20–I25) and cerebrovascular disease (ICD-9: 430–438; ICD-10: I60–I69). Incident cases were ascertained if one of the above codes appeared in the linked medical records between baseline recruitment and death, emigration or end of follow-up, whichever came first (event/censoring). Acute coronary events (ICD-9:410,411; ICD-10:I20.0, I21, I23, I24), ischemic stroke (ICD-9:433,434; ICD-10:I63), and hemorrhagic stroke (ICD-9: 431; ICD-10: I60, I61, I62) were also ascertained.

Participants who reported prevalent CVD, including hypertension, heart attack, angina and stroke at baseline questionnaire, were excluded from analyses. Further, by screening medical records, participants who had CVD diagnosed prior to baseline recruitment were also excluded (Table 1).

2.3. Exposure assessment

A simplified version (Morley et al., 2015) of the CNOSSOS-EU (Common noise assessment methods in European Union) noise modelling framework (Kephalopoulos et al., 2014) was developed and run for each cohort.

Noise sound pressure level was estimated on all roads within 500 m of home address at recruitment. Noise propagation due to refraction and diffraction, absorption from buildings, distance and angle of view were considered in the model. Road network geography, calculated hourly vehicle flows using a daily average traffic profile, building heights, land cover and meteorological data (2001 − 2010) were obtained for the respective study areas. To account for participants living on minor roads that were not captured in the national level traffic datasets, a fixed low-level baseline flow (600 vehicles per day) was assigned (Gulliver et al., 2015). Traffic data were for the year 2009 and land cover data for the year 2006. Annual mean A-weighted sound pressure level in decibels (dB(A)) for daytime noise (averaged sound level from 07:00 to 19:00), night-time noise (averaged sound level from 23:00 to 07:00) and weighted 24-hour average noise (L_{den}) were modelled at baseline home address of participants in all three cohorts.

For all three cohorts, annual mean particulate matter with aerodynamic diameter ≤ 10 μm (PM_{10}) and nitrogen dioxide (NO_2) air pollution estimates at baseline home addresses for the year 2007 were assigned from a harmonised European Land Use Regression (LUR) model at a resolution of 100 × 100 m (Vienneau et al., 2013). The harmonised European LUR model was developed using monitored air pollution data from over 1500 monitoring sites across Western Europe, satellite-based ground-level concentrations of $PM_{2.5}$ (particulate matter with aerodynamic diameter ≤ 2.5 μm) and NO_2 on a 10-km grid, land-use and traffic variables obtained from GIS (Geographic Information System).

Additional annual estimated $PM_{2.5}$ concentrations for year 2010 were available for EPIC-Oxford and UK Biobank, using the LUR models developed by the European Study of Cohorts for Air Pollution Effects (ESCAPE) project (Eeftens et al., 2012).

A detailed summary of variables used in exposure modelling was described in Supplementary Table S1.

2.4. Statistical analyses

We used Cox proportional hazards models, stratified by sex, with