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Association of long-term exposure to local industry- and traffic-specific particulate matter with arterial blood pressure and incident hypertension

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ABSTRACT

Background: Long-term exposure to fine particulate matter ($PM_{2.5}$) may lead to increased blood pressure (BP). The role of industry- and traffic-specific $PM_{2.5}$ remains unclear.
Objective: We investigated the associations of residential long-term source-specific $PM_{2.5}$ exposure with arterial BP and incident hypertension in the population-based Heinz Nixdorf Recall cohort study.
Methods: We defined hypertension as systolic BP ≥ 140 mmHg, or diastolic BP ≥ 90 mmHg, or current use of BP lowering medication. Long-term concentrations of $PM_{2.5}$ from all local sources ($PM_{2.5ALL}$), local industry ($PM_{2.5IND}$) and traffic ($PM_{2.5TRA}$) were modeled with a dispersion and chemistry transport model (EURAD-CTM) with a 1 km^2 resolution. We performed a cross-sectional analysis with BP and prevalent hypertension at baseline, using linear and logistic regression, respectively, and a longitudinal analysis with incident hypertension at 5-year follow-up, using Poisson regression with robust variance estimation. We adjusted for age, sex, body mass index, lifestyle, education, and major road proximity. Change in BP (mmHg), odds ratio (OR) and relative risk (RR) for hypertension were calculated per 1 $\mu g/m^3$ of exposure concentration.
Results: $PM_{2.5ALL}$ was highly correlated with $PM_{2.5IND}$ (Spearman's $\rho = 0.92$) and moderately with $PM_{2.5TRA}$ ($\rho = 0.42$). In adjusted cross-sectional analysis with 4539 participants, we found positive associations of $PM_{2.5ALL}$ with systolic (0.42 [95%-CI: 0.03, 0.80]) and diastolic (0.25 [0.04, 0.46]) BP. Higher, but less precise estimates were found for $PM_{2.5IND}$ (systolic: 0.55 [−0.05, 1.14]; diastolic: 0.35 [0.03, 0.67]) and $PM_{2.5TRA}$ (systolic: 0.88 [−1.55, 3.31]; diastolic: 0.41 [−0.91, 1.73]).

We found crude positive association of $PM_{2.5TRA}$ with prevalence (OR 1.41 [1.10, 1.80]) and incidence of hypertension (RR 1.38 [1.03, 1.85]), attenuating after adjustment (OR 1.19 [0.90, 1.58] and RR 1.28 [0.94, 1.72]). We found no association of $PM_{2.5ALL}$ and $PM_{2.5IND}$ with hypertension.

Abbreviations: AOD, automated oscillometric device; ATC, anatomical therapeutic chemical/defined daily dose classification; BP, blood pressure; BPLM, BP lowering medication; dB, decibel; EURAD, the European air pollution dispersion; CTM, chemistry transport model; HNR, Heinz Nixdorf Recall study; IQR, interquartile range; L_{den}, noise level day-evening-night; MODE, modal aerosol model for Europe; MONICA, multinational monitoring of trends and determinants in cardiovascular disease; NO_2, nitrogen dioxide; O_3, ozone; OR, odds ratio; PM, particulate matter; PM10, particulate matter with aerodynamic diameter ≤10 μm; PM2.5, particulate matter with aerodynamic diameter ≤2.5 μm; ALL, from all local sources; IND, from local industry; TRA, from local traffic; noTRA, model output without local traffic sources; noIND, model output without local industry sources; RR, relative risk; SES, socio-economic status; WHO, World Health Organization.

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Conclusions: Long-term exposures to all-source and industry-specific $PM_{2.5}$ were positively related to BP. We could not separate the effects of industry-specific $PM_{2.5}$ from all-source $PM_{2.5}$. Estimates with traffic-specific $PM_{2.5}$ were generally higher but inconclusive.

1. Introduction

According to the Global Burden of Disease Study, long-term air pollution is an important risk factor, mostly contributing to chronic diseases in adults (Lim et al., 2012). Exposure to particulate matter (PM) air pollution is linked to the development and progression of cardiopulmonary diseases and mortality (Brook et al., 2010). Of particular interest is the relationship of PM air pollution with arterial blood pressure (BP) in the general population, because high BP is the most important risk factor for mortality and disability worldwide (Lim et al., 2012). Even small increases or decreases in BP on the population level are linked to major changes in population-attributable risk for cardiovascular disease and mortality (Whelton et al., 2002).

Associations of long-term air pollution with elevated BP and/or hypertension have been observed in recent studies from Canada, China, Spain, Taiwan, and the USA (Chan et al., 2015; Chen et al., 2013; Chuang et al., 2011; Coogan et al., 2012; Dong et al., 2013; Johnson and Parker, 2009; Schwartz et al., 2012), as well as in our previous analysis of the Heinz Nixdorf Recall (HNR) cohort in Germany (Fuks et al., 2011). However, the evidence is not consistent. For example, in our recent cross-sectional meta-analysis of 15 European cohorts, high residential traffic exposure was positively associated with BP in participants taking BP lowering medication (BPLM) and with an elevated odds ratio (OR) for prevalent hypertension, while no consistent associations of air pollutants with BP or hypertension were observed (Fuks et al., 2014). Moreover, a negative association of long-term exposure to traffic-specific air pollution with BP and prevalent hypertension and no association with incident hypertension have been reported in a large population-based cohort in Denmark (Sørensen et al., 2012).

One reason for diverging results might be the diverse nature of particulate matter across study regions. PM air pollution originates from a number of natural and anthropogenic sources and is a heterogeneous mixture of solid and liquid particles that have different chemical composition, structure, or size, determining PM toxicity. The major industrial sources of primary and secondary fine PM in West Germany are energy production, foundries and steel plants, bulking of minerals and ores, and stockpiles deflation, emitting PM that contains metallic compounds, mineral oxides, sulfur dioxide, carbon and wood dust (Kuhlbusch et al., 2009). Traffic, another important source of within-city gradients of fine PM, emits particles from tailpipe emissions, including mainly submicron particles of soot and organic carbon, from tire, break and street abrasion and re-suspension, and submicron secondary particles generated from gaseous precursors (Kuhlbusch et al., 2009). It is therefore necessary to take different components or sources of the complex PM mixture into account when analyzing long-term effects on BP, as they may act differently on the cardiovascular system.

Another reason for inconsistencies between study results could be the difficulties in taking different degrees of BP medication into account, which obscures the true underlying BP. Cross-sectional analysis of air pollution and blood pressure, the most common analysis in the literature so far, may be compromised by participants' concurrent intake of BPLM, which prevents the analysis of the true underlying blood pressure (Tobin et al., 2005). Specific analyses using right censoring of blood pressure values in those under medication and analysis of incidence of hypertension might therefore provide more reliable assessment of PM action on blood pressure.

In this study, we expand our previous work on long-term exposure to PM and BP/hypertension by comparing the associations of PM from two major local anthropogenic emissions, namely industry (chemical, energy production, steel industry, construction, recycling) and traffic, with BP and hypertension. We use longitudinal health data from a large, well-characterized population-based cohort with follow-up measurements of BP to conduct cross-sectional and longitudinal analysis, and to account for BPLM intake when analyzing BP.

2. Materials and methods

2.1. Study population

We used baseline (2000–2003) and five year follow-up data from the ongoing HNR Study, a population-based, prospective cohort study from 3 adjacent cities of Essen, Bochum and Muelheim, situated in the highly urbanized Ruhr Area in Western Germany (Schmermund et al., 2002). In total, 4814 subjects aged 45–75 years participated at baseline, and 4157 took part in the five-year follow-up investigation. The cross-sectional analysis sample consisted of 4359 participants with non-missing information on exposure, BP, BPLM and covariates (Supplemental material, Fig. S1). In the longitudinal analysis, we excluded participants with prevalent hypertension at baseline (N = 2269), participants with missing data (N = 83) and those who had changed their residence during the follow-up period (N = 335). The resulting analysis sample consisted of 1470 participants

The study was approved by the local institutional review board. All participants gave informed consent. The HNR participants submitted a self-administered questionnaire, underwent face-to-face computer-assisted interviews for personal risk factor assessment, and physical examinations including anthropometric measurements and comprehensive laboratory tests.

2.2. Exposure assessment

2.2.1. Air pollution

We used the time-dependent three-dimensional European Air Pollution Dispersion Chemistry Transport Model (EURAD-CTM). This model has been evaluated in two studies so far (Büns et al., 2012; Memmesheimer et al., 2004). The details on exposure model evaluation can be found in the Supplemental material. The EURAD-CTM is a sophisticated multi-layer and multi-species Eulerian grid system, which allows simulation of transport, chemical transformation and deposition of gaseous pollutants (nitrogen oxides, ozone, sulphur dioxide, ammonia, and carbon dioxide) and PM (PM_{10}, $PM_{2.5}$, PM_{coarse}, and PM_1) on a regional and local scale (Büns et al., 2012; Memmesheimer et al., 2004). This model is operationally used for daily air quality forecasts in Europe, Central Europe, and the state of North Rhine-Westphalia in Germany (Büns et al., 2012; Nonnemacher et al., 2014). The EURAD-CTM model uses a sequential nesting method to generate initial and boundary values on smaller scales (Memmesheimer et al., 2004). The nesting scheme includes the following domains: the European

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