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Long-term associations of modeled and self-reported measures of exposure to air pollution and noise at residence on prevalent hypertension and blood pressure

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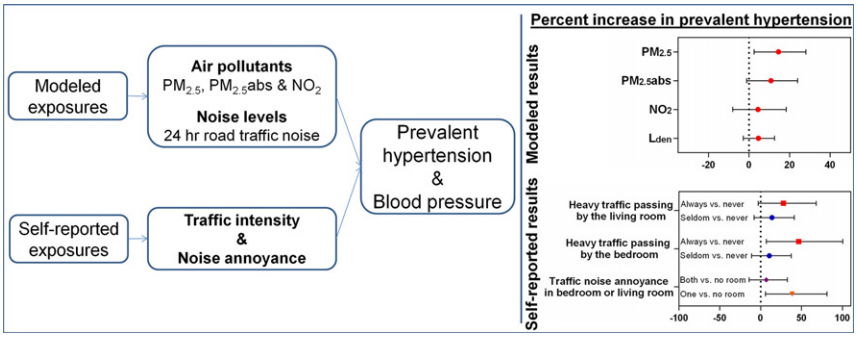
HIGHLIGHTS

- We evaluated long-term associations of modeled air pollution and noise exposure on hypertension and blood pressure.
- We further examined associations of self-reported heavy vehicle traffic intensity and noise annoyance on these outcomes.
- Hypertension was significantly associated with $PM_{2.5}$ modeled at study participants' residences.
- Diastolic blood pressure was associated with both air pollutants and road traffic noise levels.
- Air pollution effects were stronger in men and diabetic individuals.

GRAPHICAL ABSTRACT



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ABSTRACT

Air pollution, traffic noise and noise annoyance are suggested to be associated with hypertension and blood pressure (BP); however, the evidence remains inconsistent. Our study examined the long-term associations of modeled and self-reported measures of air pollution and traffic noise on prevalent hypertension and BP. We analyzed cross-sectional data from 2552 participants aged 31–72 years from the KORA F4 (2006–2008) study conducted in the region of Augsburg, Germany. Land-use regression models were used to estimate residential long-term exposure to particulate matter <2.5 μm ($PM_{2.5}$), soot content of $PM_{2.5}$ ($PM_{2.5}$abs) and nitrogen dioxide (NO_2). Road traffic noise levels at the facade of the dwellings were estimated for the participants' residences. Participants filled-in a questionnaire on noise annoyance and heavy traffic passing their residence. Linear and logistic regression models adjusting for confounders were used to assess the association between exposure measures and hypertension and BP. An interquartile increase in annual mean $PM_{2.5}$ (1 μg/m^3) was significantly associated

Abbreviations: BP, blood pressure; CI, confidence interval; DBP, diastolic blood pressure; IQR, interquartile range; L_{den}, 24-hour road traffic noise levels; L_{night}, night-time road traffic noise levels; NO_2, nitrogen dioxide; $PM_{2.5}$, particulate matter with aerodynamic diameter < 2.5 μm; $PM_{2.5}$abs, soot content of $PM_{2.5}$; SBP, systolic blood pressure.

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Hypertension
Blood pressure
Noise annoyance
Traffic intensity

with 15% higher prevalence of hypertension, without (95% CI: 2.5; 28.0%) and with (95% CI: 0.7; 30.8%) adjustment for traffic noise. Diastolic blood pressure (DBP) was associated with air pollutants and traffic noise with percent increases in mean of 0.7 (95% CI: 0.2; 1.2), 0.6 (95% CI: 0.1; 1.1) and 0.3 (95% CI: 0.0; 0.7) for an interquartile increase in $PM_{2.5}$ (1 μg/m^3) and $PM_{2.5}$abs ($0.2 * 10^{-5}$/m), and 5 dB(A) increase in 24-hour road traffic noise, respectively. Associations of $PM_{2.5}$abs and NO_2 with hypertension or DBP were stronger in men and diabetic individuals. No clear associations were seen with systolic BP or noise annoyance. In conclusion, self-reported measures of air pollution or noise did not perform better than the objective measures. Our findings provide further evidence for a link between air pollution, noise and cardiovascular disease and indicate a stronger association for men and diabetic individuals.

1. Introduction

Road traffic is the predominant source of air pollution and noise. Both exposures have been linked to several clinical manifestations of cardiovascular disease like atherosclerosis, myocardial infarction and stroke (Rückerl et al., 2011; Stansfeld, 2015). The main precursor for the cardiovascular events is high blood pressure, which increases the tension in arterial wall and promotes atherosclerosis. Epidemiological studies investigating associations between long-term air pollutant exposures and blood pressure or hypertension have shown conflicting results; some studies suggest a positive association, while others show a negative or null association (Paolo et al., 2016). Most studies used objectively modeled long-term air pollution concentrations estimated by land-use regression or dispersion models as exposure to air pollution. However, some studies also used a subjective assessment of traffic exposure such as self-reported traffic intensity as indicator of exposure to road traffic pollution (Ciccone et al., 1998; Duhme et al., 1996). Self-reported traffic intensity may not be a direct subjective measure of exposure to air pollution. But, motor vehicle emissions, especially from heavy vehicles are a significant source of traffic-related air pollution. Therefore, measures of traffic flow near home, focusing directly on sources, have been suggested to be a reliable indicator of air pollution, even in the absence of direct measurements of pollutants (Ciccone et al., 1998). However, the validity and reliability of the self-reported traffic exposure is questionable. Thus, Heinrich et al. compared self-reported traffic intensity and modeled air pollution concentrations and found only a weak association (Heinrich et al., 2005). Moreover, it is unclear whether self-reported heavy vehicle traffic intensity and modeled traffic-related air pollutants are associated and if yes, if they show the same direction in association with health outcomes.

Association between long-term road traffic noise at residence and hypertension or blood pressure have also been inconsistent, with some studies reporting a positive association (Babisch et al., 2014a; Babisch et al., 2014b; Chang et al., 2011; Foraster et al., 2014b; Fuks et al., 2011; van Kempen and Babisch, 2012) and others reporting null association (Dratva et al., 2012; Sorensen et al., 2011). One of the reasons for conflicting results might be the individual differences in noise sensitivity (Weinstein, 1978), which is a common predictor of annoyance regardless of the actual noise exposure (Paunovic et al., 2009). Noise sensitive people tend to react more and adapt slower to noises than less noise sensitive people (Stansfeld, 1992). Although annoyance is a subjective response to noise exposure, it is not explained entirely by noise levels (Waye and Öhrström, 2002), however a fair correlation has been reported between objective and subjective measures of noise (Birk et al., 2011). The degree of annoyance is mainly related to personal factors (e.g. sensitivity to noise, fear of harm connected with the source, personal evaluation of the source, and coping capacity with respect to noise) and social factors (e.g. general evaluation of the source, trust or misfeasance with source authorities, history of noise exposure, and expectations of residents) (Guski, 1999). There is evidence for a positive association between noise annoyance due to road traffic and hypertension (Babisch, 2006; Babisch et al., 2012). It is unclear whether objective or subjective measures of noise exposure have a stronger influence on health outcomes.

Although a number of studies examined the effects of either air pollution or noise on hypertension and blood pressure, only a few reported the associations of both exposures while adequately controlling for co-pollutants or noise (Babisch et al., 2014a; Dratva et al., 2012; Foraster et al., 2014b; Sorensen et al., 2011). In addition, no study so far examined the associations of self-reported heavy vehicle traffic intensity on hypertension or blood pressure. Therefore, the aim of our study was to evaluate the long-term associations of modeled and self-reported measures of exposure to air pollution and road traffic noise on prevalent hypertension and blood pressure within the KORA (Collaborative Health Research in the Region of Augsburg) F4 study. While self-reported measures are based on participants' perception and objective measures are based on modeling, we were also interested in examining whether modeled air pollution and noise can be explained by self-reported heavy vehicle traffic intensity and noise annoyance along with additional factors like participant characteristics.

2. Materials and methods

2.1. Study population and clinical measurements

The study was based on a cross-sectional design which comprised 3080 participants from the KORA F4 study (2006–2008) conducted in the city of Augsburg and its two adjacent counties Augsburg and Aichach-Friedberg in Germany (Holle et al., 2005). The KORA F4 study is a follow-up of the KORA S4 (1999–2001) population based health survey. From a total sample of 6640 subjects, drawn from the target population consisting of all German residents of the region aged 25–74 years, 4261 participated in the KORA S4 study. The study population was a stratified random sample based on age, sex and region. Further details on the study design and sampling method of the KORA S4 study are described in detail elsewhere (von Lengerke and Mielck, 2012). Of these, 3080 participated in the F4 follow-up study. Persons who died in the meantime, lived outside the study region, were lost to follow-up, demanded deletion of address data were not considered for the F4 study ($n = 394$). Of the remaining 3867 eligible persons, 787 were either not willing to participate or unable to be contacted, which resulted in a response rate of 79.6%. All participants underwent a standardized face to face interview for information on sociodemographic and lifestyle factors as well as physical examinations. Intake of antihypertensive medication was assessed by questionnaire. Blood pressure (BP) was measured using a standardized protocol three times during the clinical interview at the right arm in a sitting position after at least 5-min at rest with a 3-min interval between measurements. A validated automatic device (OMRON HEM 705-CP) was used for BP measurement. Participants were not fasting before BP measurement, but most did not smoke or drink coffee before and between the measurements. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were calculated as the average readings of the second and third measurements. Participants were classified as hypertensive based on blood pressure