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Long-term exposure to residential ambient fine and coarse particulate matter and incident hypertension in post-menopausal women

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ABSTRACT

Background: Long-term exposure to ambient particulate matter (PM) has been previously linked with higher risk of cardiovascular events. This association may be mediated, at least partly, by increasing the risk of incident hypertension, a key determinant of cardiovascular risk. However, whether long-term exposure to PM is associated with incident hypertension remains unclear.

Methods: Using national geostatistical models incorporating geographic covariates and spatial smoothing, we estimated annual average concentrations of residential fine ($PM_{2.5}$), respirable (PM_{10}), and course ($PM_{10-2.5}$) fractions of particulate matter among 44,255 post-menopausal women free of hypertension enrolled in the Women's Health Initiative (WHI) clinical trials. We used time-varying Cox proportional hazards models to evaluate the association between long-term average residential pollutant concentrations and incident hypertension, adjusting for potential confounding by sociodemographic factors, medical history, neighborhood socioeconomic measures, WHI study clinical site, clinical trial, and randomization arm.

Results: During 298,383 person-years of follow-up, 14,511 participants developed incident hypertension. The adjusted hazard ratios per interquartile range (IQR) increase in $PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$ were 1.13 (95% CI: 1.08, 1.17), 1.06 (1.03, 1.10), and 1.01 (95% CI: 0.97, 1.04), respectively. Statistically significant concentration-response relationships were identified for $PM_{2.5}$ and PM_{10} fractions. The association between $PM_{2.5}$ and hypertension was more pronounced among non-white participants and those residing in the Northeastern United States.

Conclusions: In this cohort of post-menopausal women, ambient fine and respirable particulate matter exposures were associated with higher incidence rates of hypertension. These results suggest that particulate matter may be an important modifiable risk factor for hypertension.

1. Introduction

Air pollution, and especially fine particulate matter ($PM_{2.5}$) is an established risk factor for adverse cardiovascular health outcomes (Brook et al., 2010; Gehring et al., 2006; Miller et al., 2007; Dockery et al., 1993; Pope et al., 2002; Abbey et al., 1999). In 2012, the World Health Organization (WHO) attributed 7 million deaths worldwide - one of every eight deaths - to air pollution, with nearly 80% of these due to cardiovascular causes (WHO, 2014). The elevated cardiovascular morbidity and mortality associated with $PM_{2.5}$ may be explained, in part, by its increasing the risk of hypertension.

Hypertension is a highly prevalent, established risk factor for cardiovascular disease (James et al., 2014). As of 2009–2012, 70 million American adults had hypertension, and an additional 33% had pre-hypertension (Nwankwo et al., 2013). Hypertension is known to increase the risk of death by myocardial infarction, stroke, heart

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failure and kidney disease, with 360,000 deaths directly or indirectly attributable to hypertension in 2013 (Mozaffarian et al., 2015). While a number of modifiable (obesity, physical inactivity, poor diet, alcohol and tobacco use) and non-modifiable (age, family history) hypertension risk factors have been described (NIH, 2006), a growing body of evidence implicates air pollution as a possible risk factor. Specifically, several studies have identified positive associations between markers of long-term exposure to $PM_{2.5}$ and hypertension prevalence (Dong et al., 2013; Babisch et al., 2014) or blood pressure elevations (Chuang et al., 2010a; Chan et al., 2015), while others have found no association (Fuks et al., 2011; Sorensen et al., 2012). A separate body of literature has evaluated the association between daily changes in pollutant levels and blood pressure measures (Yang et al., 2012; Franck et al., 2011). However, only a few previous studies have investigated links between long-term air pollution and incident hypertension (Sorensen et al., 2012; Zhang et al., 2016; Coogan et al., 2012; Chen et al., 2013). Of these studies, only one has been performed in the context of a large, national US cohort, and that study examined associations with self-reported hypertension (Zhang et al., 2016). As hypertension is an established risk factor for adverse cardiovascular health outcomes (James et al., 2014), additional research is needed to further elucidate the association between $PM_{2.5}$ and the risk of incident hypertension. Additionally, while a number of studies have linked $PM_{2.5}$ to hypertension and blood pressure, much less is known about the potential associations of long-term exposure to other particulate matter size fractions (i.e. $PM_{10-2.5}$, PM_{10}), which may or may not be associated with hypertension risk (Bell et al., 2009; Franck et al., 2014; Straney et al., 2014; Shah et al., 2013; Fuks et al., 2011; Zanobetti and Schwartz, 2009).

To address these gaps in the literature, we examined the association between long-term exposure to various particulate matter size fractions ($PM_{2.5}$, $PM_{10-2.5}$, PM_{10}) and the risk of incident hypertension in a prospective cohort of post-menopausal women.

2. Methods

2.1. Population

Data from the Women's Health Initiative clinical trials (WHI CT) was used to quantify the association between incident hypertension and air pollution exposure. The WHI is a large, national, prospective cohort study of post-menopausal women aged 50–79 years at enrollment focused on investigating strategies for the prevention of heart disease, cancer, and osteoporosis morbidity and mortality (Anderson et al., 2003). The WHI CT included 68,132 women recruited between 1993 and 1998; randomized into trials evaluating the effects of hormone replacement therapy (n = 27,347), dietary modification (n = 48,835), and calcium/vitamin D supplementation (n = 36,282) and followed until 2005 (see Supplemental material for additional details of the study inclusion/exclusion criteria and recruitment details). A subsequent five-year extension (2005–2010) was conducted during which 82.4% of the original WHI cohort (n = 52,174) continued to be followed. Our study followed participants originally enrolled in all WHI CTs from enrollment (1993–1998) through the end of the first study extension (2010). Participants who did not have data on follow-up time in the first extension (n = 221) were excluded, affording 67,911 participants for the current analysis.

2.2. Exposure assessment

Daily $PM_{2.5}$ measurements obtained from the US Environmental Protection Agency's (EPA) AQS and IMPROVE networks were used to calculate annual averages of $PM_{2.5}$ (EPA, 2009; IMPROVE, 2013). Using these data, partial least-squares regression models incorporating a number of geographic covariates were used in a national, universal kriging model to estimate average $PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$ concentrations across the United States for each participant. The geocoded address history of all WHI participants from baseline through 2010, accounting for changes of address, at baseline, and for each year of follow-up, were linked with specific exposure estimates using geographic information systems software (Vine et al., 1997). The model has previously been shown to predict concentrations with high cross-validation accuracy for both $PM_{2.5}$ ($R^2 = 0.88$) and PM_{10} ($R^2 = 0.40–0.63$) (Bergen et al., 2013; Sampson et al., 2013). Estimates of coarse particulate matter exposure ($PM_{10-2.5}$) were calculated by subtracting the estimated $PM_{2.5}$ for a given time interval from the estimated PM_{10}. Annual moving average estimates of $PM_{2.5}$, PM_{10}, and $PM_{10-2.5}$ were calculated for 1980–2010.

2.3. Outcome assessment

At baseline and then annually through 2005, blood pressure was ascertained at WHI clinical centers after participants had been seated for 5 min using standardized procedures (Anderson et al., 2003; Margolis et al., 2008). Two separate measurements were taken ≥ 30 s apart from the right arm in all participants with a conventional mercury blood pressure cuff at baseline and at each subsequent visit (Anderson et al., 2003; Margolis et al., 2008). The mean of the two measurements from each visit were calculated for use in analyses.

At the time of study enrollment, WHI participants were queried whether they have been diagnosed with high blood pressure or hypertension by a physician and/or whether they were taking medications prescribed to treat hypertension. As in previous studies from WHI (Wassertheil-Smoller et al., 2000), participants were considered to have prevalent hypertension if at enrollment they had: a systolic blood pressure (SBP) ≥ 140 mm Hg, a diastolic blood pressure (DBP) ≥ 90 mm Hg, a history of physician-diagnosed hypertension, or reported use of anti-hypertension medication. Based upon these criteria, 34.8% of participants (n = 23,656) were identified as having hypertension at baseline and were thus excluded from analysis. From 1993 to 2005, presence or absence of hypertension was assessed annually for each participant using both conventional sphygmomanometer measurements and self-reported anti-hypertensive medication use (participants were queried: “Do you now take pills for high blood pressure”). Blood pressure was measured annually using standard procedures (described above). From 2005 to 2010, participants self-reported new diagnoses of hypertension and/or new use of medication prescribed for hypertension on standardized questionnaires. As in previous studies (Kingsley et al., 2015; Margolis et al., 2012), we defined incident hypertension as first self-report of medication prescribed for hypertension, SBP ≥ 140 mm Hg, or DBP ≥ 90 mm Hg.

2.4. Covariates

Potential confounders were assessed by self-administered questionnaires at time of study enrollment and annually thereafter. Demographic covariates included age and race/ethnicity (Asian, Hispanic, Native American/Alaskan Native, Black, White). Socioeconomic covariates included educational attainment (completed graduate school, completed college/vocational school versus other), household income (> $100,000 per year, $50,000–$100,000 per year versus < $50,000 per year), employment status (current versus other), insurance coverage (current versus other), and a U.S. Census tract-level, neighborhood socioeconomic status (SES) summary Z-score of wealth/income, education and occupation (Roux et al., 2001). Health behavior covariates included smoking status (current/historical or never), self-reported sodium intake and physical activity level (quantified as average metabolic equivalents per week). Health status variables included body mass index (BMI), self-reported history of coronary artery disease, diabetes and high cholesterol. Additionally, indicator variables for 36 unique WHI study clinical sites were included to control for potential geospatial confounding. Information on participa-