Environment International 88 (2016) 228–234

Contents lists available at ScienceDirect

Environment International

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

Short-term fluctuations in personal black carbon exposure are associated with rapid changes in carotid arterial stiffening

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ARTICLE INFO

Article history:
Received 3 July 2015
Received in revised form 17 December 2015
Accepted 19 December 2015
Available online 11 January 2016

Keywords:
Arterial stiffness
Carotid artery
Air pollution exposure
Black carbon
Epidemiology

ABSTRACT

Background: Vascular changes may underpin the association between airborne black carbon (BC) and cardiovascular events. Accurate assessment of personal exposure is a major challenge in epidemiological research. BC concentrations are strongly related to time–activity patterns, which is particularly relevant when investigating short-term effects. We investigated associations between arterial stiffness and personal short-term BC exposure.
Methods: This panel study included 54 healthy adults (92% women, mean age 40.7 years). BC exposure was monitored individually with a micro-aethalometer during one workweek. Functional and structural properties of the carotid artery were examined ultrasonographically on two separate days. The effect of different short-term personal BC exposure windows (1, 2, 4, 6, 8, 24 and 48 h before the ultrasound examination) on carotid artery stiffness was estimated using mixed models while adjusting for other known correlates of arterial stiffness.
Results: Median personal BC exposures within the same day ranged from 599.8 to 728.9 ng/m^3 and were associated with carotid arterial stiffness measures. Young's elastic modulus and pulse wave velocity, both measures of stiffness, were positively associated with BC exposure, while the distensibility and compliance coefficient, measures of elasticity, were negatively associated with BC exposure. The strongest associations were observed with BC exposure 8 h before the clinical examination. For each 100 ng/m^3 increase in exposure within this time window, Young's elastic modulus increased by 2.38% (95% CI: 0.81 to 3.97; $P = 0.0033$), while the distensibility coefficient decreased by 2.27% (95% CI: −3.62 to −0.92; $P = 0.0008$).
Conclusions: Short-term elevations in personal BC exposure, even within hours, are associated with increased arterial stiffness. This response may reflect a pathway by which air pollution triggers cardiovascular events.

1. Introduction

Increases in particulate air pollution levels have been associated with an increase in cardiovascular morbidity and mortality in numerous epidemiological studies (Nawrot et al., 2011; Brook et al., 2010; Pope and Dockery, 2006). Altered cardiac autonomic function (Pieters et al., 2012; Sun et al., 2010; Mills et al., 2009), atherosclerosis (Sun et al., 2010; Mills et al., 2009; Provost et al., 2015) and changes in vascular function (Sun et al., 2010; Mills et al., 2009; Brook et al., 2002) are potential pathophysiological pathways through which particulate air pollution can influence the cardiovascular system.

Evidence from animal studies indicates that particulate matter can initiate and accelerate atherosclerosis (Chen et al., 2010; Araujo and Nel, 2009; Niwa et al., 2007; Sun et al., 2005; Suwa et al., 2002). Different epidemiological studies show an association between long-term exposure to particulate air pollution and intima-media thickness. Intima-media thickness is a parameter for vascular structural changes that is frequently used as a subclinical marker of atherosclerosis (Bauer et al., 2012). Measures of arterial stiffness on the other hand provide a manner to investigate functional vascular changes. Acute changes in vascular stiffness may be a factor in explaining how acute exposure to particulate air pollution can trigger cardiovascular events, such as myocardial infarction (Vlachopoulos et al., 2010). However, epidemiological studies investigating the association between short-term exposure to particulate air pollution and arterial stiffness are limited (Adamopoulos et al., 2010; Mehta et al., 2014). Research by Mehta and colleagues shows that short-term increases in air pollution levels, that were monitored centrally, were

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http://dx.doi.org/10.1016/j.envint.2015.12.023
0160-4120/

associated with changes in the augmentation index and augmentation pressure, both measures of vascular stiffness (Mehta et al., 2014). Adamopoulos et al. report cross-sectional associations between augmentation pressure and short-term exposure to particulate matter with a diameter smaller than 10 μm (PM_{10}) in men (Adamopoulos et al., 2010).

Black carbon (BC) is formed during traffic-related combustion and BC concentration is used as a general measure for exposure to traffic particles (Richmond-Bryant et al., 2009). BC is considered as an important component of particulate air pollution that induces adverse health effects (Janssen et al., 2011; Grahame et al., 2014). The estimation of individual BC exposure levels is a challenge in epidemiological research. Most studies evaluate individual exposure based on central monitoring stations, estimation models or proxies for exposure such as distance to major roads. These approaches do not consider the fact that individual exposure is strongly related to time–activity patterns (Dons et al., 2011). Modeled exposure based on the place of residence may result in exposure misclassification, especially for traffic-related BC with a high spatio-temporal gradient (Dons et al., 2011; Alexeeff et al., 2015). Such exposure misclassification may bias results toward the null, leading to an imprecise estimation of the health effects associated with particulate air pollution exposure and BC in particular (Alexeeff et al., 2015). Better personal estimates of BC in relation to location can now be obtained with small continuous BC sensors (Dons et al., 2011; Nieuwenhuijsen et al., 2015; Setton et al., 2011).

We report the results of a repeated measures study that analyzed markers related to vascular structure, i.e. carotid intima-media thickness, and to vascular function, i.e. carotid artery stiffness parameters in a cohort of healthy adults. We investigated for the first time the association of the latter parameters with personal short-term exposure to BC.

2. Materials and methods

2.1. Study population

130 nurses from two hospitals in Belgium, i.e. Ziekenhuis Oost-Limburg (ZOL, Genk) and Universitair Ziekenhuis Antwerpen (UZA, Antwerp), were invited to participate in the study. 99 (76%) nurses agreed to participate, of which 56 (56%) were assigned at random to this study. The number of participants was fixed because of the limited availability of the BC measuring devices. We conducted our study according to the principles outlined in the Helsinki declaration for research on human participants. The ethics committee of Hasselt University and Universitair Ziekenhuis Antwerpen approved the study. All participants gave written informed consent. A questionnaire provided detailed information on social and medical characteristics of the participants, including transportation modes, use of medication, smoking habits and incidence of cardiovascular disease in their family. All participants reported to be free of clinical cardiovascular diseases and diabetes.

A panel study design was used to investigate the association between measures of arterial stiffness and short-term exposure to BC. BC exposure of the participants was monitored continuously during 7 consecutive days of an average workweek between April and May 2013. Repeated clinical examinations of the same participant were performed on two separate days within this period, i.e. on days 3 and 6. The examination took place between 7.00 h and 19.00 h at the hospital where the nurses worked. During the clinical examination, participants were questioned on their current medication, caffeine and alcohol use, current smoking status and time spent in traffic during the past 24 h. Smokers ($n = 2$) were excluded from all statistical analyses.

2.2. Health measurements

Clinical examinations were performed by one trained observer and included an ultrasound examination of the common carotid artery, blood pressure and heart rate measurements and a single blood test at the end of the week. Measurements on non-fasting blood included blood cell distribution, serum creatinine, HDL and total cholesterol, blood glucose level and γ-glutamyltransferase (GGT) level, a biomarker for alcohol intake.

2.2.1. Carotid stiffness measures

Ultrasound measurements were performed by one trained investigator using an ultrasound device with automatic boundary detection software in RF-mode (MyLabOne, Esaote Benelux, Maastricht, The Netherlands) according to previously reported protocols (Stein et al., 2008). Longitudinal scanning of a 1 cm segment of the right common carotid artery (CCA) at 1 cm proximal to the dilatation of the carotid bulb visualizes the lumen–intima and media–adventitia interfaces of the far arterial wall. Carotid intima-media thickness (CIMT) was determined under three different angles; i.e. 90, 130 and 180° using Meijer's Arc with the participants at rest for 10 min in a supine position, with their head slightly turned to the left, according to the recommendations of the Mannheim Carotid Intima-Media Thickness Consensus (Touboul et al., 2007). Measurements obtained at the three angles were averaged.

The carotid distensibility (DC) and compliance (CC) coefficients are inversely related to arterial stiffness such that higher values of these parameters represent less stiffness; whereas pulse wave velocity (PWV) is a direct measure of arterial stiffness. These parameters are derived from the ultrasound measurements averaged over 8 cardiac cycles and from supine brachial blood pressure measured during the ultrasound examination. We computed the distensibility and compliance coefficients from the diastolic cross-sectional area (A), the systolic increase in cross-sectional area (ΔA) and the local pulse pressure (ΔP) according to the formula: $DC = (\Delta A/A) / \Delta P$ and $CC = \Delta A / \Delta P$ (O'Rourke et al., 2002). A and ΔA were calculated as $A = \pi \times (D/2)^2$ and $\Delta A = \pi \times [(D + \Delta D) / 2]^2 - \pi \times (D/2)^2$. Pulse wave velocity was calculated as $PWV = 1 / \sqrt{\rho \times DC}$ with ρ as blood density. Young's Elastic Modulus (YEM) combines measures of arterial wall elasticity with wall thickness and increases in YEM represent an increase in arterial stiffness. Young's elastic modulus is calculated as $YEM = D/(CIMT \times DC)$.

Intra-observer coefficients of variation ranged from 5.61% to 11.91% for the different stiffness parameters, indicating good reproducibility of the measurements. These results are in line with previously published results on variability and reproducibility of carotid structural and functional parameters assessed with transcutaneous ultrasound by Caviezel and colleagues in the SAPALDIA cohort (Caviezel et al., 2013).

2.2.2. Blood pressure and heart rate

Blood pressure and heart rate were measured according to the guidelines of the European Society of Hypertension (Parati et al., 2008). Participants rested for five minutes, after which heart rate (HR), systolic (SBP) and diastolic (DBP) blood pressure were measured five times consecutively with an automated device (Omron 705 IT, MSH, Glabbeek, Belgium). The average of the last three measurements was used to calculate mean arterial pressure.

2.3. Personal BC exposure assessment

Measurements of BC were recorded in one minute intervals using the portable MicroAeth® Model AE51 BC aerosol monitor (AethLabs, San Francisco, CA, USA). A short tube was attached to the inlet of the device, which allowed participants to put the portable device in their pocket, purse or backpack. The filter was replaced every two days to prevent filter saturation. The participants were instructed to take the instrument wherever they went. When the participants were indoors, they were allowed to keep the instrument static in the room where the majority of their time was spent.

Air was drawn in over a Teflon-coated borosilicate glass fiber filter at a flow rate of 100 ml/min. The attenuation of light was measured at a