Environmental Research 161 (2018) 345–353

Contents lists available at ScienceDirect

Environmental Research

journal homepage: www.elsevier.com/locate/envres

Impacts of exposure to black carbon, elemental carbon, and ultrafine particles from indoor and outdoor sources on blood pressure in adults: A review of epidemiological evidence

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ARTICLE INFO

Keywords:
Air pollution
Black carbon
Ultrafine particles
Blood pressure
Epidemiology

ABSTRACT

Introduction: Ambient particulate air pollution is known to have detrimental effects on cardiovascular health but less is known about the specific effects of black carbon or elemental carbon (BC/EC) and ultrafine particles (UFP).

Methods: We present a narrative review of the epidemiological evidence related to the impact of exposure to BC/EC and UFP on blood pressure in adults. We searched PubMed and EMBASE in September 2017, using a predefined search strategy. Abstracts were screened using predefined inclusion criteria. Data collection was completed using a standard data extraction form. We focused on main effect estimates for associations between short (≤7 days) and long-term exposures to BC/EC and UFP and systolic (SBP) and diastolic blood pressure (DBP). Effect estimates were rescaled to enable direct comparisons between studies.

Results: Thirty publications were included in the review: 19 studies examined outdoor exposure to BC/EC, 11 examined outdoor UFP, three studies examined indoor BC and one study examined indoor UFP. In general, existing evidence supports a positive association between BC/EC and blood pressure. Evidence for outdoor UFP exposures were less clear as effect estimates were small in magnitude and confidence intervals often included the null.

Conclusions: Existing evidence supports a positive association between BC/EC and blood pressure in adults, whereas UFPs do not appear to have a meaningful impact on blood pressure.

1. Introduction

Three of the leading contributors to global disease burden include elevated systolic blood pressure, ambient particulate air pollution, and household air pollution from solid fuel burning which are thought to be responsible for millions of premature deaths on an annual basis (Forouzanfar et al., 2016). Moreover, since the adverse health effects of particulate air pollution are driven largely through their detrimental impact on cardiovascular health (Forouzanfar et al., 2016), reducing population exposures to air pollution may have an important impact on population health worldwide.

Numerous studies have evaluated the relationship between short and long-term exposure to fine particulate air pollution ($PM_{2.5}$) and changes in blood pressure, and existing evidence generally supports a positive association (Giorgini et al., 2016; Liang et al., 2014). Moreover, recent cohort studies have reported small but statistically significant associations between long-term exposure to ambient $PM_{2.5}$ and incident hypertension (Chen et al., 2014; Zhang et al., 2016). Possible biological mechanisms explaining the relationship between $PM_{2.5}$ and blood pressure include altered cardiovascular autonomic regulation resulting in increased sympathetic nervous system activation as well as vascular dysfunction resulting from reduced bioavailability of endogenous nitric oxide (Liang et al., 2014; Pieters et al., 2012). However, less is known about the potential impact of other air pollutants on systolic and diastolic blood pressure including important combustion-related pollutants such as ultrafine particles (< 0.1 μm; UFP) and black carbon or elemental carbon (BC/EC) which may also contribute to cardiovascular morbidity (Luben et al., 2017; Weichenthal, 2012). Indeed, these pollutants have also been associated with changes in heart rate variability (a marker of autonomic function) and peripheral arterial tone (a measure of endothelial function) and thus may also contribute to changes in blood pressure (Bind et al., 2016; Huang et al., 2013; Weichenthal,

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https://doi.org/10.1016/j.envres.2017.11.030
Received 31 May 2017; Received in revised form 15 November 2017; Accepted 19 November 2017

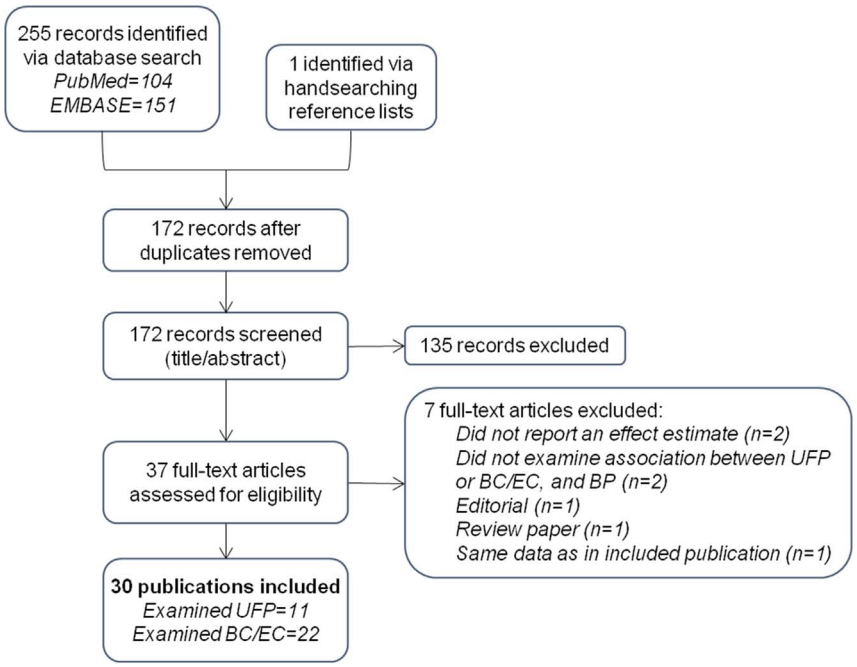

Fig. 1. Flow diagram for review of the association between ultrafine particles (UEP) black carbon (BC) or elemental carbon (EC) and blood pressure (BP).

2012; Weichenthal et al., 2011). In this review, we evaluated existing epidemiological evidence related to the impact of short and long-term exposures to BC/EC and UFP on blood pressure.

2. Methods

Studies were identified using PubMed (1966 to current) and EMBASE (1996 to current) in September 2017. The search strategy combined a search term for *blood pressure*, with pollutant specific search terms for UFP *(ultrafine particle, ultrafines, UFP, number concentration, traffic particle, nucleation mode, accumulation mode, Aitkin mode, $PM_{0.1}$)* and for BC/EC (*black carbon, black particles, soot, black smoke, elemental carbon*). The title and abstract fields were searched in PubMed and keyword search terms were used in EMBASE; when available the search included MeSH terms in PubMed, and subject heading terms in EMBASE (Online Appendix 1: Search Strategy). Date restrictions were not applied to the searches. Search results were imported into EndNote and de-duplicated. Reference lists of included papers were also examined for additional publications meeting the inclusion criteria.

To be included in the review, publications had to: (i) report an effect estimate (i.e. beta coefficient and corresponding confidence intervals) for the association between continuously measured/modeled air pollutant concentrations and continuous measures of blood pressure; (ii) be conducted in adults (≥18 years); and (iii) had to be written in English. We excluded studies reporting only p-values (with no effect estimates reported), controlled exposure/chamber studies, studies of manufactured nanoparticles, and occupational studies. Abstracts were screened by one reviewer (S.M.) and the studies that met the inclusion criteria were reviewed by a second reviewer (S.W.). Full-texts were reviewed if we were unable to determine eligibility solely based on the title and abstract. For all title/abstracts meeting the inclusion criteria the full-texts were reviewed to confirm eligibility. Each publication that met the inclusion criteria was comprehensively reviewed using a data extraction form developed specifically for this review and was pilot tested prior to use (Online Appendix 2: Data Extraction Items). One reviewer (S.M.) extracted all data from the included publications, and any uncertainties were resolved through discussion.

Given that we focused on continuous exposures and outcomes, the effect measure of interest was the mean change in blood pressure associated with a unit change in air pollutant levels, or a percent change in blood pressure per unit change in air pollutant levels. We considered main effects only. If possible, the effect estimates were rescaled in order to estimate the change in blood pressure associated with the same unit change in air pollutant level, to allow for direct comparisons across studies. The following units were used: 10,000/cm^3 for UFP and 1 μg/m^3 for BC/EC. Unless otherwise stated, the effect estimates reported in this review are the rescaled values. To calculate rescaled 95% confidence intervals, we halved the width of the reported 95% confidence intervals, as an approximation of the standard error, and used this approximation to calculate rescaled confidence intervals. We contacted corresponding authors when the necessary information was not reported in the paper to enable rescaling of effect estimates. Rescaled effect estimates are presented using forest plots, which were stratified by outcome scale (per unit increase in mmHg and percent change), duration of exposure (short and long-term exposure) and exposure measurement type (personal/area and fixed-site monitors). We grouped area exposure monitors with studies that used personal exposure monitors; area exposure monitors are defined as exposure monitors that were installed onsite close to study participants. Both lagged exposures (e.g. averaged air pollution levels for the third day prior to the blood pressure measurement) and cumulative exposures (e.g. averaged air pollution levels from time of the blood pressure measurement to three days prior) were considered.

We organized our review based on pollutant and duration of exposure measurement. In particular, studies are summarized by the length of exposure time that was measured in the study: dichotomized into short-term and long-term exposure studies. Studies in which exposure was measured for 7 days or less were classified as short-term exposure measurement studies; and those that measured exposures for time periods greater than 7 days were classified as long-term exposure measurement studies. We recognize that exposure measurement over a shorter-term time period may be representative of longer-term exposure. However, we used this classification to stratify studies based on the actual time period that the air pollutant was measured in the study. For simplicity we use the terms short-term exposure and long-term exposure, throughout, to refer to the actual duration of exposure measurement used in a given study.