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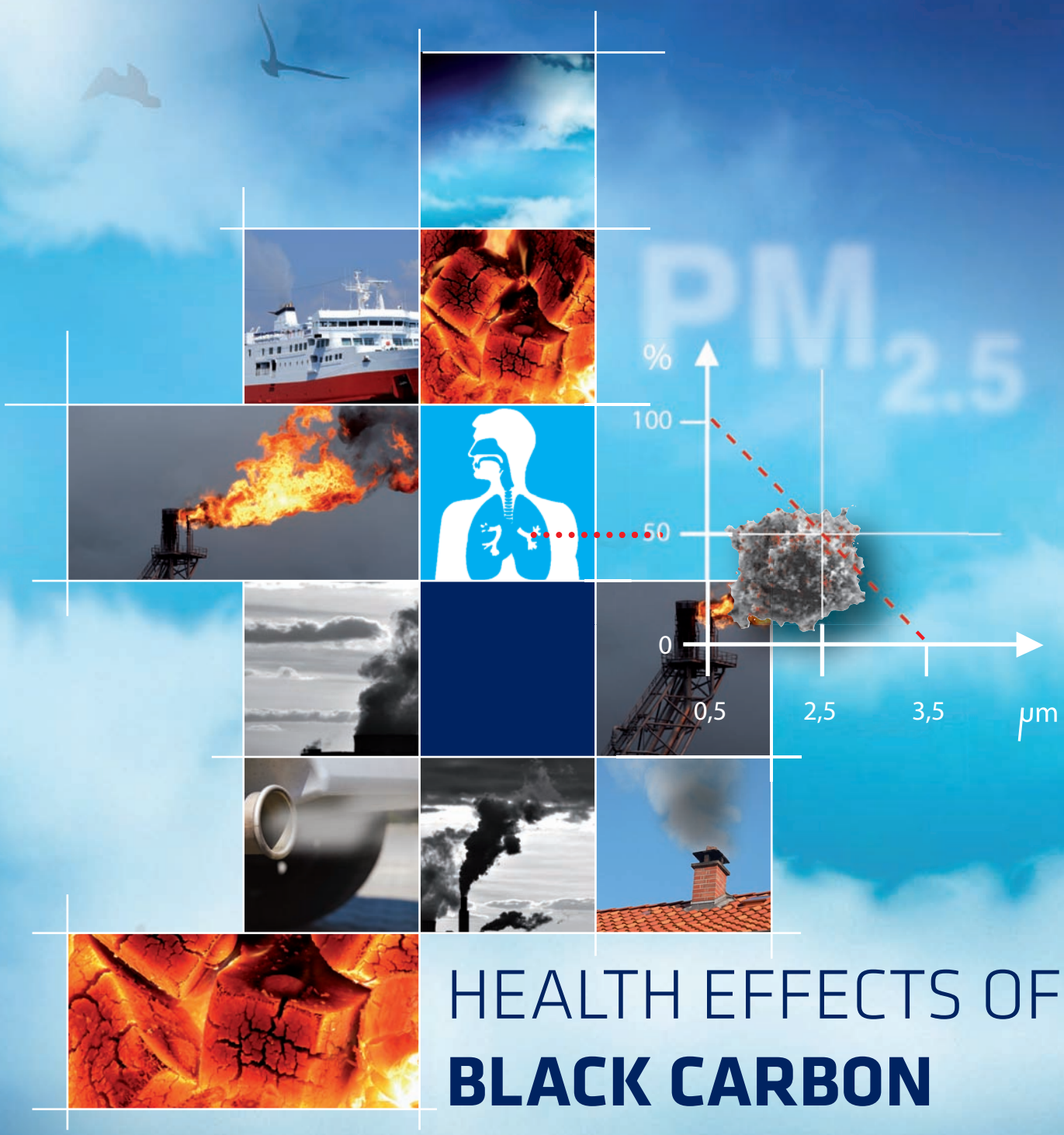

HEALTH EFFECTS OF **BLACK CARBON**

World Health Organization
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Health effects of black carbon

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ABSTRACT

This report presents the results of a systematic review of evidence of the health effects of black carbon (BC). Short-term epidemiological studies provide sufficient evidence of an association of daily variations in BC concentrations with short-term changes in health (all-cause and cardiovascular mortality, and cardiopulmonary hospital admissions). Cohort studies provide sufficient evidence of associations of all-cause and cardiopulmonary mortality with long-term average BC exposure. Studies of short-term health effects suggest that BC is a better indicator of harmful particulate substances from combustion sources (especially traffic) than undifferentiated particulate matter (PM) mass, but the evidence for the relative strength of association from long-term studies is inconclusive. The review of the results of all available toxicological studies suggested that BC may not be a major directly toxic component of fine PM, but it may operate as a universal carrier of a wide variety of chemicals of varying toxicity to the lungs, the body's major defence cells and possibly the systemic blood circulation. A reduction in exposure to $PM_{2.5}$ containing BC and other combustion-related PM material for which BC is an indirect indicator should lead to a reduction in the health effects associated with PM.

Keywords

AIR POLLUTION – adverse effects
SOOT – toxicity
INHALATION EXPOSURE – adverse effects
PARTICULATE MATTER – analysis
RISK ASSESSMENT

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Convention on Long-range Transboundary Air Pollution

Abbreviations

Abs	absorbance
BC	black carbon
BCP	black carbon particles
BS	black smoke
CVD	cardiovascular disease
DE	diesel engine exhaust
EC	elemental carbon
IQR	inter-quartile range
NIOSH	National Institute for Occupational Safety and Health
OC	organic carbon
PAH	polycyclic aromatic hydrocarbons
PM	particulate matter
POM	particulate organic matter
RSS	rice-straw smoke
RR	relative risk
TOR	thermal optical reflectance
TOT	thermal optical transmittance
UFP	ultrafine particles

Executive summary[1]

Following decision 2010/2 of the Executive Body for the Convention on Long-range Transboundary Air Pollution (ECE/EB.AIR/106/Add.1, para 8(b)(i)), the Task Force on Health Aspects of Air Pollution working under the Convention conducted an assessment of the health effects of black carbon (BC) as a component of fine particulate matter ($PM_{2.5}$). The Task Force's discussion focused on formulating the conclusions presented below, on the basis of the working papers prepared for it and comments received from external reviewers.

BC is an operationally defined term which describes carbon as measured by light absorption. As such, it is not the same as elemental carbon (EC), which is usually monitored with thermal-optical methods. Current measurement methods for BC and EC need to be standardized so as to facilitate comparison between the results of various studies. The main sources of BC are combustion engines (especially diesel), residential burning of wood and coal, power stations using heavy oil or coal, field burning of agricultural wastes, as well as forest and vegetation fires. Consequently, BC is a universal indicator of a variable mixture of particulate material from a large variety of combustion sources and, when measured in the atmosphere, it is always associated with other substances from combustion sources, such as organic compounds. The spatial variation of BC is greater than that of $PM_{2.5}$. Although, in general, ambient measurements or model estimates of BC reflect personal exposures reasonably well and with similar precision as for $PM_{2.5}$, the differences in exposure assessment errors may vary between studies and possibly affect estimates of risk.

The systematic review of the available time-series studies, as well as information from panel studies, provides sufficient evidence of an association of short-term (daily) variations in BC concentrations with short-term changes in health (all-cause and cardiovascular mortality, and cardiopulmonary hospital admissions). Cohort studies provide sufficient evidence of associations of all-cause and cardiopulmonary mortality with long-term average BC exposure.

Health outcomes associated with exposure to $PM_{2.5}$ or thoracic particles (PM_{10}) are usually also associated with BC (and vice versa) in the epidemiological studies reviewed. Effects estimates (from both short- and long-term studies) are much higher for BC compared to PM_{10} and $PM_{2.5}$ when the particulate measures are expressed per unit of mass concentration ($\mu g/m^3$). Effect estimates are, however, generally similar per inter-quartile range in pollutant levels. Studies of short-term health effects show that the associations with BC are more robust than those with $PM_{2.5}$ or PM_{10}, suggesting that BC is a better indicator of harmful particulate substances from combustion sources (especially traffic) than undifferentiated PM mass. In multi-pollutant models used in these studies, the BC effect estimates are robust to adjustment for PM mass, whereas PM mass effect estimates decreased considerably after adjustment for BC. The evidence from long-term studies is inconclusive: in one of the two available cohort studies, using multi-pollutant models in the analysis, the effect estimates for BC are stronger than those for sulfates, but an opposite order in the strength of relationship is suggested in the other study.

[1] Also published as part of *Effects of air pollution on health. Report of the Joint Task Force on Health Aspects of Air Pollution* (2011). Geneva, United Nations Economic and Social Council (ECE/EB.AIR/WG.1/2011/11) (http:// www.unece.org/fileadmin/DAM/env/ documents/2011/eb/wge/ece.eb.air.wg.1.2011.11.pdf, accessed 12 December 2011).

There are not enough clinical or toxicological studies to allow an evaluation of the qualitative differences between the health effects of exposure to BC or to PM mass (for example, different health outcomes), of quantitative comparison of the strength of the associations or of identification of any distinctive mechanism of BC effects. The review of the results of all available toxicological studies suggested that BC (measured as EC) may not be a major directly toxic component of fine PM, but it may operate as a universal carrier of a wide variety of, especially, combustion-derived chemical constituents of varying toxicity to sensitive targets in the human body such as the lungs, the body's major defence cells and possibly the systemic blood circulation.

The Task Force on Health agreed that a reduction in exposure to $PM_{2.5}$ containing BC and other combustion-related PM material for which BC is an indirect indicator should lead to a reduction in the health effects associated with PM. The Task Force recommended that $PM_{2.5}$ should continue to be used as the primary metric in quantifying human exposure to PM and the health effects of such exposure, and for predicting the benefits of exposure reduction measures. The use of BC as an additional indicator may be useful in evaluating local action aimed at reducing the population's exposure to combustion PM (for example, from motorized traffic).

Introduction

The health effects of combustion-related air pollution indicated by black particles were identified decades ago, when the monitoring of black smoke (or "British smoke" – BS) was a widespread method for air quality assessment in Europe. The evidence about the health effects of this pollution was used to recommend the first guidelines for exposure limits (then) consistent with the protection of public health (WHO, 1979). In the 1990s, BS was one of the indicators of air quality used, for example, in European time-series studies linking mortality with pollution (Katsouyanni et al., 2001). A recognition of the difficulties in standardizing BS measurements and an appreciation of the health effects of the non-black components of particulate matter (PM) attracted the attention of researchers and regulators to the mass concentration of inhalable or respirable fractions of suspended PM such as PM_{10} and $PM_{2.5}$ (WHO Regional Office for Europe, 2000). BS is not addressed by air quality regulations and the intensity of BS monitoring has decreased.

New scientific evidence has led to a recognition of the significant role of black particles (black carbon – BC) as one of the short-lived climate forcers. Measures focused on BC and methane are expected to achieve a significant short-term reduction in global warming. If they were to be implemented immediately, together with measures to reduce CO_2 emissions, the chances of keeping the earth's temperature increase to less than 2 °C relative to pre-industrial levels would be greatly improved (UNEP, 2011). The same measures would also directly benefit global health and food security.

The synergy between action to address global warming and air quality has been noted by the parties to the Convention on Long-range Transboundary Air Pollution. Taking into account the conclusions of the report of the Ad Hoc Expert Group on Black Carbon (UNECE, 2010a), the Executive Body of the Convention decided to include consideration of BC, as a component of PM, in the revision process of the 1999 Gothenburg Protocol to Abate Acidification, Eutrophication and Ground-level Ozone (Gothenburg Protocol) (UNECE, 2010b). The Executive Body also requested the Joint Task Force on the Health Aspects of Air Pollution (the Task Force on Health) to look at the adverse effects on human health of black carbon as a component of $PM_{2.5}$.

There is still no systematic comparison of health effects estimated using PM versus BC indicators. A WHO working group has acknowledged that the evidence on the hazardous nature of combustion-related PM (from both mobile and stationary sources) was more consistent than that for PM from other sources (WHO Regional Office for Europe, 2007). Grahame & Schlesinger (2010) reviewed the evidence of the effects of BC on cardiovascular health endpoints and concluded that it may be desirable to promulgate a BC $PM_{2.5}$ standard. Conversely, Smith et al. (2009) noted that although the results of their time-series meta-analysis suggest greater effects per unit mass of sulfate than BS, this distinction was less clear in the few studies that directly compared the estimated effects of both indicators. This indicates the need for a critical comparison of studies that have measured PM mass as well as BC particles.

In response to the request from the Executive Body of the Convention, and in view of the lack of a systematic review of the accumulated evidence on the health effects of BC, the Task Force on Health launched the review by addressing the following specific questions.

1. What metrics have been used to estimate the health effects of exposure to BC?
    a. What are their respective strengths and weaknesses?
    b. How is personal exposure related to ambient levels?
2. What are the effects of BC exposure observed in epidemiological studies (health outcomes, exposure/response function)?
    a. What are the effects of short-term exposure?
    b. What are the effects of long-term exposure?
    c. Are they different qualitatively (for example, different health outcomes) and/or quantitatively from the effects of:
        i. $PM_{2.5}$ mass concentration
        ii. other measured components of $PM_{2.5}$?
3. What are the effects of BC in the human controlled exposure experiments? Are they different qualitatively (for example, different health outcomes) and/or quantitatively from the effects of:
    a. $PM_{2.5}$ mass concentration
    b. other measured components of $PM_{2.5}$?
4. What are the mechanisms of the effects of BC indicated by toxicological studies?
    a. Are they different from the mechanisms of effects attributed to undifferentiated $PM_{2.5}$ mass concentrations or other measured components of $PM_{2.5}$?
    b. Is there evidence supporting the thesis that (some of) the mechanisms are specific for BC?

Leading the Task Force on Health, WHO invited selected experts to prepare concise background papers summarizing evidence corresponding to each of the above questions. The experts signed the WHO declaration of interest, assuring the absence of any conflicts of interests related to their contributions to the assessment. The papers were based on a systematic review of the literature, with relevant documentation of the protocol of the review and of the evidence reviewed (see Annex 1).

The conclusions of the review were prepared by WHO and the authors of the background papers based on the papers. The summary also rated the quality of the evidence supporting each conclusion based on the approach used in the WHO *Indoor air quality guidelines* (WHO Regional Office for Europe, 2010, p 6). Both the papers and the summary were subject to review by another group of experts, and their comments were made available to all members of the Task Force on Health in advance of the 14th Task Force Meeting, held in Bonn on 12–13 May 2011 (list of participants in Annex 2). The discussion at the Meeting focused on finalizing the summary assessment, which has been published in the Task Force Report (UNECE, 2011). This summary also forms the Executive Summary of this report.

The background papers presented in this report were revised after the Task Force Meeting, based on the comments of the reviewers before and at the Meeting.

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1. Metrics used to estimate the exposure to BC in health studies: strengths and weaknesses

Raimo O Salonen

Introduction

There are several types of measurement method and commercial instrument available for continuous, semi-continuous and integrated filter sample-based optical and thermal-optical measurements of aerosol parameters reflecting combustion-derived **char, soot**, **black carbon** or **elemental carbon** contents in PM. The concentrations of these carbonaceous material are low or moderate (close to source) in atmospheric PM, and much higher in emissions from common combustion sources (diesel engines, power plants or ship engines using heavy oil, or small residential heaters using wood or other biomass).

The following are explanations of the bolded terms in common language according to Han et al. (2007; 2010).

- Char is defined as carbonaceous material obtained by heating organic substances and formed directly from pyrolysis, or as an impure form of graphitic carbon obtained as a residue when carbonaceous material is partially burned or heated with limited access of air (typical of burning vegetation and wood in small residential heaters).
- Soot is defined as only those carbon particles that form at high temperature via gas-phase processes (typical of diesel engines).
- Black carbon (BC) refers to the dark, light-absorbing components of aerosols that contain two forms of elemental carbon.
- Elemental carbon (EC) in atmospheric PM derived from a variety of combustion sources contains the two forms “char-EC” (the original graphite-like structure of natural carbon partly preserved, brownish colour) and “soot-EC” (the original structure of natural carbon not preserved, black colour) with different chemical and physical properties and different optical light-absorbing properties.

A thermal optical reflectance method can be applied to differentiate between char-EC and soot-EC according to a stepwise thermal evolutional oxidation of different proportions of carbon under different temperatures and atmosphere (more details under Measurement methods of the dark component of PM, below). The health significance of the separate char-EC and soot-EC is not known. In general, EC or BC are regarded as having negligible toxic effects on human and animal lungs in controlled studies and on airway cells such as macrophages and respiratory epithelial cells. Instead, it has been suggested that they exert an indirect key role in toxicity as a universal carrier of toxic semi-volatile organics and other compounds co-released in combustion processes or attached to their surface during regional and long-range transport (see Chapter 4).

The optimal combustion of fuel at high temperature, such as the current low-sulfur fossil diesel fuel in modern diesel engines, results in the emission of large numbers of very small soot particles (aerodynamic diameter 1–5 nm) that rapidly grow in size (10–100 nm) in the tailpipe by coagulation to form aggregated chains, and further by condensation of the simultaneously released semi-volatile organic substances on their surfaces in the atmosphere. The speed of growth depends on air temperature, sunlight, concomitant oxidants, etc. (D’Anna, 2009).

The burning of solid fuels, such as wood and coal, is usually not optimal, especially in small residential heaters, since there is, to a varying degree, incomplete smouldering combustion due to the relative shortage of oxygen. Subsequently, the aerodynamic diameter of emitted PM in flue gas becomes larger (150–600 nm) than in the case of diesel oil combustion in car engines, because in addition to thermochemically-formed EC there are incompletely burnt tar-like organics attached to it. As with diesel car PM, these emitted PM continue to grow in the atmosphere by condensation of semi-volatile organics on their surface. The combustion of solid fuels, such as wood and coal, tends to produce much larger amounts of semi-volatile organics than combustion of low-sulfur diesel oil (Naeher et al., 2007; Kocbach Bolling et al., 2009).

While ageing in the atmosphere for several hours or days, the combustion-derived particles become even larger (up to 1 μm in diameter) because inorganic salts originating from both NO_2 and SO_2, together with atmospheric water, attach to the surfaces of hygroscopic carbonaceous particles.

Taking into account the wide variations in the formation and composition of combustion-derived PM, and the fact that some of its chemical composition is known to exert not only light-absorbing (soot/BC/EC) but also considerable light-scattering (organics, inorganics) properties, it is no surprise that many indirect optical measurement techniques and thermal optical analysis methods, which have been used for many years in air quality measurements by aerosol and by health scientists, have proved to give only a rough proxy of the BC or EC concentration in ambient air without instrument-specific corrective measures. Some methods have also had instrument-specific technical problems during operation in large methodological inter-comparison studies conducted by the leading aerosol scientists in Asia, Europe and the United States (Müller et al., 2011; Chow et al., 2009; Reisinger et al., 2008; Kanaya et al., 2008; Hitzenberger et al., 2006).

Measurement methods of the dark component of PM

Combustion-derived soot and char (in practice, their dark components) have been determined in epidemiological studies by the following techniques:

- light reflectance from (absorbance (Abs), BS) or light transmission through (basis of measurement of BC) integrated PM samples usually collected at 24-hour intervals on thin cellulose fibre filter or other filter material, followed by conversion of the optical measurement units to mass-based units;
- real-time photometers measuring light absorption of PM sample spots (BC) at 1–5 minute intervals and automatically giving readings in mass-based units;
- chemical determination of EC and organic carbon (OC) using thermal optical analysis methods either semi-continuously with mass-based readings given every 30 minutes to 3 hours, or from integrated PM samples collected at 24-hour intervals on quartz filters (Müller et al., 2011; Janssen et al., 2011; Chow et al., 2009).

The absorption coefficient of PM and BS measured with a reflectometer and BC measured with an optical transmissometer are metrics that are based on the blackness of aerosol material collected on a filter. Light is focused on the filter sample and the amount of light reflected or transmitted is measured. For BS and Abs, the amount of reflected light is converted into PM mass units (OECD standard) (OECD, 1964) or the black smoke index ISO standard 9835:1993 (ISO, 1993), whereas in the BC method the light transmitted is converted to represent the mass of EC. BS measurement has been used in Europe since the 1920s, when urban air pollution was dominated in many places by smoke from coal combustion. Although BS and Abs determinations are expressed in $\mu g/m^3$, there is no clear relationship to PM mass, as conversion

of the optical measurement results into mass units depends on location, season and type of combustion particle.

Absorption photometers for real-time application have been available since the 1980s. These are filter-based instruments that measure at intervals of one to five minutes the changes in transmittance through a fibrous filter tape as particles are deposited. The complex relationship between changes in light transmission and aerosol absorption and scattering on the filter requires an adequate calibration of these methods, including the selection of an effective wavelength for a valid absorption co-efficient, determination of filter spot size and characterization of the aerosol flow (Müller et al., 2011). Algorithms have been published for correcting artefactual enhancement of light absorption by filter-loading, back-scattering, and multiple scattering caused by PM and the filter matrix in connection with aethalometers and particle soot absorption photometers. The multi-angle absorption photometer is the only real-time absorption photometer that corrects for these artefacts by design (Müller et al., 2011; Chow et al., 2009) (Table 1).

Thermal optical methods are based on OC and EC removed from sampling substrates (such as quartz-fibre filter) by volatilization and/or combustion at selected temperatures, and by conversion of the released gases to carbon dioxide (CO_2) or methane (CH_4). This is followed by infrared absorption (CO_2) or flame ionization (CH_4) detection. EC is not volatile and is only released by oxidation. Most of the atmospheric OC tends to evolve at temperatures ≤550 °C in pure helium atmosphere and, thus, it can be separated from EC that needs to be oxidized in helium 98%/oxygen 2% at temperatures ≥550 °C. Heating in an inert helium atmosphere, however, causes certain OC compounds to pyrolyse or char, thereby exaggerating the atmospheric EC in the sample. In thermal optical carbon analysis, this can be corrected by simultaneous measurement of thermal optical reflectance (TOR) or thermal optical transmittance (TOT). Although the principles of thermal methods appear to be similar, they contain variations with respect to: location of the temperature monitor (thermocouple) relative to the sample, analysis atmospheres and temperature ramping rates; temperature plateaus; residence time at each plateau; optical pyrolysis monitoring configuration; carrier gas flow through or across the sample; and oven flushing conditions. Chow et al. (2005; 2009) and Han et al. (2007; 2010) have done a lot of development and comparisons of thermal optical methods. Currently, their Interagency Monitoring of Protected Visual Environments (IMPROVE_A) thermal optical reflectance protocol (IMPROVE_A_TOR) seems the best thermal optical method for separating various OC fractions from each other as well as for separating char-EC from soot-EC (Table 1).

Comparison of the optical measurement methods with each other and with more sophisticated methods

BS/PM_{10} ratios measured with the reflectometer have varied widely in Europe and many times exceeded one in some locations (Hoek et al., 1997), as the Abs units are converted to BS values in $\mu g/m^3$ by using a constant conversion factor. This is a major source of bias, because the greatly varying OC/EC ratio in PM affects Abs due to scattering of light from combustion-type organic material. A typical OC/EC ratio in urban traffic environments is two, while the OC/EC ratio can be five in rural background areas with more prevalent biomass combustion. Thus, BS data from different types of site or from different seasons or from decade-long time-series at the same site are not comparable. BS measurement should always be accompanied by local calibration of the conversion factor from Abs units to BS values in $\mu g/m^3$ on the basis of the OC/EC ratio in PM (Schaap & Denier van der Gon, 2007).

Table 1. Summary of methodological aspects in relation to measurement of light Abs, BS, BC or EC in atmospheric PM

PM metrics	General information	Methodological principle	Strengths and limitations
PM Abs, BS, BC. Cheap and simple measurements from integrated filter samples	*Reflectometer*. Collection of usually 24-hour PM samples on Whatman paper filter at sampling flow volume of 2±0.2 m^3/day, absorption coefficient measured from PM on filter using simple reflectometer consisting of a light source and a detector (ISO 9835:1993 (E)). Originally, there was an OECD standard (1964) for BS measurement from total suspended particulate samples. *Optical transmissometer.* This portable instrument can perform rapid, non-destructive BC determination from PM material collected on different types of filter (diameter 25 mm, 37 mm or 47mm). The instrument has a movable tray with two filter-holder slots, one inside and the other outside. The outside holder is used to measure light attenuation through the sample filter, while a simultaneous measurement is made through the reference (blank) filter placed in the inside holder. The analysis time for an individual measurement is less than one minute.	*Reflectometer*. Analogue or digital readout of either percentage reflectance (linear scale, recommended range 35–95%) or absorption coefficient (logarithmic scale, recommended range 0.64–13.13 × 10^{-5}) that can be transformed into a BS index (ISO 9835, 1993). According to the OECD standard (1964), there is a conversion of the reflectance data into gravimetric units (μg/m^3). The same has been done with absorption by using a fixed conversion factor: 1 unit of Abs equals an increase of 10 μg/m^3 BS (Roorda-Knape et al., 1998). *Optical transmissometer.* The OT-21 is based on the optics used in some aethalometer models. It measures the transmission intensity of light at 880 nm and 370 nm passing through a particle-loaded filter and determines the attenuation of light compared to the intensity of a blank filter.	*Reflectometer*. Standardized, traditional and cheap method; long time-series in several central European countries according to the OECD (1964) specifications. Baseline reflectance of unused filters may vary from batch to batch. Scattering of light from PM sample rich in organics or due to some inorganics results in biased reflectance values. BS (R^2=0.82–0.93) and absorption (R^2=0.85-0.98) methods have had high correlations with thermal optical EC, but the slopes of the association show wide variations (BS 10 μg/m^3 equals EC 0.5–1.8 μg/m^3) (Janssen et al., 2011). A study in the Netherlands showed that BS readings depended on the OC/EC ratio in ambient air (r^2=0.85 for urban sites and r^2=0.75 for rural sites) and the slopes of association varied with the type of measurement site and local combustion sources (Schaap & Denier van der Gon, 2007). *Optical transmissometer*. The results obtained from three different types of site in the United States (New York State) and one site in Turkey showed that the relationships between BC values obtained from the OT-21 and thermal optical BC values from a semi-continuous carbon analyser were linear. The slopes for the data from the sites varied from 0.75 to 1.02 (r^2= 0.44 to 0.88), which was mainly attributed to the different chemical composition of aerosols as well as their age in the atmosphere. When the data were combined and plotted as monthly average BC, the two methods showed excellent agreement (slope 0.91, r^2=0.84) (Ahmed et al., 2009).
BC. Absorption photometers for real-time application (averageging time 1–5 minutes).	*Real-time absorption photometers*. Filter-based instruments measure the change of transmittance through a fibrous filter tape as	*Aethalometer* (Hansen, Rosen & Novakov, 1984). Offered in different configurations. Multispectral (370–950 nm) absorption coefficients provide insight into chemical composition in PM sample. PM	*Unit-to-unit variability between similar instruments*. Up to 30% for PSAPs and aethalometers, while less than 5% for multi-angle absorption photometers. Reasons for the high variability

PM metrics	General information	Methodological principle	Strengths and limitations
	particles are deposited. The complex relationship between change in light transmission and aerosol absorption and scattering on the filter requires a calibration of these methods (effective wavelength for valid absorption coefficient, determination of filter spot size, aerosol flow characterization) (Muller et al., 2011). Published algorithms for correction of artefactual enhancement of light absorption by filter-loading, backscattering and multiple scattering by PM and the filter matrix in connection with aethalometers and particle soot absorption photometers. Multi-angle absorption photometers correct by design for these artefacts (Muller et al., 2011; Chow et al., 2009; Kanaya et al., 2008).	collection on quartz-fibre filter tape, flow rate 6.7 litres/ minute and averaging time 5 minutes. *Particle soot absorption photometers* (Bond, Anderson & Campbell, 1999). Absorption coefficients measured at variable wavelengths (467–660 nm). Dependence of response on PM size and cross-sensitivity to particle scattering that can be controlled by simultaneously measured nephelometer data. PM collection on glass-fibre filter tape, typical flow rate 0.5–1 litre/minute and averaging time 3 seconds. *Multi-angle absorption photometers* (Petzold & Schonlinner, 2004). Measures radiation transmitted through and scattered back from a PM-loaded filter. A two-stream radiative transfer model used to minimize the cross-sensitivity to particle scattering. Usual emission at wavelength 670 nm. PM collection on glass-fibre filter tape, flow rate 16.7 litres/minute. Minimum detection limit as specified by the manufacturer is BC<0.1 μg/m^3 with an averaging time of 2 minutes (Chow et al., 2009).	were identified as variations in sample flow and spot size and as cross-sensitivity to PM scattering (Müller et al., 2011). Correlations in absorption coefficients between different instruments. Particle soot absorption photometers versus multi-angle absorption photometers (R2=0.96–0.99), aethalometers versus multi-angle absorption photometers (R2=0.96) (Muller et al., 2011). In a campaign in the United States (Fresno supersite), agreement in BC between corrected aethalometers (660 nm) and multi-angle absorption photometers (670 nm) was within 1%. BC concentrations determined with the semi-continuous carbon analyser were highly correlated (R≥0.93) but were 47% and 49% lower than BC measured with aethalometers and multi-angle absorption photometers, respectively (Chow et al., 2009). Elevated BC-to-EC ratios with multi-angle absorption photometers possibly connected to biomass-derived abundant OC fraction volatilizing at high temperatures (Reisinger et al., 2008, Kanaya et al., 2008) and to aged BC with coating by transparent materials causing a lensing effect in optical measurements (Kanaya et al., 2008).
Comparison of absorption photometers with more advanced measurement techniques	*Photoacoustic instrument*. This is regarded as an unofficial reference or benchmark method for BC.	*Photoacoustic instrument* (Arnott et al., 1999). PM are drawn into a cavity and illuminated by a laser with the desired wavelength modulated at the resonant frequency of the cavity. The heating and cooling of the particle in response to the absorbed light creates a sound wave that is detected by a microphone. The intensity of the acoustic wave is related to PM light absorption by calibration with NO_2 absorption. Typical flow rate 1 litre/minute and averaging time 3–4 seconds.	*Comparison with photoacoustic instrument*. In the Fresno supersite campaign, uncorrected PM light absorptions with aethalometers were 4.7–7.2 times, and with PSAP 3.7–4.1 times, higher than those with a photoacoustic instrument. After applying published algorithms to correct for the artefacts, the adjusted values for aethalometers were 24–69% higher, and for PSAP 17–28% higher, than those for the photoacoustic instrument. The greater differences were at higher wavelengths. Multi-angle absorption photometers gave 51% higher PM light absorption than the photoacoustic instrument. However, all uncorrected and corrected aethalometer, particle soot absorption photometer and multi-angle absorption photometer data were highly corre-

PM metrics	General information	Methodological principle	Strengths and limitations
			lated (R≥0.95) with photoacoustic instrument data (Chow et al., 2009). *Comparison with thermal optical methods*. The average differences between BC concentration by adjusted 7-AE (660 nm) and multi-angle absorption photometers (670 nm) versus EC concentration by IMPROVE_A_TOR were 0 and 6%, respectively. The BC analysed by semi-continuous carbon analyser using the National Institute for Occupational Safety and Health (NIOSH) 5040_TOT protocol (660 nm) was 47% lower than the EC analysed by IMPROVE_A_TOR. In all comparisons, correlations were r≥0.87 (Chow et al., 2009).
Comparisons between EC/OC thermal optical methods	*IMPROVE_A_TOR/TOT protocol*. PM collected on a quartz-fibre filter at ambient temperature and pressure is subject to thermal carbon analysis following the IMPROVE_A protocol using the DRI Model 2001 thermal/optical carbon analyser. The correction for pyrolysed OC is done by monitoring laser reflectance (TOR) or laser transmittance (TOT). *STN TOR/TOT protocol*. PM collection the same as above. Thermal/optical transmission/reflectance analysis applied to the US $PM_{2.5}$. *Speciation Trends Network* (STN). Filter transmittance is monitored to split OC and EC (STN_TOT). With the DRI Model 2001 thermal/optical carbon analyser, reflectance can also be recorded during the analyses (STN_TOR). *Semi-continuous carbon analyser_TOT*. PM collected on the quartz fibre filter tape is	*IMPROVE_A_TOR/TOT*. The evolved carbon is converted to CO_2 and reduced to CH_4 that is detected using a flame ionization detector. Pure helium is used as the carrier gas in stepwise rising temperatures from 30°C to 550 °C or 580 °C to separate various OC fractions from each other. The separation of various EC fractions from each other is done in helium 98%/oxygen 2% at temperatures from 550 °C or 580 °C to 800 °C or 840 °C: char-EC separated from soot-EC at around 700 °C or 740 °C (Chow et al., 2009; Han et al., 2007; Chow et al., 2005). Reports 24-hour concentrations of EC and OC (including their sub-fractions), total carbon and PM light absorption. *STN TOR/TOT*. Pure helium is used as the carrier gas in stepwise rising temperatures from 30 °C to 900 °C to separate various OC fractions. Helium 98%/oxygen 2% is applied to EC fractions at temperatures from 600 °C to 920 °C. Reports 24-hour concentrations of EC, OC and total carbon. *Semi-continuous carbon analyser_TOT*. Evolved CO_2 is analysed by the non-dispersive infrared sensor. In the NIOSH 5040 protocol. Pure helium is used as the carrier gas in stepwise rising temperatures from 30 °C to 840 °C for various OC fractions. Helium 98%/oxygen 2% is applied to EC fractions at tempe-	*IMPROVE_A_TOR/TOT*. The residence time (150–580 seconds) at each temperature plateau in the IMPROVE_A protocol is flexible to achieve well-defined carbon fractions, and depends on when the flame ionization detector signal returns to the baseline (Chow et al., 2005; 2009). *STN TOR/TOT and NIOSH 5040_TOT*. The STN protocol has short and fixed residence times (45–120 seconds), as does the NIOSH 5040 protocol (30–120 seconds) for each temperature plateau. They cannot, therefore, report distinguishable carbon fractions. *Comparison between thermal optical protocols*. In the Fresno supersite study, 24-hour EC concentration by TOR was 23% higher than EC by TOT following the IMPROVE_A protocol, and 29% higher following the STN protocol. These differences were smaller when TOR was used to determine the OC/EC split. EC by STN_TOR was 10% lower than by IMPROVE_A_TOR. NIOSH 5040_TOT of the semi-continuous carbon analyser gave 45% lower integrated 24-hour EC concentration than that by IMPROVE_A_TOR. In all cases, the pairwise correlations were r≥0.87 (Chow et al., 2009).

PM metrics	General information	Methodological principle	Strengths and limitations
	subjected to thermal optical analysis following the NIOSH 5040_TOT protocol. Typical flow rate 8.5 litres/minute and averaging time 1 hour. Used as a field instrument for air quality and health studies.	ratures from 550 °C to 850 °C. Laser transmittance (TOT) is used to correct for pyrolysis. During the PM collection phase, light transmission through the filter is monitored to quantify BC similarly to aethalometers. All measurements at 660 nm. Reports 1-hour concentrations of BC, EC, OC and total carbon for ambient conditions.	

The variability in the chemical composition of BC aerosol at different locations also biases the BC data of optical transmissometers. It has been suggested that these should be calibrated with the help of more sophisticated and reliable measurement techniques using statistically significant numbers of samples for the specific sites (Ahmed et al., 2009). As with reflectometers, however, controlling the measurement bias by local calibrations may not be easy, because the OC/EC ratio in PM can also vary with the season and with day-to-day temperatures at the same site due to variations in biomass combustion for residential heating.

Aerosol scientists have produced valuable information about the type and quantity of sources of measurement error in relation to absorption photometers for real-time application (Müller et al., 2011; Chow et al., 2009; Reisinger et al., 2008; Kanaya et al., 2008; Hitzenberger et al., 2006). In fact, the use of filter-based instruments to derive information on aerosol light Abs and BC is a matter of debate (Müller et al., 2011), as is the use of older optical measurements of BS and Abs (see Janssen et al., 2011). Currently, there is no generally accepted standard method to measure BC or EC. It has, however, been possible to make comparisons of several filter-based instruments of aerosol light Abs with more sophisticated instruments such as the photoacoustic analyser (Chow et al., 2009).

Several workshops have been conducted to investigate the performance of individual instruments, for example, two workshops with large sets of aerosol absorption photometers in 2005 and 2007. The data from these instruments have been corrected using existing methods, but still the most recent inter-comparison has shown relatively broad variations in responses to PM light absorption in connection with some instruments (Müller et al., 2011). Significant biases associated with filter-based measurements of PM light absorption, BC and EC are method-specific. Correction of these biases must take into account the variations in aerosol concentration, composition and sources (Chow et al., 2009).

The key results from the comparisons of the real-time optical measurement methods with each other and with more sophisticated methods of measuring BC and EC, and from the comparisons of BS and Abs with EC (Janssen et al., 2011) are summarized in Table 1. The literature search and the criteria for selection of the literature cited are described in Annex 1.

Conclusions

BC is an operationally defined term, which describes carbon as measured by light absorption. As such, it is not the same as EC, which is usually monitored with thermal-optical methods. Despite intensive efforts during the past 20 years, there are no generally accepted standard methods to measure BC or EC in atmospheric aerosol. While most of the measurement methods of BC or

EC seem to be well-correlated, biases in filter-based light absorption and thermal optical carbon measurements need to be identified and corrected for accurate determination of aerosol light absorption, BC and EC in different environments. Variations in the OC/EC ratio bias filter-based PM light absorption in addition to other artefacts. The multi-angle absorption photometer is currently the only type of real-time absorption photometer that corrects for these biases and artefacts of BC measurement by design. However, aethalometer data can be corrected using published algorithms. The IMPROVE_A protocol in thermal optical carbon analyser, equipped with laser reflectance (TOR) to correct for pyrolysed OC, currently seems to be the most reliable method to measure OC and EC concentrations from atmospheric PM in integrated filter samples. The flexible residence time (150–580 seconds) at each temperature plateau also enables the measurement of well-defined OC and EC sub-fractions, which may be useful in PM source analysis. At their best in a field campaign, the 24-hour concentrations of BC by multi-angle absorption photometer and from corrected aethalometer data have been nearly equal to the 24-hour EC concentration measured by IMPROVE_A_TOR. Current methods of measuring BC and EC need standardization to facilitate comparison between various study results.

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2. Assessment of exposure to BC in epidemiological studies

Timo Lanki

The light Abs of $PM_{2.5}$ filter samples has been used in most European epidemiological studies as a measure of exposure to black carbon particles (BCP), whereas in studies in the United States, the EC content of the samples has mostly been used for the purpose. In some studies, Abs has been further converted into BS, which was widely used in the past in Europe for air quality monitoring. However, the conversion factor found in ISO standard 9835:1993 (ISO, 1993) is not suitable for present-day particulate air pollution mixture, but local calibration factors should be used. In earlier studies, the coefficient of haze may have been used as a measure of BCP. Because of similar measurement principles, the method gives results that are highly correlated with BC concentrations obtained with more modern methods such as aethalometers. Real-time BC measurement methods will undoubtedly increase in popularity with time, especially in settings where filter sampling is not needed for other purposes.

Short-term exposures

Time-series study design has been the most frequently used method to evaluate the acute effects of BC exposure on population health. The design is based on comparing short-term (typically daily) variations in exposure with short-term variations in population health, for example, mortality or hospitalization. In the setting, population exposure is assessed by measuring BCP at one or more centrally located outdoor monitoring stations. The accuracy of estimates of the effects on health eventually depends on how well daily BCP levels measured at the central outdoor monitoring site (ambient BCP) reflect daily changes in personal exposure to BCP (personal BCP) in the study area. It should be noted that ambient concentration is a valid proxy for personal exposure even when an *individual's exposure* on a given day may not be predicted very accurately because of random error (Zeger et al., 2000). In contrast, an inability by outdoor monitoring to reflect daily *mean exposure* in the study population leads to biased health effect estimates. Panel studies with repeated clinical and air pollution measurements similarly rely on accurate assessment of day-to-day variability in exposure.

Only a few studies have evaluated longitudinal relationships between daily ambient and personal BCP concentrations (Table 2). Considering the large proportion of the 24-hour cycle typically spent in the home, the observation of a high correlation between repeated daily measurements of personal BCP and BCP indoors (indoor BCP) (median Pearson's $r > 0.9$ for individual regression results) is not surprising (Janssen et al., 2000). One study linking ambient BCP with indoor BCP has, therefore, been included in Table 2.

In European studies, the Abs of $PM_{2.5}$ filters has been used as a measure of BCP (Table 2). Ambient Abs was found to be more strongly associated with respective personal and indoor levels than $PM_{2.5}$ in these studies. It is noteworthy that indoor EC was reasonably correlated with indoor Abs ($R=0.57–0.85$) in the Dutch-Finnish study (Janssen et al., 2000), but the slope was different for homes with and without environmental tobacco smoke. In the studies included in this report, Abs has been measured from $PM_{2.5}$ filters, and thus size-fraction is not mentioned any more. $PM_{2.5}$ Abs has been reported as capturing most of the particulate Abs in ambient air (Cyrys et al., 2003).

Table 2. Relationships of ambient BCP and $PM_{2.5}$ with respective indoor and personal concentrations in longitudinal studies with repeated 24-hour measurements

Study population/ locations	Abs or EC (n)	Study area	Relationships between:				Reference
			ambient, personal BC	ambient, personal $PM_{2.5}$	ambient, indoor BC	ambient, indoor $PM_{2.5}$	
Individual regression results, median Pearson's r (slope)							
82 cardio-vascular patients	Abs (463)	Amsterdam, Netherlands	0.93 (0.92)	0.79 (0.43)	0.96 (0.84)	0.84 (0.47)	Janssen et al., 2000
		Helsinki, Finland	0.81 (0.62)	0.73 (0.45)	0.74 (0.49)	0.70 (0.51)	
152 homes	Abs (one week/ home)	Helsinki, Finland			0.79	0.70	Hoek et al., 2008b
		Athens, Greece			0.64	0.40	
		Amsterdam, Netherlands			0.92	0.80	
		Birmingham, United Kingdom			0.90	0.55	
Mixed models, r^2(slope)							
15 senior adults	EC (335)	Steubenville, United States (summer)	0.08 (0.33)	0.60 (0.73)			Sarnat et al., 2006b
		Steubenville, United States (autumn)	0.44 (0.70)	0.47 (0.63)			
25 homes	EC (167)	Boston, United States (winter)	0.30 (0.60)	0.17 (0.37)	0.30[a](0.91)	0.17 (0.29)	Brown et al., 2008
		Boston, United States (summer)	0.41 (0.08)	0.55 (0.75)	0.05 (0.29)	0.55 (0.89)	
Cross-sectional correlation, Pearson's r (slope)							
38 cardio-vascular patients	Abs (162)	Barcelona, Spain	0.69[b](0.67)	0.14[b](0.04)			Jacquemin et al., 2007

[a] Excluding one home with candle burning.
[b] Excluding days with ETS exposure.

Longitudinal studies conducted in the United States have evaluated BCP exposure as EC, and have found associations of ambient EC with personal EC to be similar or stronger than those of $PM_{2.5}$ during the winter (Table 2). It was speculated that the weak link between ambient and personal EC during the summer may be due to more measurement error and less variability in concentrations. Similarly in another study in the United States conducted mainly during the warm season (Delfino et al., 2006), no (cross-sectional) correlation was found between personal EC and ambient EC. Personal and indoor sampling of EC may be especially affected by errors in measurement because of lower concentrations.

A point estimate of an individual's exposure is dependent on long-term (for example, annual concentration at the residential area) and short-term (daily variations in concentration) components of exposure. Thus, correlations observed in cross-sectional exposure studies between daily ambient concentrations and personal exposures are of lesser value when interpreting time-series studies (that rely on within-person variability in exposure). In any case, cross-sectional studies also suggest that with the use of ambient measurements, exposure can be estimated at least as accurately for BCP as for $PM_{2.5}$ (Johannesson et al., 2007).

In Table 3, repeated "front-door" outdoor measurements of BCP (known as outdoor BCP: an outdoor measurement site as close as possible to the indoor measurements, on a balcony, in a garden or courtyard, etc.) have been linked with daily variations in indoor BCP. In most of the studies, outdoor concentrations of BCP were highly correlated with indoor concentrations. The slope of the corresponding regression equation can be interpreted as an infiltration factor, thus the results suggest that infiltration for BCP is somewhat more efficient than for $PM_{2.5}$. An exception is the German study conducted in a hospital building (Cyrys et al., 2004), where infiltration was less efficient for BCP than for $PM_{2.5}$. The authors hypothesized that BCP in the study area fell into the smallest size categories, for which penetration is less efficient but the deposition rate higher than for larger particles (included in $PM_{2.5}$). Indeed, the size of ambient BCP is not constant, but near emission sources (such as major roads) they are at their smallest and include ultrafine (aerodynamic diameter <100 nm) particles.

Table 3. Relation of daily outdoor BCP and $PM_{2.5}$ with respective indoor concentrations in studies with repeated measurements

Study locations	Study area	Abs/ EC	R BC	R $PM_{2.5}$	Infiltration BC	Infiltration $PM_{2.5}$	Reference
152 homes	Helsinki, Finland	Abs	0.96	0.74	0.63	0.48	Hoek et al., 2008b
	Athens, Greece		0.88	0.63	0.84	0.42	
	Amsterdam, Netherlands		0.96	0.85	0.78	0.39	
	Birmingham, United Kingdom		0.93	0.35	0.71	0.34	
25 homes	Boston, United States	EC	0.53[a,b]	0.55[a,b]	0.47[b]	0.53[b]	Brown et al., 2008
2 retirement communities in the Los Angeles basin[c]	Site A, summer	EC			0.73	0.71	Polidori et al., 2007
	Site B, autumn				0.71	0.60	
	Site A, winter				0.77	0.59	
	Site B, winter				0.64	0.45	
Hospital building	Erfurt, Germany	Abs	0.91	0.88	0.53	0.79	Cyrys et al., 2004
18 homes, 16 (pre)schools	Stockholm, Sweden	Abs			0.46	0.25	Wichman et al., 2010
28 homes	Huddersfield, United Kingdom	Abs	0.83	0.59			Kingham et al., 2000

[a] The paper reported R^2 for a mixed model.
[b] Excluding one home with candle burning.
[c] Outdoor measurement sites at a distance of 300 m from the buildings.

Outdoor BCP has also been found to be more strongly associated with respective indoor levels than $PM_{2.5}$ in cross-sectional studies (Gotschi et al., 2002). It should be noted that there are substantial differences in infiltration rates between geographical areas due to differences in building codes and human behaviour, and thus generalizability of the results from single (-city) studies is limited.

In the absence of indoor sources, indoor/outdoor concentration ratios can be interpreted to reflect infiltration directly. The effect of indoor sources can be eliminated by taking measurements at night or in an uninhabited building. Such studies have also reported higher infiltration for BCP than for $PM_{2.5}$: 0.84 versus 0.48 in Los Angeles homes (Sarnat et al., 2006a), and 0.61 versus 0.41 for a home in Clovis, California (Lunden et al., 2008).

Concentrations measured at a central outdoor site have been found to reflect well temporal variability in 24-hour concentrations of both $PM_{2.5}$ and BC across urban areas (Puustinen et al., 2007). Considering that BCP and $PM_{2.5}$ do not seem to differ in that respect, the higher infiltration rate for BCP may be the main reason for the observed higher ambient–personal correlation for BCP. Overall, measurement errors for BCP and $PM_{2.5}$ seem comparable, which means that effect estimates obtained in epidemiological studies for the two can be directly compared.

Even hourly peak exposures may be relevant to health as potential triggers of cardiorespiratory events. Although ambient 24-hour levels of BCP seem to reflect personal exposures well, it can be assumed that the correlation is lower on shorter time-scales due to short-term changes in ventilation (for example, opening windows at home or in a car) and the microenvironment (such as an office or in a public transport vehicle). Short-term BCP exposures are noticeably elevated during commuting (Adams et al., 2002), and the differences between background concentrations and concentrations measured in traffic by cyclists and passengers in vehicles seem to be even greater for BCP than for $PM_{2.5}$ (Zuurbier et al., 2010).

BCP also has significant indoor sources, such as cooking and environmental tobacco smoke, which may lead to peaks in exposure (Lanki et al., 2007; Raaschou-Nielsen et al., 2010). A reasonable assumption is that these indoor sources do not confound the association between ambient BCP and health outcomes because the strength of the source is not related to ambient levels.

Distinguishing between the effects of highly correlated air pollutants is always challenging because of potential problems caused by multi-collinearity in statistical models. The extent of correlation between ambient BCP and $PM_{2.5}$ does not rule out a calculation of reliable effect estimates in two-pollutant models (Table 4). It should, however, be noted that there is no single still acceptable value for a correlation coefficient (often limit of $R<0.7$ is used), but the robustness of the models should always be tested. Because of comparable infiltration factors, inter-correlations outdoors can be assumed also to reflect correlations between personal BCP and personal $PM_{2.5}$. Because BCP acts as an indicator for combustion particles and is measured from $PM_{2.5}$, two-pollutant models separate in practice between the health effects of combustion and non-combustion $PM_{2.5}$.

Daily variations in BCP in urban areas are most strongly associated with local traffic emissions (Vallius et al., 2005), although factors such as long-range transported air pollution, local industry, open biomass burning, and residential wood and coal combustion may also affect the concentrations (Larson et al., 2004). The considerable correlation between EC and OC suggests that the health effects associated in epidemiological studies with BCP may be at least partly due to organic compounds, which are typically not measured. Even if inter-correlations at some study areas allow two-pollutant models of BCP and OC, their interpretation is challenging because of common emission sources. The EC/OC ratio is location-specific and varies in time (Jeong et al., 2003; Schaap & Denier van der Gon, 2007), which further complicates reasoning on causal factors.

Long-term exposures

In the calculation of effect estimates in long-term epidemiological studies, contrasts in long-term exposure between persons are used. Consequently, the aim of exposure assessment is to accurately predict spatial variability in outdoor concentrations and further in personal exposures. For BCP, within-city variability in concentrations is larger than for $PM_{2.5}$ owing to the considerable effect of local combustion sources, especially traffic, on concentrations (Hoek et al., 2002; Janssen et al., 2008). Within-city variability may exceed between-city variability, which underlines the

Table 4. Correlations between daily outdoor $PM_{2.5}$, BCP and OC in longitudinal epidemiological studies

Study areas and years	BC/ EC	Correlation coefficients between outdoor			Reference
		BCP–$PM_{2.5}$	BCP–NO_2	BCP–OC	
Time-series/case-crossover studies					
Phoenix, United States (1995–1997)	EC	0.84	0.82	0.91	Mar et al., 2000
6 counties in California, United States (2000–2003)	EC	0.53		0.61	Ostro et al., 2007
119 counties in the United States (2000–2006)	EC	0.46		0.64	Peng et al., 2009
Boston, United States (1995–1999)	BC	0.66	0.40		Zanobetti & Schwartz, 2006
Vancouver, Canada (2000)	EC	0.17	0.24	0.92	Rich et al., 2004
Panel studies					
Steubenville, United States (2000)	EC	0.51	0.65		Sarnat et al., 2006c
St. Louis, United States (2001)	EC	0.53	0.62		Rich et al., 2006
Fresno, United States (2000–2005)	EC	0.76	0.68		Mann et al., 2010
Southern California, United States (2003)	EC	0.55	0.70	0.87	Delfino et al., 2006
Atlanta, United States (1999–2000)	EC	0.59	0.58		Suh & Zanobetti, 2010
Helsinki, Finland (1998–1999)	BC	0.70			Jacquemin et al., 2009
Amsterdam, Netherlands (1998–1999)	BC	0.73			

importance of taking into account small-scale variations in BCP in epidemiological studies. Vehicular traffic leads to marked BCP concentration gradients along busy roads (Roorda-Knape et al., 1998), and residential wood combustion, harbours, and point sources such as power stations may lead to lasting elevations in local BCP concentrations (Lu et al., 2006; Polidori et al., 2010; Snyder et al., 2010).

Some epidemiological studies on the long-term effects of BCP have relied on a crude estimation of exposure: BCP concentrations measured at a single outdoor monitoring site have been assumed to reflect exposure within a city or even over a whole county. In others, the monitoring network has been dense enough to allow interpolation of exposures over an urban area. Neither method is able sufficiently to take into account small-scale variations in BCP concentrations, which may lead to an underestimation of the effects of BCP. In contrast, land-use regression models have proved their efficiency in a number of recent studies (Table 5). Physico(chemical) dispersion models are another possibility, but they need substantial computational power and detailed information on emissions, which may not be available.

Land-use regression models are stochastic models that typically use predictor variables obtained through geographic information systems. These rather simple regression models can explain similar proportions of variability in long-term outdoor concentrations as can dispersion models (Hoek et al., 2008a). The explained proportion of BC variability has reached 80% in some studies, but a considerable variability in R^2 is also evident in Table 5. The difference between models in R^2 for the same location (Munich) shows that geography is not the sole reason for the differences in the performance of the models, but that the selection of variables is most important, as suggested by Hoek et al. (2008a). In Vancouver, R^2 for BCP was apparently later improved to 0.52 (from 0.41 in Table 5), but model validation results have not been presented (Brauer et al., 2008). In any case, based on the still limited number of studies available, it is possible to construct for BCP land-use regression models that perform even better than the

Table 5. Comparison of performance of land-use regression models for long-term $PM_{2.5}$ and BCP

Study area	Predictor variables for BC	Model R^2		RMSE[a]		Reference
		BCP	$PM_{2.5}$	BCP [$*10^{-5}m^{-1}$]	$PM_{2.5}$ [$\mu g/m^3$]	
Netherlands	High traffic roads 250 m, address density 300 m, distance to major road, region	0.81	0.73	0.31	1.59	Brauer et al., 2003
Munich, Germany	Traffic intensity 50 m and 50–250 m, population density 300 m and 300–5000 m	0.67	0.56	0.31	1.35	
Stockholm county, Sweden	Traffic intensity on nearest road, population density 1000–5000 m	0.66	0.50	0.22	1.10	
North Rhine-Westphalia, Germany	Heavy vehicle traffic 100–10 000 m, total traffic 100 m, distance to highway	0.82	0.17	0.16	2.3	Hochadel et al., 2006
Munich, Germany	Household density 2500–5000 m, distance federal road, land cover factor 100–250 m, length to country roads 1000 m	0.42	0.36	0.46	1.48	Morgenstern et al., 2007
Vancouver, Canada	Length to expressway 1000 m, length to major roads 100 m, distance to highway, open area 500 m	0.41	0.52	0.4	1.5	Henderson et al., 2007

[a] Root mean squared error of model validation.

models for $PM_{2.5}$. Naturally, the models predict best the BCP concentrations for the periods closest in time to the collection of air pollution and geographic information systems data. The success of interpolation of concentrations back in time depends on how much the emission landscape has changed during the previous years.

As can be seen in Table 5, various indicators of traffic always end up as predictors in the regression models for BCP. Indeed, land-use regression models typically work best for the traffic-originating particles, whereas the effect of, for example, point sources and residential wood combustion on air quality may not be fully taken into account owing to the lack of reliable indicators of emissions.

According to the available evidence, no studies have evaluated the ability of land-use regression-modelled outdoor BCP levels to predict annual BCP exposures, which is an obvious research gap. However, a number of studies have shown that traffic intensity near the home is an important determinant of long-term BCP exposure (for example, Raaschou-Nielsen et al., 2010; van Roosbroeck et al., 2006), and probably a more important determinant of BCP than of $PM_{2.5}$ (Fischer et al., 2000; Lanki et al., 2007). Thus, it is plausible that reasonably modelled outdoor BCP levels also predict long-term personal exposures in traffic-impacted communities.

Not too many studies were available to judge spatial correlations between long-term $PM_{2.5}$ and BCP (Table 6), but it seems that at least an occasionally high correlation will make the separation of the long-term health effects of $PM_{2.5}$ and BCP very difficult. It is not clear how much of the high correlation is due to the modelling itself (for example, a large number of predictors that are identical for BCP and $PM_{2.5}$), and consequently whether improved modelling, including finer resolution, would lower the correlation. Further, the correlation of BCP with NO_2

may be even higher, which implies that the long-term health effects of ultrafine particles, which are highly correlated with NO_2, are also hard to separate from the BCP effects. Estimation of the effects of long-term exposure to BCP on health is further complicated by the fact that traffic noise, another correlate, has been suggested as playing a role in the exacerbation of cardiovascular diseases (CVD).

Table 6. Correlations between long-term outdoor concentrations of BCP and $PM_{2.5}$

Study area	BC/ EC	R BCP-$PM_{2.5}$	R BCP-NO_2	R BCP-OC	Reference
Modelled with land-use regression models					
North Rhine-Westphalia, Germany	BC	0.52	0.93		Hochadel et al., 2006
Munich, Germany	BC	0.49	0.59		Morgenstern et al., 2007
Netherlands	BC	0.99	0.96		Brauer et al., 2002
Netherlands (only pregnancy time)	BC	0.75	0.84		Gehring et al., 2011
South-west British Columbia, Canada	BC	0.56			MacIntyre et al., 2011
Measured (representing large areas)					
California, United States	EC	0.84		0.51	Ostro et al., 2007
Southern California, United States	EC	0.91		0.82	Gauderman et al., 2004

Conclusions

Light Abs of $PM_{2.5}$ filter samples has been used in most European epidemiological studies as a measure of exposure to BCP, whereas studies in the United States have mainly used the EC content of the samples for this purpose. In some studies, Abs has been further converted into BS, which was widely used in the past in Europe for air quality monitoring. The conversion factor found in ISO standard 9835:1993 (ISO, 1993) is not, however, suitable for the present-day particulate air pollution mixture.

Vehicular traffic, especially diesel-powered, is a major source of BCP in urban areas. However, in some areas residential burning of wood or coal, and at least periodically open biomass-burning, may be even more important sources of BCP. More locally, harbours and industrial facilities may have a pronounced effect on BCP concentrations. Altogether, when interpreting effect estimates for BCP in epidemiological studies, information on the main sources of BCP in the area should be used.

Based on the studies reviewed, daily BCP concentrations measured at a central outdoor site well reflect daily personal exposures to BCP. The correlation between ambient levels and exposure seems to be slightly higher for BCP than for $PM_{2.5}$, possibly because of the higher infiltration rate of BCP. Measurement errors being comparable for $PM_{2.5}$ and BCP, exposure assessment is not likely to create significant differences between effect estimates for BCP and $PM_{2.5}$ in epidemiological studies on the acute health effects of air pollution.

Within-city variability is clearly greater for long-term outdoor BCP than for $PM_{2.5}$, which is a challenge for exposure assessment in epidemiological studies on the long-term effects of air pollution. A high proportion of spatial variation in ambient BCP can, however, be explained with the use of land-use regression models with carefully selected predictors. In the few available studies including both BCP and $PM_{2.5}$, the performance of the model has typically been at least as good for BCP as for $PM_{2.5}$.

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3. Effects of BC exposure observed in epidemiological studies[2]

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As in the Janssen et al. (2011) paper, this chapter systematically reviews the effects of the combustion particle metrics BS, BC, EC and Abs compared to PM mass for time-series studies of daily mortality and hospital admissions and cohort studies of mortality and morbidity. For acute health effects, only time-series studies on daily mortality and hospital admissions or emergency department visits were considered, as these are generally more similar in design and are therefore more likely to allow meta-analyses than studies of, for example, symptoms or biomarkers. For this chapter, panel (diary) studies of respiratory symptoms in symptomatic children were added, as these were also expected to provide sufficient comparable studies based on a recent systematic review and meta-analysis of the short-term effects of PM_{10} and NO_2 on respiratory health among children with asthma or asthma-like symptoms by Weinmayr et al. (2010).

Only studies that provided information on PM mass as well as combustion particle metrics were included. As described in the previous chapters of this report, the terms BS, EC, soot, BC, Abs (absorption coefficient) and light-absorbing carbon are used in different studies referring to different methods to measure or express concentrations of BCP. In this chapter, the abbreviation BCP is used as a generic term for any of the different metrics (BS, EC, BC or Abs) in general, but the study-specific terms are used when individual studies are described.

The different optical measurements for BCP (BS, BC and Abs) are highly correlated (Quincey 2007; Roorda-Knape et al., 1998). However, the quantitative relation between thermally determined EC and optical measures of BC varies between countries, cities and types of location (for example, regional, urban, traffic), highlighting the need for site-specific calibrations (Cyrys et al., 2003; Schaap & Denier van der Gon, 2007). Differences between EC measurement methods add to this variation. To facilitate comparisons among studies that used different measures of BCP, the BS to EC conversion factor was used, based on the average increase in EC associated with a 10 $\mu g/m^3$ increase in BS reported by 11 studies with information on both measures (Janssen et al., 2011). On the basis of this analysis, it is assumed by default that 10 $\mu g/m^3$ BS is equivalent to 1.1 $\mu g/m^3$ EC. In addition, sensitivity analyses were conducted using conversion factors over the range of the estimates from the individual studies (0.5–1.8 $\mu g/m^3$ EC per 10 $\mu g/m^3$ BS; see Annex 3A for details).

For the time-series studies, a meta-analysis was performed. Pooled fixed and random effects relative risk (RR) estimates were calculated for all health endpoints for which estimates from at least three different studies were available for the same age group and for different cities. Random effect estimates are reported as significant heterogeneity was observed ($P<0.05$) among individual estimates for some endpoints. In cases of no heterogeneity, fixed and random effect estimates are similar, so random effect estimates are reported for all endpoints for reasons of consistency. If estimates for multiple lags were reported, the estimate discussed by the author was used and indicated as "selected" lag in the Air Pollution Epidemiology Database maintained by St George's, University of London, and used to identify suitable studies for this analysis. If multiple risk estimates were available from the same city, only the most recent estimate was included, and if the

[2] This chapter is based on a paper by Janssen NAH et al., 2011.
[3] Additional contributions by Milena Simic-Lawson.

study area was part of a larger administrative area included in another paper (for example, the Netherlands rather than Amsterdam), only the results for the larger area were included. Finally, city-specific estimates for which PM_{10} was partly derived from BS were excluded.

For the panel studies, the methods used by Weinmayr et al. (2010) were followed for definition of asthmatic or symptomatic children as well as definition of the evaluated outcomes asthma symptoms and cough.

Summary fixed and random effects estimates were calculated using the *metan* procedure in Stata, as described by Harris et al. (2008). In order to calculate pooled estimates and compare estimated effects for BS and PM per mass unit, RRs for BS were converted to RRs for EC using the average conversion factor (10 $\mu g/m^3$ BS equals 1.1 $\mu g/m^3$ EC) or the range of conversion factors from individual studies (0.5–1.8) for sensitivity analysis.

Pooled effect estimates were expressed per 10 $\mu g/m^3$ (for BS and PM_{10}) or 1 $\mu g/m^3$ (for $PM_{2.5}$ and EC). To compare the effects based on comparable contrasts, the average ratio was calculated of the inter-quartile ranges (IQR) for PM mass/BCP and compared to the ratio of the RRs for BCP/PM mass. It was not possible to use study-specific IQRs to estimate pooled effects as IQRs were not available for all studies.

Results

Time-series studies of daily mortality and hospital admissions

Studies of BCP and PM_{10}

The majority of the papers concerned time-series studies of PM_{10} and BS (as a measure of BCP) conducted in Europe. Random effects estimates for the percentage change in each outcome with a 10 $\mu g/m^3$ increase in PM_{10} or BS are presented in Table 7. Information and effect estimates for all individual studies, and tests of heterogeneity and fixed effects estimates for studies included in meta-analyses, are reported separately for each outcome in Annex 3B (Tables 3B 1–10). Single-city estimates for the percentage change in all-cause mortality with a 10$\mu g/m^3$ increase in BCP and PM_{10} are also presented in Fig. 1. The available data were dominated by estimates from the Air Pollution and Health: a European Approach (APHEA) study (Katsouyanni et al., 2001; Analitis et al., 2006; Atkinson et al., 2001; Le Tertre et al., 2002).

For most outcomes, pooled effects estimates for a 10 $\mu g/m^3$ increase in exposure are greater for BS than PM_{10}, especially for mortality and hospital admissions for cardiovascular causes (Table 7). However, the average ratio of the IQRs for PM_{10}/BS (1.7, see Annex 3B, Tables 3B 1–10) was consistent with the ratios of RR for BS/PM_{10} (for example, 0.90/0.6=1.5 for cardiovascular mortality in Table 7), which suggests that effects estimates expressed for a similar increase in concentration (IQR) would be more or less equivalent. When a 10 to 1.1 conversion factor was used to transform the estimated effect of a 10-$\mu g/m^3$ increase in BS to the estimated effect for a 1 $\mu g/m^3$ increase in EC, the pooled random effect estimate for all-cause mortality changed from 0.68% (95% confidence interval (CI) 0.31–1.06) to 0.62% (that is, 0.68/1.1; CI 0.26–0.96). When study-specific conversion factors were used, the estimated effects for a 1$\mu g/m^3$ increase in EC ranged from 0.38% to 1.36% (for conversion factors of 1.8 and 0.5, respectively), which suggests that the effect of a 1 $\mu g/m^3$ increase in EC on all-cause mortality is at least eight times larger than the estimated effect of a 1 $\mu g/m^3$ increase in PM_{10} (0.05%).

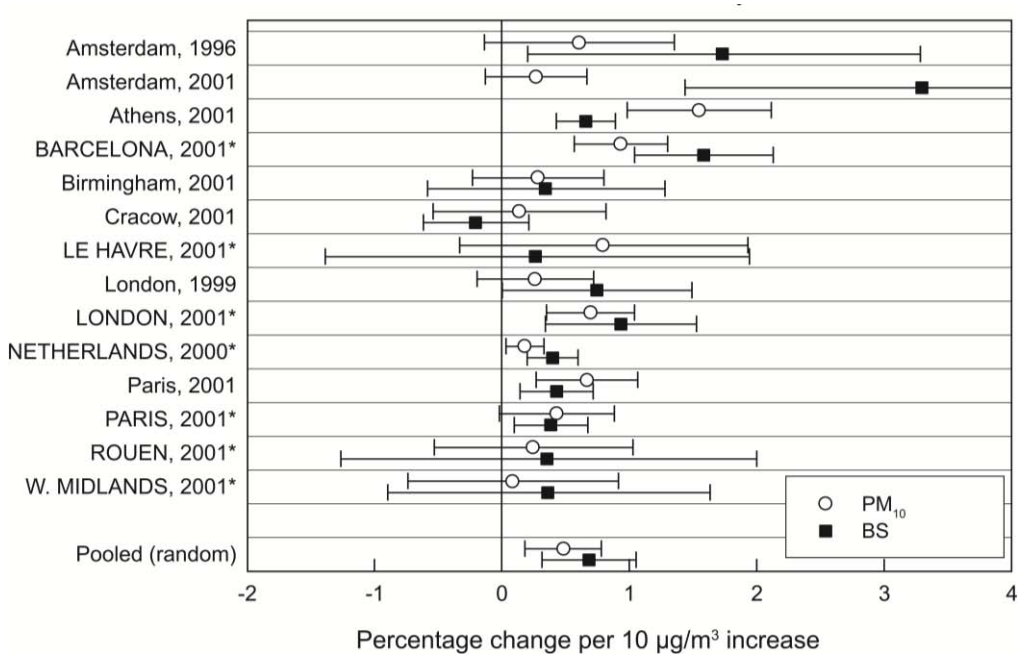
Table 7. Summary of comparison of pooled effects for PM_{10} and BS from time-series studies

Health outcomes	No. of estimates	Percentage change per 10 µg/m3 increase (95% CI)	
		PM_{10}	**BS**
Mortality			
All causes	7	0.48* (0.18–0.79)	0.68* (0.31–1.06)
CVD	7	0.60* (0.23–0.97)	0.90* (0.40–1.41)
Respiratory diseases	7	0.31 (-0.23–0.86)	0.95 (-0.31–2.22)
Hospital admissions			
All respiratory diseases, elderly people	6	0.70* (0.00–1.40)	-0.06 (-0.53–0.44)
Asthma + chronic obstructive pulmonary disease, elderly people	5	0.86* (0.03–1.70)	0.22 (-0.73–1.18)
Asthma, children	5	0.69 (-0.74–2.14)	1.64* (0.28–3.02)
Asthma, young adults	5	0.77 (-0.05–1.61)	0.52 (-0.51–1.55)
Cardiac, all ages	4	0.51* (0.04–0.98)	1.07* (0.27–1.89)
Cardiac, elderly people	4	0.67* (0.28–1.06)	1.32* (0.28–2.38)
Ischaemic heart disease, elderly people	5	0.68* (0.01–1.36)	1.13* (0.72–1.54)

* *P* <0.05.

Source: Janssen et al., 2011.

Fig. 1. Single-city, single-pollutant estimates for PM_{10} and BS and all-cause mortality



*Cities or areas included in the pooled estimate (year indicates year of publication).

Source: Janssen et al., 2011.

Studies of BCP and $PM_{2.5}$

Less, but more recent, information was available from studies in which both $PM_{2.5}$ and BCP were measured. Three studies provided estimates of $PM_{2.5}$ and EC, both for all-cause mortality and for cardiovascular mortality. Only two studies provided estimates for respiratory mortality (Klemm et al., 2004; Ostro et al., 2007; Cakmak, Dales & Blanco Vida, 2009; Mar et al., 2000) (see Annex 3C, Tables 3C 1–3). In pooled analyses, a 1 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 0.19% (0.03–0.35%) increase in all-cause mortality and a 0.29% (0.07–0.50%) increase in cardiovascular mortality. For EC, a 1 $\mu g/m^3$ increase was associated with a 1.45% (1.32–1.57%) increase in all-cause mortality and a 1.77% (1.08–3.08%) increase in cardiovascular mortality. Thus, expressed per mass unit, effect estimates are much larger (7–8 times) for EC than for $PM_{2.5}$. However, if the ratio of the IQR for $PM_{2.5}$/EC (~11) is taken into account, effect estimates were similar.

Information on the effect of PM and EC on hospital admissions or emergency department visits was even more limited than for mortality, and no pooled estimates could be calculated (Zanobetti & Schwartz, 2006; Ostro et al., 2009; Peng et al., 2009; Tolbert et al., 2007; Cakmak et al., 2009; see Annex 3D, Table 3D 1). When expressed per 1 $\mu g/m^3$ increase, effect estimates were generally 10–30 times higher for EC compared to $PM_{2.5}$. However, IQRs for EC were lower by a similar factor (for example, the ratio of the IQRs for $PM_{2.5}$/EC from Zanobetti & Schwartz (2006) (8.9/1.0) was similar to the ratio of the effect estimates for pneumonia with a 1 $\mu g/m^3$ increase in EC/$PM_{2.5}$ (0.054/0.0037), suggesting comparable effects with a comparable change in exposure.

Two-pollutant models of PM mass and BCP

In total, six papers included results of two-pollutant models that included a measure of PM mass as well as BCP. These included studies of mortality as well as hospital admissions and emergency department visits (Table 8). With one exception, effect estimates for BCP either increased or they decreased by ≤ 33% after adjustment for PM mass. In contrast, adjusting for BCP substantially reduced most effect estimates for PM mass (effect estimates became negative in three out of nine studies and decreased by >50% in five of the six other studies), suggesting that the effect of BCP is more robust than the effect of PM mass.

Studies of BCP and other PM components

In addition to the effects of BCP compared to PM mass, the relative health effects of BCP compared to other PM components are of interest. Specifically, it was interesting to evaluate whether the effects of BCP remained significant after adjustment for other potentially relevant components such as metals. Eight studies that reported effect estimates for EC and PM mass also reported estimates for PM components, including OC, sulfate and metals (Cakmak, Dales & Blanco Vida, 2009; Cakmak et al., 2009; Klemm et al., 2004; Mar et al., 2000; Ostro et al., 2007; Peng et al., 2009; Sarnat et al., 2008). In general, effects per IQR increase in exposure were greater for EC than for most of the six other frequently reported components (Annex 3E, Table 3E 1). For cardiovascular mortality and morbidity, four out of five studies reported significant associations with an IQR increase in OC, four out of four reported significant associations with potassium, and three out of four reported significant associations with zinc. Estimated effects of an IQR increase in EC on cardiovascular mortality and morbidity were significant in all five studies. For respiratory mortality and morbidity, the results were more diverse, with the strongest effects observed in two EC studies (Cakmak, Dales & Blanco Vida, 2009; Cakmak et al., 2009), with OC and/or sulfate in three studies (Ostro et al., 2009; Peng et al., 2009; Sarnat et al., 2008), and no significant (P<0.05) effects for any of the measured components in a sixth study (Ostro et al., 2007).

Table 8. Results from single- and two-pollutant models of time-series studies including PM_{10} or $PM_{2.5}$[a] and BCP (measured as BS in all studies shown)

Reference/ location	Health endpoint	R[b] PM-BS	Percentage change (per 10 µg/m^3)			
			PM single-pollutant	PM two-pollutant[c]	BS single-pollutant	BS two-pollutant[c]
Mortality						
Bremner et al., 1999, London, United Kingdom	Respiratory mortality	NA	1.3 (0.3–2.3)	0.4 (-1.0–1.8)	1.9 (0.2–3.7)	2.0 (-0.4–4.4)
	CVD mortality		0.6 (-0.1–1.2)	0.2 (-0.6–1.0)	1.2 (0.1–2.2)	0.8 (-0.6–2.2)
Hoek et al., 2000 Netherlands	Total mortality	0.77	0.3 (0.0–0.5)	0.1 (-0.3–0.6)	0.7 (0.4–0.9)	0.4 (-0.6–1.4)
	CVD mortality		0.2 (-0.2–0.5)	-0.6 (-1.3–0.1)	0.8 (0.4–1.2)	2.1 (0.5–3.7)
Admissions						
Anderson et al., 2001, West Midlands, United Kingdom	Respiratory admissions, all ages	0.64	0.6 (-0.5–1.7)	Considerably reduced[d]	1.1 (-0.1–2.2)	2.0 (0.3–2.8)
Atkinson et al., 1999a,[c] London, United Kingdom	Accident and emergency visits for asthma by children	NA	2.4 (0.7–4.1)	2.0 (-0.1–4.2)	2.8 (-0.0–5.7)	0.9 (-3.0–5.1)
Atkinson et al., 1999b, London, United Kingdom[e]	Cardiovascular admissions, >65 years	0.6–0.7	0.5 (-0.0–1.0)	-0.1 (-0.8–0.5)	1.9 (0.9–3.0)	2.3 (0.8–3.8)
Le Tertre et al., 2002, APHEA study	Cardiac admissions: cardiac disease, >65 years	0.5–0.8	0.5 (0.2–0.8)	-0.2 (-1.2–0.8)	1.1 (0.4–1.8)	1.6 (-0.3–3.5)
			0.7 (0.4–1.0)	0.1 (-0.4–0.7)	1.3 (0.4–2.2)	1.5 (0.3–2.7)
	ischaemic heart disease, >65 years		0.8 (0.3–1.2)	0.2 (-0.9–1.4)	1.1 (0.7–1.5)	0.8 (-1.1–2.7)

[a] $PM_{2.5}$ for Anderson, 2001; PM_{10} for all other studies.
[b] Coefficient of the correlation (R) between PM and BS.
[c] PM two-pollutant=PM from model with both PM and BS. BS two-pollutant=BS from model with both BS and PM.
[d] Quantitative information not available. Paper states that the effect of $PM_{2.5}$ was considerably reduced when BS was included in the model.
[e] Results only described qualitatively in the paper. Quantitative estimates provided by the authors on request.

Source: Janssen et al., 2011.

Three studies also reported effects estimates based on multi-pollutant models that included a variety of PM components (see Annex 3E, Table 3E 2). Two studies conducted in Santiago, Chile, reported significant associations with mortality (total, cardiac and respiratory) and hospital admissions (all non-accidental and respiratory) for EC, OC and 10–15 of 16 individual elements based on single-pollutant models, but effects estimates for only EC and OC remained significant after adjustment for all other pollutants measured (Cakmak, Dales & Blanco Vida, 2009; Cakmak et al., 2009). In a study on emergency department visits for cardiovascular and respiratory disease in 119 urban communities in the United States (Peng et al., 2009), seven major PM components were considered (sulfate, nitrate, silicon, EC, OC, sodium ion and ammonium ion). These seven components in aggregate constituted 83% of the total $PM_{2.5}$ mass, whereas all other components contributed <1% individually. In single-pollutant models, cardiovascular admissions were significantly associated with same-day concentrations of five out of seven major PM components, including EC. In multi-pollutant models with all seven components, only EC remained significant. For respiratory admissions, only same-day OC concentrations were significant, in both single- and multi-pollutant models. In a study of associations between hospital admissions for cardiovascular and respiratory disease in 106

counties in the United States that related admissions to the fraction of 20 elements to the total $PM_{2.5}$ mass rather than the concentration, RRs for cardiovascular and respiratory hospitalizations were highest in counties with a high $PM_{2.5}$ content of nickel, vanadium and EC (Bell et al., 2009). Here, nickel was the most robust component in multi-pollutant analyses, especially for cardiovascular admissions. Peng et al. (2009) reported statistically significant heterogeneity among effect estimates for different PM components, with the strongest estimated risk of cardiovascular admissions associated with EC concentrations. Cakmak et al. (2009) and Cakmak, Dales & Blanco Vida (2009) also reported that the 95% CI of the estimated effect of an IQR increase in EC did not overlap the 95% CIs of the other elements, with the exception of OC and 2–3 of the other 16 elements, indicating that the effect per IQR for EC was significantly greater than the estimated effects of most other single elements.

Panel studies of asthma symptoms and cough among children with asthma or asthma-like symptoms

The 9 papers on panel studies included 8 papers that provided single-city estimates and 1 paper that provided pooled effect estimates for the 28 panels from 14 countries in the Pollution Effects on Asthmatic Children in Europe (PEACE) study (Roemer et al., 1998). Table 9 summarizes effects estimates for the PEACE study and random pooled effects for the other eight studies. Information and effect estimates for all individual studies, and tests of heterogeneity and fixed effects estimates for studies included in meta-analyses, are reported separately for each outcome in Annex 3F (Tables 3F 1–4).

Table 9. Summary of comparison of pooled effects for PM_{10} and BS from panel studies among children with asthma or asthma-like symptoms

Study	No. of panels and children	Lag	Percentage change per 1 μg/m^3 increase (95% CI)	
			PM_{10}	BCP
Asthma				
PEACE study	28 panels; 2010 children[a]	Lag 0 Lag 1	-0.07 (-0.16 to 0.01) 0.00 (-0.10 to 0.10)	-0.66 (-1.42 to 0.11) -0.76 (-1.88 to 0.38)
Other studies	8 panels; 791 children[b]	Lag 0 Lag 1	0.27 (0.03 to 0.51)[c] 0.19 (-0.13 to 0.51)	4.27 (0.19 to 8.52)[c] 2.85 (-1.01 to 6.86)
Cough				
PEACE study	28 panels; 2010 children[a]	Lag 0 Lag 1	-0.04 (-0.08 to 0.02) -0.03 (-0.08 to -0.01)*	-0.37 (-0.80 to 0.06) 0.18 (-0.44 to 0.81)
Other studies	7 panels; 734 children[b]	Lag 0 Lag 1	0.03 (-0.02 to 0.09) 0.04 (-0.03 to 0.12)	1.33 (-0.28 to 2.96) 1.07 (-0.86 to 3.04)

* P <0.05.
[a] All children studied in the winter of 1993/1994.
[b] Children studied in different periods between 1990 and 2004.

Pooled effect estimates for the PEACE study were generally negative, and significantly negative (P <0.05) for cough at lag 1. Random pooled effect estimates for the other studies were all positive, but only significant (P <0.05) for asthma at lag 1 (both for PM_{10} and BCP). Random pooled effect estimates for all studies, including the PEACE as a single study, were also

generally positive. Effects estimates for BCP per μg/m^3 were an order of magnitude higher for BCP compared to PM_{10}, but none of the pooled estimates were significant (Annex 3F).

Significant pooled effect estimates for PM_{10} were found in the review by Weinmayr et al. (2010). Compared to the studies presented in Table 9, a total of 42 single-panel estimates for asthma were included. These included the 28 panel-specific estimates from the PEACE study as well as 16 single-panel estimates from other studies. The percentage change per 1 μg/m^3 increase from random pooled effect estimates for asthma at lag1 were 0.15% (95% CI, 0.04–0.26%) for all 42 panels and 0.25% (95% CI, 0.10–0.39%) after excluding the PEACE study (Weinmayr et al., 2010). The non-significant effects found in this analysis are, therefore, possibly caused by the lower number of studies and the relatively larger influence of the PEACE study in the selection of studies that provided effect estimates for PM mass as well as BCP.

Of the studies summarized in Table 9, only one provided information on two-pollutant models for PM mass and BCP (Delfino et al., 2003). In this study, both PM_{10} and EC were significantly associated with asthma symptoms in single-pollutant models. In a two-pollutant model that included both PM_{10} and EC, the OR for PM_{10} was reduced to 1.0 while the OR for EC remained stable (Delfino et al., 2003).

Cohort studies of long-term exposure to BCP and PM and mortality and morbidity

Cohort studies of mortality

Seven papers were identified that presented results from four different cohort studies, two of which included effect estimates for BS and PM and two for EC and PM (Table 10). Table 10 shows a recalculation of effects estimates for BS to effects estimates for EC on the assumption that EC=0.11 BS.

Table 10. RR for mortality related to long-term exposure to $PM_{2.5}$ and EC per 1 μg/m^3

Reference	Cohort	R PM-BCP	Cause	RR $PM_{2.5}$	RR EC
Smith et al., 2009	500 000 adults, aged 20–87 years, United States	NA	All causes	1.006 (1.002–1.010)	1.06 (1.01–1.11)
			Cardiopulmonary	1.012 (1.008–1.018)	1.11 (1.03–1.19)
Lipfert et al., 2006	70 000 male veterans, United States	0.54	All causes	1.006 (0.993–1.020)	1.18 (1.05–1.33)
Beelen et al., 2008[a]	120 852 adults; aged 55–69 years, Netherlands	>0.8[b]	Natural causes	1.006 (0.997–1.015)	1.05 (1.00–1.10)
			Respiratory	1.007 (0.972–1.043)	1.20 (0.99–1.45)
			Cardiovascular	1.004 (0.990–1.019)	1.04 (0.95–1.12)
			Lung cancer	1.006 (0.980–1.033)	1.03 (0.89–1.18)
			Other	1.008 (0.996–1.021)	1.04 (0.97–1.11)
Filleul et al., 2005[a,c]	14 284 adults, aged 25–59 years, France	0.87[d]	Natural causes	1.010 (1.004–1.016)	1.06 (1.03–1.09)
			Cardiopulmonary	1.012 (1.002–1.023)	1.05 (0.98–1.11)
			Lung cancer	1.000 (0.983–1.019)	1.03 (0.93–1.14)
Pooled effect (fixed)[e]			All causes	1.007 (1.004–1.009)	1.06 (1.04–1.09)

[a] RR for EC in European studies estimated from BS as EC 0.11 BS.
[b] For regional and urban component, NA for total.
[c] RR for $PM_{2.5}$ estimated from total suspended particles (TSP) as $PM_{2.5}$=0.5 × TSP.
[d] For all 24 sites, whereas RR presented for 18 sites (non-traffic).
[e] Pooled effect when using EC=0.18BS: 1.04 (1.02–1.06); when using EC=0.05BS: 1.10 (1.06–1.14).

When using the average conversion factor of 10 μg/m^3 BS=1.1 μg/m^3 EC, RRs for all-cause or natural cause mortality per 1 μg/m^3 EC in the two European studies and in the study by Smith et al. (2009) range from 1.05 to 1.06. RRs for EC and all-cause mortality in the veterans study were about three times larger than RRs for the same outcomes from the other studies, but as the standard error in the veterans study was two to four times higher compared to the other studies, this study contributes less to the pooled estimate (1.06[95% CI 1.04–1.09] per μg/m^3 EC). Pooled estimates for a 1 μg/m^3 increase in EC derived using high- and low-end conversion factors of 1.8 and 0.5 μg/m^3 per 10 μg/m^3 BS were 1.05 and 1.11, respectively. When expressed per 1 μg/m^3, the RR for EC is, therefore, 7–16 times higher than that for $PM_{2.5}$ mass (pooled estimate 1.007 per 1 μg/m^3). However, ratios of IQRs for $PM_{2.5}$/EC for the studies by Smith et al. (2009) and Beelen et al. (2008) were 14 and 9, respectively, and it was estimated that there was a ratio of about 5 based on graphical data presented for the study by Filleul et al. (2005). For the study by Lipfert et al. (2006), IQRs were not available but RRs expressed for the difference between the mean concentration and the minimum were 1.06 per 9.5 μg/m^3 for $PM_{2.5}$ and 1.09 per 0.5 μg/m^3 for EC. Hence, it appears that effects estimates for $PM_{2.5}$ and EC from cohort studies would also be similar if expressed for an IQR increase in exposure instead of a 1μg/m^3 exposure contrast.

Multi-pollutant modelling was applied in the studies by Lipfert et al. (2006) and Smith et al. (2009). Based on four-pollutant models that included EC, OC, sulfate and nitrate, Lipfert et al. (2006) concluded that EC had the greatest estimated impact on all-cause mortality, and that nitrate was the next most important constituent. In the analysis of data from the American Cancer Society study by Smith et al. (2009), the EC estimate for all-cause mortality was reduced by about 50% and lost statistical significance after adjustment for sulfate and/or ozone. For cardiopulmonary mortality, EC fell by about 33% and remained significantly associated after adjustment for sulfate, but fell by about 80% and lost significance after additional adjustment for ozone.

Cohort studies on morbidity

The eight papers on respiratory health outcomes in children included six papers describing results from one Dutch and two German birth cohorts, analysed using the same exposure assessment strategy, and two papers on lung function growth in two cohorts of children in southern California (Brauer et al., 2002; 2006; 2007; Clark et al., 2010; Gauderman et al., 2002; 2004; Gehring et al., 2002; 2010; Morgenstern et al., 2007; 2008; see Annex 3G, Tables 3G 1 and 2). For most of the studies, $PM_{2.5}$ and BCP were highly correlated (R>0.9). Overall, consistent with other types of study, estimated effects of a 1 μg/m^3 increase in BCP were greater than estimated effects of a 1 μg/m^3 increase in $PM_{2.5}$, whereas effects estimated for IQR increases were similar for BCP and $PM_{2.5}$.

Discussion

Single-pollutant effects estimates for daily mortality or hospital admissions generally were an order of magnitude higher for BCP compared to PM_{10} and $PM_{2.5}$ when expressed per μg/m^3. When differences in IQRs were accounted for, effects estimates were generally similar. It should be noted that there was a moderate to moderately high correlation between PM_{10} and BS measurements reported by the individual studies included in the pooled estimates (Pearson correlations of 0.5 to 0.8), consistent with correlations between daily wintertime PM_{10} and BS concentrations from a study in 14 European study areas (Hoek et al., 1997). Although this raises concerns about the ability to distinguish effects due to PM_{10} versus BS, there is at least some variation in the temporal patterns of these exposures.

In studies examining a variety of different PM components, BCP generally showed significant associations, especially with cardiovascular health endpoints, both before and after adjusting for other components. For cohort studies, pooled estimates for all-cause mortality per 1 $\mu g/m^3$ were 5 to 14 times higher for BCP compared to $PM_{2.5}$, but IQRs for $PM_{2.5}$ were higher than those for BCP by a similar factor.

The available evidence from two-pollutant models for time-series studies suggests that the effect of BCP is more robust than the effect of PM mass. Two-pollutant models with BCP and PM mass were not, however, conducted in any of the cohort studies. Although, overall, the results of multi-pollutant analysis including BCP, sulfate and ozone in the ACS study suggest that sulfate has the most robust association with all-cause and cardiopulmonary mortality, Smith et al. (2009) indicate that this can also be caused by differential amounts of measurement error. In the ACS study, where exposure was assessed at the metropolitan area level, estimates of the spatial distribution of EC probably contain more measurement errors than the assigned sulfate exposures as EC is more locally generated, as opposed to sulfate, which is a secondary pollutant with little spatial variation. When there are errors in measurement, variables measured with high precision will tend to dominate model-based predictions relative to variables measured with less precision (Smith et al., 2009). For time-series studies, there are no great differences in temporal relationships between central-site ambient concentrations and personal exposure for BCP and $PM_{2.5}$ (Janssen et al., 2005). In addition, issues related to the correlation between different pollutants and the extent to which they can act as surrogates for the etiological agent(s) complicate the interpretation of results from multi-pollutant models (Tolbert et al., 2007). This report's interpretation that the results from two-pollutant models for the time-series studies suggest that BCP is a more health-relevant indicator in these studies than PM mass is supported by Roemer & van Wijnen (2001; 2002), who calculated separate effects estimates with separate exposure estimates using background and traffic-influenced measurement stations for the total population and for people living along busy roads. Effects estimates for urban background BS were greater in the population living along busy roads than for the total population, suggesting that this subpopulation is more highly exposed. Indeed, effects estimates for the population living along busy roads using BS measured at traffic stations were more or less equivalent to effects estimates for the total population using BS measured at urban background stations.

It is also important that the spatial variation of BCP is much larger than that of PM mass (Hoek et al., 2002; Puustinen et al., 2007). This has in particular been demonstrated in relation to traffic. In a review of studies that simultaneously measured PM mass and BCP concentrations <50 m from busy roads and at background concentrations, Janssen et al. (2011) found that, on average, BCP concentrations near busy roads were twice as high as urban background BCP concentrations, whereas PM concentrations near busy roads were only about 20% higher than background. The one-time series study that explicitly took this phenomenon into account was conducted in Amsterdam (Roemer & van Wijnen, 2001; 2002) and showed that mortality effects were more closely associated with BS than with PM_{10}.

Based on the absolute differences in concentrations between street and background locations, it is estimated that, on average, 55% of the roadside increment in $PM_{2.5}$ was comprised of EC (Annex 3H). Given this relatively large proportion of carbon particles in the roadside increment of $PM_{2.5}$ mass, it can be expected that traffic abatement measures will result in greater reductions in BCP relative to reductions in PM mass. An illustration of an evaluation of the health benefits of a hypothetical traffic abatement policy measure using reported effect estimates for $PM_{2.5}$ mass and BCP, respectively, is included in Annex 3H. This calculation can be interpreted as an indication of the potential difference in a health impact assessment based upon $PM_{2.5}$ or BCP for

populations living along major roads. It can also be interpreted as the potential health gain for policies that reduce concentrations in approximately the same ratio as the current roadside increment, for example, a limitation of overall traffic intensity. This evaluation illustrates that the health effects of such policies may be seriously underestimated when based on effects estimates for $PM_{2.5}$ or PM_{10}. The implication of similar effects per IQR is that for policies that reduce all relevant components of PM proportionate to current levels, the estimated health benefits would be similar based on either indicator.

In the studies reviewed, ambient measurements of various BCP metrics were used. Although motorized traffic was an important source of BC in most of these studies, they included the impact of all combustion sources on BCP concentrations, including coal- and wood-burning, shipping emissions and industrial sources. In a review of source apportionment studies for fine particle EC, Schauer (2003) found that the combined contribution of diesel- and gasoline-powered vehicles ranged from 74% to 98%; the contribution from biomass-burning ranged from 0.7% to 25% and the contribution from other sources ranged from 0.5% to 17%. The derived risks therefore represent those for BCP as a general indicator of combustion particles, not exclusively traffic. Issues remain when these risk estimates are applied to specific combustion sources such as traffic or wood-burning. This report, however, holds that BCP more closely resembles the harmful components in these air pollution mixtures than general $PM_{2.5}$ does.

The systematic review was conducted for studies published up to January 2010 (Janssen et al., 2011). Although this review was not systematically updated with studies published after that date, the authors are aware of some recent studies that also provided effects estimates for PM mass as well as BCP. For cohort studies on mortality, Ostro et al. (2010) reported RRs for all-cause, cardiopulmonary and ischaemic heart disease mortality for a cohort of 45 000 women in California (United States). When expressed per 1 $\mu g/m^3$, the RR for all-cause mortality for the population living within 30 km of a monitor was 5 times higher for EC (RR 1.03) compared to $PM_{2.5}$ (RR 1.006). Effects per IQR were similar.[4] Gan et al. (2011) conducted a population-based cohort study on coronary heart disease mortality and hospitalizations for all 450 000 residents in metropolitan Vancouver (Canada). When expressed per 1 $\mu g/m^3$, the sex, age, co-morbidity and socioeconomic status-adjusted RR for coronary heart disease mortality was 10 times higher for BC (RR 1.06) compared to $PM_{2.5}$ (RR 1.006). The effects per IQR were 6 times higher (RR 1.06 for BC and 1.01 for $PM_{2.5}$). In three-pollutant models, including BC, $PM_{2.5}$ and NO_2, the effect for BC was unaffected (RR 1.06; 95% CI, 1.03–1.09), whereas the RR for $PM_{2.5}$ was reduced to 1.00. In two recent birth cohort studies on respiratory health in children, both PM and BCP were significantly associated with adult onset-incident asthma, with similar effects per IQR and higher effects for BCP per 1 $\mu g/m^3$ (Gehring et al., 2010; Clark et al., 2010). In a third recent birth cohort study, $PM_{2.5}$ was significantly associated with incident asthma, whereas BC was not (Carlsten et al., 2011). In two recent panel studies, neither $PM_{2.5}$ nor EC were significantly associated with wheeze at lag 0 or lag 1 in one study (Mann et al., 2010), whereas only BC (and not $PM_{2.5}$) was significantly associated with wheeze at lag 0 and lag 1 in the second study (Patel et al., 2010). Overall, these more recent studies do not change the conclusions based on the studies published up to January 2010.

Is it now possible to answer the question as to whether the effects of BCP are different from effects of $PM_{2.5}$ mass or other components in airborne PM? In general, the appropriate comparison would be the one based on IQR effect estimates, and those do not provide unequivocal support for BCP being more important than the remainder of the PM mass.

[4] Effects estimates reported in the original paper were recently adjusted in an erratum.

However, for assessments of exposure conditions dominated by combustion sources, or policies directed towards specific combustion sources, the comparison of RRs expressed per one unit change in mass is relevant. Additional evidence comes from the quoted two- and multi-pollutant studies which predominantly show that BCP effects estimates are robust to adjustment for PM mass, whereas PM mass effects estimates decrease considerably after adjustment for BCP. Limited evidence from the one study comparing effects estimates based on traffic-exposed and urban background monitoring stations also supports this view. For the cohort studies, the database is small and, especially for the birth cohort studies, characterized by often very high spatial correlations between PM mass and measures of BCP. Multi-pollutant models with BCP and PM mass were only conducted in one recent cohort study on coronary heart disease mortality (Gan et al., 2011); this showed that the significant effect observed with BC was robust for adjustment for $PM_{2.5}$, whereas no significant effect of $PM_{2.5}$ on coronary heart disease mortality was observed.

Collectively, these studies show that BCP is associated with health effects that are not reflected quantitatively in the same way by the PM mass concentration. Based on the considerations mentioned, the conclusion is that BCP represents one of the more health-relevant components of PM, especially for cardiovascular effects. BCP could, therefore, be a valuable additional air quality indicator to evaluate the health risks of air quality dominated by primary combustion particles.

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4. Evidence from toxicology, including human clinical studies

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Introduction

BC or any other alternative (optical) methods such as reflection on filters are seldom assessed (or reported) in toxicity studies on ambient air, either in controlled human exposure studies or in studies using experimental animals or in vitro systems. For this reason, this chapter will also consider exposure to carbon-rich test atmospheres such as (diluted) combustion-derived PM as the next best measure to compare with measures in the air quality area and epidemiology. There has been broad discrimination between combustion of fossil fuels in engines and combustion of biofuels such as wood in residential heaters. In addition, studies in which EC has been used were selected for this review.

Reflectance (a metric for BC) does not solely result from combustion-derived aerosols. Wind-blown dust, iron oxide from railways and non-tailpipe emissions such as brake or tyre wear from vehicles can also contribute to BC. These sources have not, however, been considered for the assessment described below, largely because of a lack of relevant information.

This review focused on the following two points.

- What are the effects of BC in the human controlled exposure experiments? Are they differently qualitatively (for example, different health outcomes) and/or quantitatively from the effects of $PM_{2.5}$ mass concentration or other measured components?
- What are the mechanisms of the BC effects indicated by toxicological studies?

A literature search (Annex 1) focused on human clinical studies in which both PM and some measure for BC were recorded, or inhalation studies that have used diluted diesel engine exhaust, woodsmoke or EC. Studies assessing the hazard of coal dust (not relevant for the general population and PM largely >2.5 um in aerodynamic size) and other non-combustion PM sources (for example, brake wear, or rail or tyre dust) that may contribute to the blackness of ambient PM material were excluded.

This selection resulted in seven studies, in which both BC and PM mass were measured (Table 11). From these, no single study provided information on the concentration–dose–response relationships. This hampers comparison of PM mass with some measure of BC. In addition, all studies were limited by the duration of exposure (only a few hours) and no supporting evidence for longer exposure duration can be provided. Subsequently, biological responses related to the inhalation exposure were summarized and an assessment was made to establish if the effects were more strongly related to BC than to PM mass. In addition, a search was performed to identify specific mechanisms and biological pathways for BC leading to toxicity and adverse health effects as identified in epidemiological studies.

Table 11. Summary of clinical findings in human subjects exposed to ambient PM, (diluted) diesel engine exhaust, pure carbon particles in air, or combustion particles of wood and other biomass

Subjects	Exposure	Outcome	Main findings	Reference
Ambient PM				
Healthy (n=12) and asthmatic (12) subjects	2-hour exposure with intermittent exercise to on average 174 µg/m^3 $PM_{0.1-2.5}$, 10% EC.	*Respiratory function and blood.* Spirometry and routine haematological measurements. Columnar cells in post-exposure-induced sputum, mediators of blood coagulation, systemic inflammation, heart rate variability, systolic blood pressure.	Cardiovascular (but not respiratory) symptoms increased slightly in both groups.	Gong et al., 2003
Age-matched volunteers (n=12), middle-aged healthy volunteers or patients with coronary heart disease	2-hour exposure with intermittent exercise to on average 190 µg/m^3 $PM_{2.5}$, low or no EC.	*Vascular and blood parameters, respiratory inflammation.*	No effect on vasomotor or fibrinolytic function; slight increase in local airway inflammation.	Mills et al., 2008
Diesel engine exhaust (DE)				
Healthy non-smoking male volunteers (n=18), mean age 27 years	1-hour exposure with intermittent moderate exercise to 250 µg/m^3 DE, European transient cycle engine running conditions.	*Blood and arterial function*. Vascular vasomotor, fibrinolytic function 6 hours after exposure.	Impaired vascular function: attenuated vasodilatation, reduced capacity to remove blood clots (tissue plasminogen activator reduction). Impaired calcium channel-dependent vasomotor function under European transient cycle condition.	Barath et al., 2010
Healthy non-smokers aged 21–44 years	2-hour exposures to DE 350 µg/m^3 from two different engines and fuels (one in Edinburgh (United Kingdom) and the other in Umeå (Sweden)), idling engine, intermittent moderate exercise.	*Blood and thrombosis*. Ex vivo thrombus formation (Badimon), in vivo platelet activation (flow cytometry), haematology, coagulation and inflammation markers at 2 and 6 hours after exposure.	Increased thrombogenicity at 2 and 6 hours, when using two different diesel engines and fuels. Increased platelet-neutrophil (52%) and platelet-monocyte (30%) aggregates.	Lucking et al., 2008
Healthy non-smoking males aged 20–38 years	1-hour exposure to DE 300 µg/m^3, idling engine, intermittent moderate exercise.	*Vascular and blood*. Forearm blood flow by venous occlusion plethysmography, tPA, PAI-1, haematology at 2 and 6 hours post-exposure, IL-6, TNF-α, ET-1, CRP.	No differences in resting forearm blood flow or in inflammation markers. Impairment of vascular tone and endogenous fibrinolysis.	Mills et al., 2005
Healthy (n=4) and stable asthmatics (n=7)	2-hour exposure to DE 100 µg/m^3, idling diesel truck.	*Respiratory function*. Lung function (FEV_1).	No changes in lung function.	Sawant et al., 2008
Male/female healthy subjects (n=15)	1-hour exposure to DE 300 µg/m^3, European transient cycle engine running conditions, moderate intermittent exercise.	*Lung*. Airway inflammation, lung function, bronchoscopy (endobronchial mucosal biopsy) and airway lavage at 6 hours post-exposure.	Increased bronchial adhesion molecule expression. Bronchoalveolar eosinophilia, which has not been shown after exposure to DE at idling (Stenfors et al., 2004).	Sehlstedt et al., 2010a

Subjects	Exposure	Outcome	Main findings	Reference
Pure EC				
Healthy subjects	Freshly generated EC particles (mean diameter 25 nm, geometric standard deviation 1.6 nm). Exposure for 2 hours: 10 µg/m^3 at rest; 10 and 25 µg/m^3 with exercise; 50 µg/m^3 with exercise.	*Blood*. Collection of peripheral venous blood. Flow cytometry to quantify leukocyte expression of surface markers.	Increased retention of leukocytes in pulmonary vascular bed as shown by altered peripheral blood leukocyte distribution and expression of adhesion molecules.	Frampton et al., 2006
Subjects with asthma	2-hour exposure to 10 µg/m^3 with exercise.			
Healthy subjects and patients with stable angina (20/group)	1-hour exposure to carbon particles 50 µg/m^3.	*Cardiovascular and blood*. Heart rate variability, inflammation and coagulation markers.	No effects on heart rate variability or systemic inflammation.	Routledge et al., 2006
Male/female healthy subjects (n=16) aged 18–40 years	2-hour exposure to carbon ultrafine particles (UFP) 50 µg/m^3, intermittent exercise.	*Vascular and blood*. Vital signs, venous occlusion plethysmography, hypaeremia of the forearm, venous plasma nitrate and nitrite levels.	Impaired peak forearm blood flow during reactive hypaeremia.	Shah et al., 2008
Subjects with type 2 diabetes (n=16)	2-hour exposure at rest to 50 µg/m^3 of elemental carbon UFP, median diameter 32 nm.	*Vascular and blood*. Vascular activation, coagulation, systemic inflammation at 0.5, 3.5, 21 or 45 hours post-exposure.	Platelet activation with possible associated activation of blood leukocytes and vascular endothelium.	Stewart et al., 2010
Male/female healthy subjects aged 18–40 years	2-hour exposure to elemental carbon UFP 10 µg/m^3 at rest.	*Cardiovascular*. Electrocardiogram, parameters of heart rate variability, repolarization duration, morphology and variability, changes in the ST segment (distance from segments S to T of the electrocardiogram).	No marked changes in electrocardiogram-derived parameters.	Zareba et al., 2009
Two separate studies, n=12 in each	2-hour exposure to elemental carbon UFP 10 and 25 µg/m^3 with repeated exercise.			
Combustion of wood and other biomass				
Healthy individuals, subjects with mild to moderate asthma, subjects with allergic rhinitis (15 persons in each group; a total of 18 men and 27 women aged 22–54 years)	Rice straw smoke (RSS) aerosol rich in inorganic and organic carbonaceous material by purpose-built, controlled burner to simulate open burning. Of all PM, 85% smaller than 4.2 µm in diameter. Four 30-minute exposure patterns in environmental chamber for each subject group: single filtered air, single RSS-PM_{10} ~200 µg/m^3, single RSS-PM_{10} ~600 µg/m^3, and RSS-PM_{10} ~200 µg/m^3 repeated on three successive days.	*Spirometric lung functions*. Pre-exposure, and immediately and 6 hours after controlled exposure. *Bronchial washing (BW) and bronchoalveolar lavage*. 6 hours after controlled exposure.	Airway inflammation (increased neutrophils in BW and macrophages or lymphocytes in bronchoalveolar lavage fluid) in healthy individuals and subjects with asthma by RSS-PM_{10} 600 µg/m^3 and 3 × RSS-PM_{10} 200 µg/m^3. Asthmatics had higher neutrophil concentration and percentage in bronchoalveolar lavage fluid compared to healthy individuals and subjects with allergic rhinitis. In healthy individuals only, increased IL-8 in BW by 3 × RSS-PM_{10} 200 µg/m^3. No changes in lung functions by any exposure pattern in any subject group. Several indices of inflammation, including increases in neutrophils, epithelial cells and IL-8 in bronchoalveolar	Solomon et al., 2003

Subjects	Exposure	Outcome	Main findings	Reference
			lavage fluid of all subject groups indicated that 3 × RSS-PM_{10} 200 µg/m^3 caused a more powerful inflammatory stimulus to the respiratory tract than the single exposure to RSS-PM_{10} 600 µg/m^3.	
Healthy subjects (6 men and 7 women, aged 20–56 years)	Woodsmoke aerosol in a 130 m^3 environmental chamber generated by logwood heating of a small cast-iron wood stove. Two 4-hour exposure patterns: clean air or woodsmoke aerosol in two similar sessions of 6 and 7 subjects, two 25-minute periods of light exercise. Median $PM_{2.5}$ 279 µg/m^3 and 243 µg/m^3 in the two sessions (clean air 13 and 11 µg/m^3). Median total PM number concentration 180000#/cm^3 and 95000#/cm^3 (clean air 4400#/cm^3 and 7500#/cm^3). Geometric mean diameters of PM 42 nm and 117 nm, standard deviations 1.7 nm and 1.4 nm. Median BS 72 and 91 × 10^{-5}/m (clean air 1.6 × 10^{-5}/m). Benzo[*a*]pyrene 19 and 21 ng/m^3 (no data for clean air).	*Blood samples*. Collection of peripheral venous blood immediately before, and 3 and 20 hours after controlled exposure. *Exhaled breath measurements/samples*. Immediately before, and 3 and 20 hours after controlled exposure. *Urine samples*. Immediately before and 20 hours after controlled exposure.	Increased serum amyloid A (cardiovascular risk factor), increased tendency towards blood coagulation (factor VIII in plasma and factor VIII/von Willebrand factor ratio) and temporary increase in free radical-mediated lipid peroxidation (urinary excretion of free 8-iso-prostaglandin2α).	Barregård et al., 2006
			Inflammatory response and signs of increased oxidative stress in the alveolar region of the respiratory tract (exhaled nitric oxide, malondialdehyde in breath condensate, serum Clara cell protein).	Barregård et al., 2008
			DNA strand breaks decreased and significant up-regulation of the repair gene hOGG1 in human peripheral blood mononuclear cells 20 hours after exposure, but no evidence of direct genotoxic effects (timing of sampling, enhanced repair).	Danielsen et al., 2008
Healthy subjects (10 men and 9 women, aged 21–31 years)	Woodsmoke aerosol in an 18 m^3 environmental chamber generated by 15kW residential pellet burner connected to a boiler. Two 3-hour exposure patterns: filtered air or woodsmoke aerosol from low-temperature incomplete combustion; alternating 15-minute periods of light exercise and rest. Mean $PM_{2.5}$ 224 µg/m^3 (no data for clean air). Mean total PM number concentration 67000#/cm^3 (filtered air 1–2#/cm^3).	*Spirometric lung functions*. Pre-exposure and immediately, 4 and 24 hours after controlled exposure. *Symptom recording*. Pre-exposure, every 30 minutes during exposure and immediately after controlled exposure. *Exhaled air nitric oxide*. Immediately before and 3 and 20 hours after controlled exposure. *Bronchial washing (BW) bronchoalveolar lavage and endobronchial biopsies*. 24 hours after controlled exposure.	No significant changes in lung functions or exhaled air nitric oxide. Mild but progressive irritation in the nose and throat and unpleasant smell during the woodsmoke exposure (peaking between 1.5 and 2.5 hours from the beginning of exposure), but a subset (7–10 of the 19 subjects) experienced no symptoms. No significant changes in the differential cell counts in BW and bronchoalveolar lavage fluid. Antioxidant response (increased glutathione) in bronchoalveolar lavage fluid at 24 hours after woodsmoke exposure. No changes in low molecular weight antioxidant concen-	Sehlstedt et al. 2010b

Subjects	Exposure	Outcome	Main findings	Reference
	Geometric mean diameter of PM 120 nm (two modes with peaks at 100 nm and 190 nm). Chemical composition of diluted woodsmoke: PM: 60% OC, 25% EC, 13% alkali salts and 2% trace elements. Total polycyclic aromatic hydrocarbons (PAH) 760 ng/m^3 (12–26% in PM fraction).		trations in the endobronchial mucosal biopsy samples.	
Atopic subjects (10 men and 10 women, aged 19–55 years)	Woodsmoke aerosol in a 79 m^3 environmental chamber generated by logwood heating in a small cast-iron wood stove or filtered air. PM diameters in chamber aerosol not measured. Three patterns of 3.5-hour exposures: filtered air, single low exposure aiming at $PM_{2.5}$ ~200 µg/m^3 and single high exposure aiming at $PM_{2.5}$ ~400 µg/m^3. Mean $PM_{2.5}$ during low exposure 221 µg/m^3 and high exposure 354 µg/m^3 (filtered air 14 µg/m^3). Median total PM number concentration ~29000#/cm^3 and ~71000#/cm^3 (clean air 222#/cm^3). Benzo[*a*]pyrene 329 and 325 ng/m^3 (clean air 0.2 ng/m^3).	*Recording of symptoms*. Self-evaluation of 29 symptoms or perceptions with standard visual analogue scales before and every 30 minutes during the exposure.	Weak significant effects for categorized symptom indices: environmental perception, irritative body perceptions, psychological/neurological effects and weak inflammatory responses. Significant increase in self-reported general mucosal irritation. Nearly significant difference in psychological/neurological effects between high and low exposure.	Riddervold et al., 2011

Adverse health effects of BC in the controlled human exposure experiments

The vast majority of controlled human exposure studies set up to identify adverse responses took diesel engines as a source for BC emissions. These studies were limited in the duration of exposure, which did not usually last much longer than a single two-hour period. The studies used PM exposure concentrations in the range 100–350 µg/m^3. Effects related with carbonaceous emissions included oxidative stress, inflammation, lipid peroxidation and atherosclerosis, change in heart rate variability, arrhythmias, ST-segment depression (heart function), and changes in vascular function (such as blood pressure) (Grahame & Schlesinger, 2010).

For this review, studies were selected that evaluated PM mixtures relevant for the questions, mainly diesel engine exhaust, woodsmoke and concentrated ambient $PM_{2.5}$. Controlled exposure

studies compared health responses to the selected exposure with those to filtered air in order to adjust for potential effects of the experimental setup. Usually, each subject served as his/her own control. Typically, the evaluated PM exposures (100–350 μg/m^3) were higher than those encountered in ambient air, although not excessively so. Because of ethical concerns, healthy subjects or subjects with mild disease were commonly selected for the controlled exposure studies that focused only on mild, reversible adverse effects.

Ambient PM

The importance of the composition of particles is illustrated by the study of Mills et al. (2008), which saw little effect in a two-hour exposure to high $PM_{2.5}$ concentrations taken in Edinburgh (Table 11). This was attributed to the high sea salt content (90%) providing evidence that mass per se is not the best predictor for cardiovascular symptoms. It should be noted that the BC : PM mass ratio was rather low in this study. Gong et al. (2003) exposed healthy and asthmatic subjects to increased levels of fine PM in Los Angeles (United States) and presented a detailed chemical specification. Relatively high levels of BC were reported (~75 μg BC/mg PM). Both respiratory and cardiovascular effects were examined (Table 11) and only a slight increase in cardiovascular symptoms (parasympathetic stimulation of heart rate variability, blood coagulation and systemic inflammation) were observed for both groups. A higher EC concentration predicted a more positive change in the composite index of ST voltage (ST-AMD) post-exposure and a more negative change two days after exposure. On the other hand, total mass was related to the low frequency/high frequency power ratio (Gong et al., 2003). Other than this, there are no data to support the idea that EC/BC is specifically associated with subtle short-term health effects. More controlled volunteer studies have attempted to link health effects with several PM components, but the problem is that it will require a lot more subjects exposed than the 20 or so that have been included in most human controlled exposure studies. So far no component, including BC, has been successfully linked to effects from a concentrated $PM_{2.5}$ study, with the exception of a link of soluble sulfate and metals with increased bronchiolar lavaged neutrophils or increased blood fibrinogen (Huang et al., 2003). The designs of most controlled studies have not, for the most part, been suitable to test the BC-related health effects, and in any case, the statistical power (due to a lack of BC exposure contrasts between or within subjects) would be too low to give strong evidence.

Diesel engine exhaust

Several studies have documented *airway and systemic inflammation* following exposure to diesel engine exhaust, although a measure of BC was only taken in the study of Sehlstedt et al. (2010a). No changes in *respiratory function* were observed after a two-hour exposure to diesel engine exhaust in both healthy subjects and stable asthmatics (Sawant et al., 2008). Most studies that included PM mass and a measure for BC examined changes in the *cardiovascular system*. Barath et al. (2010) compared the cardiovascular effects of exhaust from an idling diesel engine with that of an engine running according to an urban transient cycle. Similar results were reported for both protocols. Even though the PM mass concentrations were not that different (255 versus 220 μg/m^3 for urban transient and idling, respectively), there was a 16-fold difference in the EC content, with the transient cycle giving the highest EC levels (88 μg/m^3). The opposite contrast was measured for OC. Unfortunately, none of these studies provided concentration–dose–response relationships, which militates against a full comparison of the predictive value of both PM mass and EC or OC concentration in the diesel engine exhaust studies. Different operating conditions, such as transient cycle and idling (as mentioned in the study by Barath et al., 2010), will result in variations in the EC : PM and EC : OC ratios, which allows comparison of the effects for PM and EC. At present, however, insufficient data have been published to allow a

thorough analysis of the role of EC in these complex mixtures, despite the fact that there has been much carbonaceous material in the test atmospheres.

All diesel engine exhaust studies included the gaseous fraction of the engine exhausts, and gases and vapours are also known to contribute to the total health effect. Yet the findings of Lucking et al. (2011) provided evidence that removing the particulates with a modern diesel particle trap (and incidentally increasing the NO_2 levels) resulted in a complete absence of the cardiovascular effects seen for the whole mixture. Similar findings were seen in a study in which the particulates were removed by filtration (Mills et al., 2011). Interestingly, these authors also exposed the volunteers to pure EC at even higher particle number counts (although slightly lower mass concentrations) than that measured in the engine exhausts. Adverse responses were only observed for whole diesel engine exhaust (increased arterial stiffness and lowered capacity to reduce blood clots) but not for EC-free exhaust or EC-only atmospheres, suggesting that EC acts as a carrier of harmful constituents to the target tissue.

The effects of diluted diesel engine exhaust on other targets, such as the brain, have also recently been described. Crüts et al. (2008a) demonstrated an immediate impact on brain activity during and shortly after a one-hour exposure to 300 $\mu g/m^3$ of diesel engine exhaust, suggesting a stress response. No BC measure was included in this study, but in a follow-up study using artificially generated carbon nanoparticles, similar responses could not be demonstrated (unpublished findings). The clinical relevance of the brain activity measured by quantitative electroencephalography is, however, questionable. No other controlled human exposure studies are known in which effects on the central nervous system have been studied.

Pure EC

Most studies that focused on the black fraction of PM were carried out at Rochester University (United States). Frampton et al. (2006) reported that inhalation of carbon particles (10–50 $\mu g/m^3$) did not result two hours after exposure in lung inflammation or in an acute phase response in the blood. The observed subtle changes in leukocyte subsets and adhesion molecule expression are, however, consistent with effects on vascular endothelial function. In a subsequent study, Frampton and colleagues exposed subjects with type 2 diabetes to 50 $\mu g/m^3$ of ultrafine EC PM (count median diameter, 32 nm) for two hours at rest, and reported slightly increased activated platelets in the blood (Stewart at al., 2010).

Routledge et al. (2006) were not able to detect any adverse effects on the cardiovascular system after a one-hour exposure to 50 $\mu g/m^3$ of EC. This was confirmed by Mills et al. (2011). From both studies, it was concluded that the adverse effects of vehicle-derived particulates are likely to be caused by more reactive species found on the particle surface rather than BC itself. Indeed, Biswas et al. (2009) demonstrated that the organic fraction absorbed on the surface of BC contributed to the most part of the redox activity of the PM, suggesting that this also represents a fraction of tailpipe exhaust that can cause acute toxicity.

In summary, acute exposure to carbon particles at levels higher than those found in ambient air do not result in clinically relevant adverse effects. The representativeness of these particles for combustion-derived BC is, however, not known.

Biomass, including wood

Many fewer controlled exposure studies in humans have been conducted on emission aerosols from residential wood combustion devices than on diesel engine exhausts (Table 11). The

commonly used $PM_{2.5}$ or PM_{10} concentrations (200–400 μg/m^3) in these studies correspond to the highest ambient air peak concentrations measured during wintertime temperature inversions in older residential areas of western countries where wood is prevalent as the primary domestic heating fuel. In developing countries, biomass combustion may increase ambient $PM_{2.5}$ or PM_{10} concentrations to exceed 1 mg/m^3 and, without a chimney, the indoor peak concentrations can be even higher. Unfortunately, it is impossible to assess the role of BC in the measured responses, because BS has been measured in one exposure set-up (Sällsten et al., 2006), and EC and OC in only one other study (Sehlstedt et al., 2010b); both of these studies had only a single exposure level in healthy subjects. Only one peer-reviewed journal paper (Riddervold et al., 2011) and one research report on the internet (Solomon et al., 2003) provide data on $PM_{2.5}$ or PM_{10} at more than one exposure level. The overall interpretation of the results from these few studies is also hampered by their highly varying protocols in both exposure duration and response recording.

Sällsten et al. (2006) and Barregård et al. (2006; 2008) from the University of Gothenburg (Sweden) used a conventional stove to generate woodsmoke. Blood and urine measurements after four-hour exposures in healthy volunteers suggested that woodsmoke was associated with systemic inflammation and an increased tendency towards blood coagulation and lipid peroxidation (Barregard et al., 2006). In addition, exposure to woodsmoke increased markers of inflammation in distal airways (Barregard et al., 2008). The exposure was also followed by significant up-regulation of the repair gene hOGG1 in human peripheral blood mononuclear cells, but no direct genotoxic effects were observed, possibly due to the timing of sampling or enhanced cellular repair (Danielsen et al., 2008).

Sehlstedt et al. (2010b) from the University of Umeå (Sweden) exposed healthy volunteers for three hours to woodsmoke aerosol from incomplete combustion dominated by carbonaceous PM composition. They found that the antioxidant glutathione concentrations were significantly increased in bronchoalveolar lavage fluid at 24 hours after exposure to woodsmoke, together with an increase in upper airway symptoms. Lung function, exhaled NO concentration or parameters of systemic or airway inflammation were not significantly altered.

Riddervold et al. (2011) from Aarhus University (Denmark) conducted two levels of three-hour exposure to woodsmoke aerosol ($PM_{2.5}$ 220 μg/m^3 and 354 μg/m^3) among non-smoking atopic human participants with normal lung function and normal bronchial reactivity. Statistically significant increases in the subjectively rated mucosal irritation were observed by woodsmoke aerosol without any major difference between the two exposure levels.

Solomon et al. (2003) from the University of California (United States) have published results in a public research report on controlled exposures among three different subjects groups to rice straw combustion aerosol adjusted to share features with poor combustion in agricultural open fires. The aerosol was physico-chemically somewhat different from conventional wood combustion aerosols; for example, about 25% of the emitted and diluted PM were larger than 1 μm in diameter (up to 30 μm), while in wood combustion practically all PM material is smaller than 1 μm in diameter. Even though the rice straw combustion aerosol was dominated by carbonaceous material, the types of organic may have differed from those of wood combustion aerosol. The results indicated that airway inflammation, as measured by neutrophils, macrophages, lymphocytes and IL-8 measured in bronchoalveolar lavage fluid at 6 hours after a 30-minute exposure, increased as a function of smoke concentration and of the total dose of smoke aerosol in all subject groups. However, none of the exposures caused significant changes in spirometric pulmonary functions measured during the exposures. Asthmatic subjects exhibited greater overall inflammatory responses than healthy individuals and subjects with allergic rhinitis. The authors suggested that the anti-inflammatory

mechanisms involved in controlling the inflammatory response to single-smoke exposure were probably exceeded in responses to the serial-day exposures.

Mechanisms of toxicity

There were only five experimental studies on urban air fine PM (three in macrophages, one in mice, two using a cell-free assay of oxidative potential) that fulfilled the criteria for selection of original science articles: animal, cell or other in vitro studies on urban air fine PM with in-depth chemical characterization of the investigated PM samples (EC, OC/particulate organic matter (POM), major inorganic ions, transition metals and insoluble soil mineral components). Three European studies from the Chemical and Biological Characterisation of Ambient Air Coarse, Fine and Ultra Fine Particles for Human Health Risk Assessment in Europe (PAMCHAR) project (Happo et al., 2008; Jalava et al., 2008; 2009) have systematically compared the toxicological associations of EC with relevant organic constituents (PAHs, dicarboxylic acids, endotoxin) and inorganic constituents (ions, water-soluble and water-insoluble elements) of the same urban air fine PM samples collected from six different source environments characterized with chemical markers. EC was not significantly associated with any toxicological responses, while dicarboxylic acids (for example, oxalate, malonate and succinate, which are atmospherically transformed organics from semi-volatiles of combustion sources) and transition metals from heavy oil combustion (vanadium, nickel) and soil-derived mineral compositions were positively associated with the inflammatory activity of fine PM. PAHs from residential biomass and coal combustion were positively associated with cytotoxicity in macrophages and immunotoxicity in the mouse lungs. The findings in these in vivo and in vitro studies agreed well with each other. Moreover, they agree very well with the findings of Hu et al. (2008), who showed that the redox activity in rat alveolar macrophages and in cell-free dithiothreitol assay by quasi-ultrafine $PM_{0.25}$ samples collected in the communities of the Los Angeles-Long Beach harbour (United States) was most strongly associated with the water-soluble and water-insoluble organics, PAHs and vanadium. In continuation, Verma et al. (2011) demonstrated that the oxidative potential of Los Angeles quasi-ultrafine $PM_{0.25}$ in the dithiothreitol assay was progressively lost by removing semi-volatile organics by heating the PM samples at 50–200 °C, which does not affect EC or metals. In a regression analysis, the cellular generation of reactive oxygen species was associated especially with OC and PAHs.

Some recent toxicological studies on wood combustion emission PM (Jalava et al., 2010) and on fossil diesel emission PM (Biswas et al., 2009) also stressed the role of organic constituents, for example, PAHs or various water-soluble organics, including organic acids, in toxicity and suggested a carrier role for EC. Given the large number of exposure variables compared to the number of observations in animals or cell cultures, the causality of certain chemical compositions cannot be assessed for certain.

So far, very few studies have aimed to distinguish between the effects due to BC and those of other constituents of fine PM, or have really focused on the mechanisms of action of BC in PM mixtures. There are also suggestions that different chemical compositions may share similar biological mechanisms, leading to the adverse effects seen in experimental studies. Thus, on the basis of present knowledge, there is no convincing evidence supporting the hypothesis that some of the mechanisms of action of ambient air PM are specific for BC.

Inhalation toxicity studies using pure carbon particles at concentrations of environmental relevance have uniformly shown no adverse effects, even when sensitive animal models have been used (Lippmann & Chen, 2009). Because PM mass equals total EC mass in these studies, they will not

expand an understanding of the role of EC or BC in the complex toxicity of ambient air PM mass in any way other than suggesting that pure EC may not possess strong toxic properties.

Conclusions

At present, it is not possible to say definitively whether health effects due to exposure to BC or PM mass are different qualitatively (for example, different health outcomes) and/or quantitatively from each other. This is partly due to the fact that an insufficient number of controlled health studies have been implemented which involve human subjects with simultaneous BC or EC measurements and other PM speciation.

The present very few toxicological in vivo and in vitro studies of urban air and combustion emission fine PM with in-depth chemical characterization of the investigated PM samples suggest that EC (or BC) may not be a directly toxic component of fine PM. It may, however, operate as a universal carrier of a wide variety of chemical constituents of varying toxicity, such as semi-volatile organics, to sensitive pulmonary and cardiovascular targets. Because of this role, EC may very well act as a good indicator for combustion-derived and potentially very harmful parts of PM. More information is, however, needed on the mechanisms of the potential toxicity of BC (or EC) both alone and as a component of urban air fine and ultrafine PM. The comparison of toxicity should be done with simultaneously released, combustion-derived and subsequently transformed organic constituents (for example, PAHs, quinones, dicarboxylic acids) and metals (for example, nickel, vanadium, arsenic), and with other transition metals and soil minerals released from other combustion sources such as the metal industry and non-combustion mechanics in traffic (brakes, tyres, etc.).

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Annex 1

LITERATURE SEARCH CRITERIA

Metrics used to estimate the exposure to BC in health studies: strengths and weaknesses

The literature search was made in the Information Sciences Institute Web of Science database, which was considered to give the largest coverage of relevant literature to the present topic. There were two reasons to limit the literature search to 2000–2010. First, it was discovered that the biases associated with the wide variety of commonly used optical and thermal optical measurement techniques had not been systematically investigated and revealed before the large inter-comparison workshops or campaigns conducted since 2002. Second, the review of literature performed for Chapter 3 (Effects of BC exposure observed in epidemiological studies) summarized the older comparisons of optical BS and Abs determinations reflecting soot with more specific thermal optical methods than for EC.

The literature search was performed on 6 April 2011 as described below.

Topic 1 = (partic*) AND **Topic 2** (black carbon AND elemental carbon AND light absorption) AND **Topic 3**: (intercomp* OR inter-comp* OR correl* OR intercorrel* OR inter-correl*). This search resulted in 33 records. The criteria for the selected articles were: review article; and original science articles with inter-comparison of at least three methods in one urban source environment, or comparison of at least two methods in more than one urban source environment or in one environment during different seasons.

Assessment of exposure to BCP in epidemiological studies

The literature search was conducted within the Web of Science database, which provides the most complete coverage of papers dealing with exposure assessment. The search syntaxes used were:

- **search 1** (absorbance OR “black smoke” OR “elemental carbon” or “black carbon”) AND “air pollution” AND exposure;
- **search 2** (absorbance OR “black smoke” OR “elemental carbon” or “black carbon”) AND personal AND indoor.

In addition, the reference lists of the papers identified were used to find additional literature.

Since the overall goal of the review was to evaluate whether BCP has any health effects that are independent of those of $PM_{2.5}$, papers that included assessments of exposure to both $PM_{2.5}$ and BCP were preferred when compiling the tables for this working paper. Further, studies in northern America and Europe were preferred in view of the focus area of the Task Force. Studies with central monitoring sites close to traffic, as well as studies where ambient $PM_{2.5}$ and BCP were not measured at the same location, were excluded. To avoid repetition, only one study per study area was usually included in each table, unless the studies were conducted at clearly different times.

Effects of BC exposure observed in epidemiological studies

For time-series studies on daily mortality and hospital admissions and cohort studies, the literature search was used as described in Janssen et al. (2011), for which a search was conducted for peer-reviewed literature in Medline (January 2010) for epidemiological studies that evaluated the health effects of (a measure of) PM mass as well as health effects of (a measure of) BCP. Here the following keywords were used: (British smoke or black smoke or black carbon or elemental carbon or EC or soot or absorbance or absorption coefficient) AND (Particles or particulate matter or particulates or particulate air pollution or fine partic* or "PM10" or "PM2.5" or "PM($_{2.5}$)" or "PM(10)" or sulfate* or sulphate*) AND (mortality or cohort or hospital or emergency).

Panel studies among children were identified by adding (panel or children or subjects or participants) to the keywords. For health effects due to long-term exposure, only cohort studies were considered as these have been the most relevant for standard-setting.

All abstracts were scanned and papers retrieved that potentially included effects estimates for PM mass as well as for BCP. For the time-series studies, APED at St George's, University of London, was also used to identify suitable studies. This database comprises standardized estimates extracted from ecological time-series studies identified by systematic review that meet certain quality criteria, with the last retrieval performed on 15 May, 2009 (Smith et al., 2009). APED was searched for estimates related to the effects of PM_{10}, $PM_{2.5}$, PM_{13}, TSP or sulfate as well as BS, BC or EC. For the panel studies, an evaluation was made of all papers included in the Weinmayr et al. (2010) review that provided effects estimates for PM_{10} to see whether they reported effects estimates of BCP as well.

Forty papers were identified on time-series studies on daily mortality or hospital admissions that included area-specific estimates for both PM and BCP, 11 papers on panel studies among symptomatic children and 17 papers on cohort studies. The APED search identified six papers that were not identified in the Medline search, but four were excluded because more recent estimates from the same city were available. The Medline search identified four papers published after the last APED systematic review in May 2009. For the panel studies, the Medline search only identified the paper on the combined effects estimates of all 28 panels from 14 countries in the PEACE study, whereas the Weinmayr et al. (2010) review included 14 papers that provided country-specific estimates. All other papers included in the Weinmayr et al. (2010) paper that included effects estimates for PM mass as well as BCP were also identified in the Medline search. The Medline search identified one panel study not included in the Weinmayr et al. (2010) paper that was published in 2009 (after the retrieval date for the Weinmayr review).

For the time-series studies on daily mortality and hospital admissions, five papers on total suspended particulates were excluded as more recent data, including effects estimates for PM_{10}, were available for most of the cities. Also, one paper was excluded on a rare health endpoint (hospital admissions for headache), resulting in a total of 34 papers included in the review. All studies included were adjusted for major confounders, specifically seasonality and non-linear function of temperature and relative humidity. For the panel studies, two papers were excluded that focused on heterogeneity/subgroup analysis of effect presented in other papers. For the cohort studies, two papers on birth outcomes were excluded.

Evidence from toxicology, including human clinical studies

After the initial search in Medline and Pubmed (21 March 2011) according to the search protocol described below, additional searches (April 2011) for human clinical studies were performed in Pubmed with similar search terms (black smoke, black carbon, elemental carbon, soot, EC, particulate matter, carbonaceous component, human clinical, toxicity, inhalation exposure) to supplement the database. References published in epidemiological journals were removed manually to reduce noise, which resulted in a database of 619 papers. Thereafter, articles in the database with some term of BC were assigned to a group (epi, mechanisms, method, tox animal, tox human, tox in vitro). A total of 359 papers were assigned to specific groups, and 260 papers remained unfiled. This resulted in 76 articles on human clinical studies and 50 articles filed under mechanisms; 110 papers presented in vitro toxicology studies and 52 papers presented animal toxicology studies.

Strategy for literature search on controlled human studies

Initial start in Medline (1990–2011) using the following search protocol:

1 exp Inhalation Exposure/ (4675)
2 exp Toxicity Tests/ (73653)
3 (toxic* or exposure* or inflammation or cardiovascular* or pulmonar* or lung or inhal* or respirat* or vascular* or allergic* or airway*).mp. [mp=protocol supplementary concept, rare disease supplementary concept, title, original title, abstract, name of substance word, subject heading word, unique identifier] (2733444)
4 1 or 2 or 3 (2774555)
5 *Smoke/ (3137)
6 exp Soot/ (290)
7 *Vehicle Emissions/ae, po, to (1503)
8 ((diesel adj3 exhaust) or wood smoke or woodsmoke or black smoke or combustion or black carbon or (elemental adj3 carbon) or carbon black or carbonaceous nanopartic* or soot).ti,ab. (11112)
9 (((air or airborne or atmospheric or ambient or carbon or ultrafine) adj6 (particle* or particulate*)) or (diesel adj3 exhaust) or wood smoke or woodsmoke or black smoke or black carbon or (elemental adj3 carbon) or carbon black or carbonaceous nanopartic* or soot or (combustion adj3 (smoke or carbon))).ti. (5086)
10 5 or 6 or 7 or 8 (14664)
11 4 and 10 (7427)
12 (human* or man or men or volunteer*).mp. [mp=protocol supplementary concept, rare disease supplementary concept, title, original title, abstract, name of substance word, subject heading word, unique identifier] (11940675)
13 11 and 12 (4460)
14 13 and 9 (744)
15 limit 14 to year=1990-2011 (665)

Combined with a search in PubMed (only last year to catch e-publications not yet published in Medline) according to the following protocol (111 records):
#1 “diesel exhaust”[ti] OR “wood smoke”[ti] OR woodsmoke[ti] OR “black smoke”[ti] OR combustion[ti] OR “black carbon”[ti] OR “elemental carbon”[ti] OR “carbon black”[ti] OR “carbonaceous nanoparticle”[ti] OR “carbonaceous nanoparticles”[ti] OR soot[Title] 05:54:24 3540

#2 toxic*[tiab] or exposure*[tiab] or inflammation[tiab] or cardiovascular*[tiab] or pulmonar*[tiab] or lung[tiab] or inhal*[tiab] or respirat*[tiab] or vascular*[tiab] or allergic*[tiab] or airway*[tiab]; limits: published in the last 1 year 06:01:29 135983
#3 #1 AND #2 limits: published in the last 1 year 06:04:31 **111.**

An additional literature search was made in the Web of Science specifically addressing the mechanisms of toxicity. The literature search covered 1990–2010 (and, to some extent, early 2011).

LITERATURE SEARCH (18 April 2011): Topic 1 = (urban air AND partic*) AND Topic 2 = (EC OR OC OR POM OR elemental carbon OR organic carbon OR partic* organic matter) AND Topic 3 = (toxic* OR inflammat* OR genotoxic* OR cytotoxic* OR blood coagul* OR immunotoxic*) AND Topic 4 = (mouse OR rat OR macrophage* OR epithel*) AND Topic 5 = (experimental OR exposure OR CAP* OR lung OR respir* OR heart OR blood OR brain). This search resulted in identification of **46 papers**.

The criteria for selection of the original science articles reviewed were: animal, cell and cell-free in vitro studies on urban air fine PM with in-depth chemical characterization of the investigated PM samples (EC, OC/POM, major ions, water-soluble and/or water-insoluble elements, including transition metals and soil mineral components). The search results were complemented by papers cited in the selected studies and by relevant new literature appearing as linked to the identified papers in the Web of Science.

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Annex 2

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Annex 3

SUPPLEMENTARY MATERIAL TO THE REVIEW OF EPIDEMIOLOGICAL STUDIES

Annex 3A. Estimation of EC from BS or Abs of $PM_{2.5}$ filters

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Annex 3H. Comparison of calculated health benefits of traffic abatement measures using $PM_{2.5}$ or BCP

Annex 3A. Estimation of EC from BS or Abs of $PM_{2.5}$ filters

Table 3A 1. Estimation of EC from BS or Abs of $PM_{2.5}$ filters

Reference	Study period	Locations	Method		R[a]	Regression equation		Increase in EC per 10 µg/m³ increase in BS[b]
			EC	BS/Abs		Intercept	Slope	
Edwards et al., 1983	NA	Washington, United States; urban + traffic	Thermal optical[c]	BS	0.82	-0.1	0.13	1.3
Erdman, Israel & Ulrich, 1993	1989/1990	Berlin, Germany; urban	VDI 3481	BS	0.93	0	0.23	1.8
Schaap & Denier van der Gon, 2007	1998/1999; 2001/2002	Netherlands; urban	Sunset	BS	0.92	0.32	0.09	0.9
		Netherlands; rural	Sunset	BS	0.87	0.16	0.06	0.6
Kinney et al., 2000	1996	New York, United States; urban + traffic	Sunset	Abs	0.95	0	0.83	0.8
Janssen et al., 2001[d]	1997/1998	Netherlands; urban + traffic	VDI 2465-part 1	Abs	0.92	0	2.11	1.7
Lena et al., 2002	1999	New York, United States; urban + traffic	Sunset	Abs	0.90	0	0.52	0.5
Adams et al., 2002	1999/2000	London, United Kingdom; urban + traffic	NIOSH	Abs	0.98	0	1.21	1.2
Cyrys et al., 2003	1999/2000	Munich, Germany; urban + traffic	VDI 2465-part 1	Abs	0.97	-1.19	2.02	1.6
Cyrys et al., 2003	1999/2000	Netherlands; rural, urban, traffic	VDI 2465-part 1	Abs	0.97	-0.26	1.61	1.3
Cyrys et al., 2003	1999/2000	Sweden; rural, urban, traffic	VDI 2465-part 1	Abs	0.85	0.36	0.90	0.7
							Mean[e]	**1.1**
							Minimum	**0.5**
							Maximum	**1.8**

[a] Coefficient of the correlation between EC and BCP concentrations.
[b] Results from studies that have used the VideoOverIP (VDI) protocol were divided by 1.25, as this method has been shown to overestimate EC by, on average, 25% (Schmid et al., 2001). An increase in 1 unit of Abs is considered to equal an increase of 10 µg/m³ BS, according to Roorda-Knape et al. (1998).
[c] Measurement method not further specified.
[d] Paper presents regression equation as Abs=EC; inverse equation, forced through zero, calculated using the original data from the study.
[e] Mean, minimum and maximum of all 11 values; mean for BS only=1.16 (range: 0.6–1.8; n=4); mean for Abs only=1.12 (range 0.5–1.7; n=7).

Source: Janssen et al., 2011 (supplemental material, Table A1).

Annex 3B. Single-city estimates for mortality and hospital admissions in studies that include both PM_{10} and BS

Table 3B 1. All-cause mortality, all ages[a]

Reference	Location	Estimate PM_{10}		Estimate BS		IQR ($\mu g/m^3$)		Concentration[b]		Correlation (R) PM–BS[c]	Period	Selected lag[d]
		Beta	Standard error	Beta	Standard error	PM_{10}	BS	PM_{10}	BS			
Verhoeff et al., 1996	*Amsterdam*	0.00060	0.00038	0.00171	0.00077	22	10	38	12	0.51	1986–1992	Lag 0
Roemer & van Wijnen, 2001a	*Amsterdam*	0.00027	0.00020	0.00324	0.00093	18	7	39	10	NA	1987–1994	Lag 1
Katsouyanni et al., 2001	Athens[d]	0.00153	0.00028	0.00065	0.00012	*NA*	*NA*	40	64	NA	1992–1996	Lag 0–1
Katsouyanni et al., 2001	**Barcelona**	0.00093	0.00018	0.00157	0.00027	24[f]	18[f]	60	39	NA	1991–1996	Lag 0–1
Katsouyanni et al., 2001	Birmingham	0.00028	0.00026	0.00034	0.00047	15[f]	7[f]	21	11	NA	1992–1996	Lag 0–1
Katsouyanni et al., 2001	Cracow[e]	0.00013	0.00035	-0.00021	0.00021	*NA*	*NA*	54	36	NA	1990–1996	Lag 0–1
Zeghnoun et al., 2001a	**Le Havre**	0.00079	0.00057	0.00026	0.00085	24	12	36	16	0.70	1990–1995	PM lag 1/ BS lag 0–1
Katsouyanni et al., 2001	***London***	0.00069	0.00017	0.00093	0.00030	14[f]	8[f]	25	11	NA	1992–1996	Lag 0–1
Bremner et al., 1999	*London*	0.00026	0.00023	0.00074	0.00038	*NA*	*NA*	28	13	NA	1992–1994	Lag 1
Hoek et al., 2000	**Netherlands**	0.00018	0.00008	0.00040	0.00010	23[f]	9[f]	34	10	0.77	1986–1994	Lag 1
Zeghnoun et al., 2001b	*Paris*	0.00066	0.00020	0.00043	0.00015	15	14	22	16	NA	1990–1995	Lag 1
Katsouyanni et al., 2001	***Paris***	0.00043	0.00023	0.00038	0.00015	13[f]	15[f]	22	21	NA	1991–1996	Lag 0–1
Zeghnoun et al., 2001a	**Rouen**	0.00024	0.00040	0.00035	0.00083	21	14	33	19	0.73	1990–1995	Lag 1
Anderson et al., 2001	**West Midlands**	0.00008	0.00042	0.00036	0.00064	*NA*	*NA*	23	13	0.64	1994–1996	Lag 0–1
Percentage change per 10 $\mu g/m^3$ increase		*Percentage*	*95% CI*	*Percentage*	*95% CI*							
Pooled fixed effects		0.34	(0.23–0.47)	0.52	(0.37–0.66)							
Pooled random effects		0.48	(0.18–0.79)	0.68	(0.31–1.06)							
Heterogeneity chi-squared (df=6)		Q=19.9	P=0.003	Q=19.2	P=0.004							

[a] Locations in italics occur more than once; locations in bold included in meta-analysis.
[b] Mean or median ($\mu g/m^3$).
[c] Coefficient of the correlation between PM_{10} and BS concentrations.
[d] In cases where multiple lags are reported in Chapter 3, the estimate discussed by the author was used, as indicated in the APED as "selected lag".
[e] Excluded from meta-analyses because PM_{10} was partly derived from BS.
[f] Taken from APHEA II paper on hospital admissions (Le Tertre et al., 2002).

Source: Janssen et al., 2011 (supplemental material, Tables B1–10).

Table 3B 2. CVD mortality, all ages[a]

Reference	Location	Estimate PM_{10}		Estimate BS		IQR (µg/m^3)		Concentration[b]		Correlation (R) PM–BS[c]	Period	Selected lag[d]
		Beta	Standard error	Beta	Standard error	PM_{10}	BS	PM_{10}	BS			
Analitis et al., 2006	Athens[e]	0.00167	0.00045	0.00069	0.00018	*NA*	*NA*	40	64	NA	1992–1996	Lag 0–1
Analitis et al., 2006	**Barcelona**	0.00055	0.00032	0.00137	0.00050	24[c]	18[f]	60	39	NA	1991–1996	Lag 0–1
Analitis et al., 2006	Birmingham	0.00021	0.00040	0.00039	0.00071	15[c]	7[f]	21	11	NA	1992–1996	Lag 0–1
Analitis et al., 2006	Cracow[e]	0.00032	0.00052	-0.00007	0.00031	*NA*	*NA*	54	36	NA	1990–1996	Lag 0–1
Zeghnoun et al., 2001a	**Le Havre**	0.00252	0.00126	0.00164	0.00155	24	12	36	16	0.70	1990–1995	PM lag1; BS lag 0–3
Analitis et al., 2006	***London***	0.00091	0.00028	0.00156	0.00046	14[f]	8[f]	25	11	NA	1992–1996	Lag 0–1
Bremner et al., 1999	*London*	0.00055	0.00031	0.00117	0.00066	*NA*	*NA*	28	13	NA	1992–1994	Lag 1
Hoek et al., 2000	*Netherlands*	0.00019	0.00018	0.00079	0.00020	23[f]	9[f]	34	10	0.77	1986–1994	lag 0–6
Analitis et al., 2006	***Netherlands***	0.00017	0.00016	0.00026	0.00027	23[f]	9[f]	33	9	NA	1990–1995	Lag 0–1
Hoek et al., 2001	*Netherlands*	0.00015	0.00018	0.00071	0.00020	23[f]	9[f]	34	10	0.77	1992/1986–1994[g]	Lag 0–6
Zeghnoun et al., 2001b	*Paris*	0.00086	0.00037	0.00036	0.00029	15	14	22	16	NA	1990–1995	PM lag 2; BS lag 1
Analitis et al., 2006	***Paris***	0.00081	0.00047	0.00063	0.00029	13[f]	15[f]	22	21	NA	1991–1996	Lag 0–1
Zeghnoun et al., 2001a	**Rouen**	0.00106	0.00069	0.00276	0.00155	21	14	33	19	0.73	1990–1995	Lag 1
Anderson et al., 2001	**West Midlands**	0.00041	0.00061	0.00089	0.00092	*NA*	*NA*	23	13	0.64	1994–1996	Lag 0–1
Percentage change per 10 µg/m^3 increase		*Percentage*	*95% CI*	*Percentage*	*95% CI*							
Pooled fixed effects		0.45	(0.22–0.68)	0.73	(0.41–1.06)							
Pooled random effects		0.60	(0.23–0.97)	0.90	(0.40–1.41)							
Heterogeneity chi-squared (df=6)		Q=9.9	P=0.127	Q=10.2	P=0.116							

[a] Locations in italics occur more than once; locations in bold included in meta-analysis.
[b] Mean or median (µg/m^3).
[c] Coefficient of the correlation between PM_{10} and BS concentrations.
[d] In cases where multiple lags are reported in Chapter 3, the estimate discussed by the author was used, as indicated in the APED as "selected lag".
[e] Excluded from meta-analyses because PM_{10} was partly derived from BS.
[f] Taken from APHEA II paper on hospital admissions (Le Tertre et al., 2002).
[g] 1992–1994 for PM_{10} ; 1986–1994 for BS.

Table 3B 3. Respiratory diseases mortality, all ages[a]

Reference	Location	Estimate PM_{10}		Estimate BS		IQR (µg/m^3)		Concentration[b]		Correlation (R) PM–BS[c]	Period	Selected lag[d]
		Beta	Standard error	Beta	Standard error	PM_{10}	BS	PM_{10}	BS			
Analitis et al., 2006	Athens[e]	0.00101	0.00122	0.00006	0.00048	*NA*	*NA*	40	64	NA	1992–1996	Lag 0–1
Analitis et al., 2006	**Barcelona**	0.00117	0.00075	0.00374	0.00100	24[f]	18[f]	60	39	NA	1991–1996	Lag 0–1
Analitis et al., 2006	Birmingham	0.00003	0.00078	0.00069	0.00130	15[f]	7[f]	21	11	NA	1992–1996	Lag 0–1
Analitis et al., 2006	Cracow[e]	0.00529	0.00216	0.00357	0.00132	*NA*	*NA*	54	36	NA	1990–1996	Lag 0–1
Zeghnoun et al., 2001a	**Le Havre**	0.00200	0.00196	0.00249	0.00294	24	12	36	16	0.70	1990–1995	PM lag 2; BS lag 0–1
Analitis et al., 2006	***London***	0.00022	0.00044	-0.00034	0.00073	14[f]	8[f]	25	11	NA	1992–1996	Lag 0–1
Bremner et al., 1999	*London*	0.00128	0.00050	0.00190	0.00084	*NA*	*NA*	28	13	NA	1992–1994	Lag 3
Analitis et al., 2006	***Netherlands***	0.00031	0.00036	0.00029	0.00061	23[f]	9[f]	33	9	NA	1990–1995	Lag 0–1
Dab et al., 1996	*Paris*	0.00155	0.00059	0.00069	0.00048	NA	NA	51	32	NA	1987–1992	PM lag 0–1; BS lag1
Analitis et al., 2006	***Paris***	-0.00121	0.00095	0.00063	0.00029	13[f]	15[f]	22	21	NA	1991–1996	Lag 0–1
Zeghnoun et al., 2001a	**Rouen**	0.00176	0.00120	0.00201	0.00310	21	14	33	19	0.73	1990–1995	Lag 0–1
Anderson et al., 2001	**West Midlands**	-0.00058	0.00100	0.00006	0.00153	*NA*	*NA*	23	13	0.64	1994–1996	Lag 0–1
Percentage change per 10 µg/m^3 increase		*Percentage*	*95% CI*	*Percentage*	*95% CI*							
Pooled fixed effects		0.31	(-0.16–0.78)	0.70	(-0.05–1.45)							
Pooled random effects		0.31	(-0.23–0.86)	0.95	(-0.31–2.22)							
Heterogeneity chi-squared (df=6)		Q=6.9	P=0.329	Q=12.5	P=0.051							

[a] Locations in italics occur more than once; locations in bold included in meta-analysis.
[b] Mean or median (µg/m^3).
[c] Coefficient of the correlation between PM_{10} and BS concentrations.
[d] In cases where multiple lags are reported in Chapter 3, the estimate discussed by the author was used, as indicated in the APED as "selected lag".
[e] Excluded from meta-analyses because PM_{10} was partly derived from BS.
[f] Taken from APHEA II paper on hospital admissions (Le Tertre et al., 2002).

Table 3B 4. Hospital admissions, respiratory diseases, elderly people[a]

Reference	Location	Estimate PM_{10}		Estimate BS		IQR (μg/m^3)		Concentration[b]		Correlation (R) PM–BS[c]	Period	Selected lag[d]
		Beta	Standard error	Beta	Standard error	PM_{10}	BS	PM_{10}	BS			
Atkinson et al., 2001	**Barcelona**	0.00198	0.00060	-0.00070	0.00083	24	18	56	39	0.5–0.8[e]	1994–1996	Lag 0–1
Atkinson et al., 2001	Birmingham	0.00090	0.00061	0.00286	0.00115	15	7	25	13	0.5–0.8[e]	1992–1994	Lag 0–1
Prescott et al., 1998	**Edinburgh**	0.00208	0.00304	0.00305	0.00338	NA	NA	21	9	0.4	1992–1995	Lag 1–3
Atkinson et al., 2001	***London***	0.00040	0.00036	-0.00111	0.00068	14	8	28	13	0.5–0.8[e]	1992–1994	Lag 0–1
Atkinson et al., 1999b	*London*	0.00096	0.00041	0.00082	0.00063	NA	NA	29	13	0.6–0.7	1992–1994	Lag 3
Atkinson et al., 2001	**Netherlands**	0.00119	0.00025	0.00000	0.00036	23	9	40	13	0.5–0.8[e]	1992/1989–1995[f]	Lag 0–1
Atkinson et al., 2001	**Paris**	-0.00010	0.00062	0.00050	0.00046	13	15	23	23	0.5–0.8[e]	1992–1996	Lag 0–1
Anderson et al., 2001	**West Midlands**	-0.00045	0.00069	-0.00018	0.00100	*NA*	*NA*	23	13	0.64	1994–1996	Lag 0–1
Percentage change per 10 μg/m^3 increase		*Percentage*	*95% CI*	*Percentage*	*95% CI*							
Pooled fixed effects		0.85	(0.49–1.20)	–0.07	(–0.58–0.44)							
Pooled random effects		0.70	(0.00–1.40)	–0.06	(–0.53–0.41)							
Heterogeneity chi-squared (df=5)		Q=13.1	P=0.023	Q=5.4	P=0.372							

[a] Locations in italics occur more than once; locations in bold included in meta-analysis.
[b] Mean or median (μg/m^3).
[c] Coefficient of the correlation between PM_{10} and BS concentrations.
[d] In cases where multiple lags are reported in Chapter 3, the estimate discussed by the author was used, as indicated in the APED as "selected lag".
[e] Range in correlation coefficient for all eight cities described in Atkinson et al. (2001) (three cities were not included in this review as no data were available on BS).
[f] 1992–1995 for PM_{10}; 1989–1995 for BS.

Table 3B 5. Hospital admissions, respiratory diseases – asthma and chronic obstructive pulmonary disease, ages ≥65 years[a]

Reference	Location	Estimate PM_{10}		Estimate BS		IQR (µg/m^3)		Concentration[b]		Correlation (R) PM–BS[c]	Period	Selected lag[d]
		Beta	Standard error	Beta	Standard error	PM_{10}	BS	PM_{10}	BS			
Atkinson et al., 2001	**Barcelona**	0.00257	0.00080	-0.00212	0.00116	24	18	56	39	0.5–0.8[e]	1994–1996	Lag 0–1
Atkinson et al., 2001	**Birmingham**	0.00050	0.00097	0.00218	0.00199	15	7	25	13	0.5–0.8[e]	1992–1994	Lag 0–1
Atkinson et al., 2001	***London***	0.00030	0.00056	0.00040	0.00103	14	8	28	13	0.5–0.8[e]	1992–1994	Lag 0–1
Atkinson et al., 1999	*London*	0.00227	0.00137	-0.00091	0.00099	NA	NA	29	13	0.6–0.7	1992–1994	Lag 3
Atkinson et al., 2001	**Netherlands**	0.00109	0.00030	0.00070	0.00046	23	9	40	13	0.5–0.8[e]	1992/1989–1995[f]	Lag 0–1
Atkinson et al., 2001	**Paris**	-0.00060	0.00098	0.00020	0.00077	13	15	23	23	0.5–0.8[e]	1992–1996	Lag 0–1
Percentage change per 10 µg/m^3 increase		*Percentage*	*95% CI*	*Percentage*	*95% CI*							
Pooled fixed effects		0.95	(0.48–1.42)	0.36	(–0.81–1.54)							
Pooled random effects		0.86	(0.03–1.70)	0.22	(–0.73–1.18)							
Heterogeneity chi-squared (df=4)		Q=8.3	P=0.08	Q=6.0	P=0.199							

[a] Locations in italics occur more than once; locations in bold included in meta-analysis.
[b] Mean or median (µg/m^3).
[c] Coefficient of the correlation between PM_{10} and BS concentrations.
[d] In cases where multiple lags are reported in Chapter 3, the estimate discussed by the author was used, as indicated in the APED as "selected lag".
[e] Range in correlation coefficient for all eight cities described in Atkinson et al. (2001) (three cities were not included in this review as no data were available on BS).
[f] 1992–1995 for PM_{10}; 1989–1995 for BS.

Table 3B 6. Hospital admissions, respiratory diseases, asthma, ages 0–14 years[a]

Reference	Location	Estimate PM_{10}		Estimate BS		IQR ($\mu g/m^3$)		Concentration[b]		Correlation (R) PM–BS[c]	Period	Selected lag[d]
		Beta	Standard error	Beta	Standard error	PM_{10}	BS	PM_{10}	BS			
Atkinson et al., 2001	**Barcelona**	0.00266	0.00392	0.00989	0.00484	24	18	56	39	0.5–0.8[e]	1994–1996	Lag 0–1
Atkinson et al., 2001	Birmingham	0.00276	0.00100	0.00198	0.00199	15	7	25	13	0.5–0.8[e]	1992–1994	Lag 0–1
Atkinson et al., 2001	***London***	0.00060	0.00072	0.00109	0.00123	14	8	28	13	0.5–0.8[e]	1992–1994	Lag 0–1
Atkinson et al., 1999	*London*	0.00324	0.00203	0.00245	0.00179	NA	NA	29	13	0.6–0.7	1992–1994	Lag 3
Atkinson et al., 2001	**Netherlands**	-0.00090	0.00062	0.00139	0.00091	23	9	40	13	0.5–0.8[e]	1992/1989–1995[f]	Lag 0–1
Atkinson et al., 2001	**Paris**	0.00070	0.00113	0.00090	0.00087	13	15	23	23	0.5–0.8e[d]	1992–1996	Lag 0–1
Anderson et al., 2001	**West Midlands**	0.00797	0.00321	0.00714	0.00329	*NA*	*NA*	23	13	0.64	1994–1996	Lag 0–1
Percentage change per 10 $\mu g/m^3$ increase		*Percentage*	*95% CI*	*Percentage*	*95% CI*							
Pooled fixed effects		0.24	(–0.56–1.05)	1.47	(0.41–2.54)							
Pooled random effects		0.69	(–0.74–2.14)	1.64	(0.28–3.02)							
Heterogeneity chi-squared (df=4)		Q=9.5	P=0.050	Q=5.6	P=0.231							

[a] Locations in italics occur more than once; locations in bold included in meta-analysis.
[b] Mean or median ($\mu g/m^3$).
[c] Coefficient of the correlation between PM_{10} and BS concentrations.
[d] In cases where multiple lags are reported in Chapter 3, the estimate discussed by the author was used, as indicated in the APED as "selected lag".
[e] Range in correlation coefficient for all eight cities described in Atkinson et al. (2001) (three cities were not included in this review as no data were available on BS).
[f] 1992–1995 for PM_{10}; 1989–1995 for BS.

Table 3B 7. Hospital admissions, respiratory diseases, asthma, ages 15–64 years[a]

Reference	Location	Estimate PM_{10}		Estimate BS		IQR ($\mu g/m^3$)		Concentration[b]		Correlation (R) PM–BS[c]	Period	Selected lag[d]
		Beta	Standard error	Beta	Standard error	PM_{10}	BS	PM_{10}	BS			
Atkinson, 2001	**Barcelona**	0.00040	0.00202	0.00208	0.00121	24	18	56	39	0.5–0.8[e]	1994–1996	Lag 0–1
Atkinson, 2001	Birmingham	0.00247	0.00121	0.00276	0.00239	15	7	25	13	0.5–0.8[e]	1992–1994	Lag 0–1
Atkinson, 2001	***London***	0.00139	0.00076	0.00178	0.00137	14	8	28	13	0.5–0.8[e]	1992–1994	Lag 0–1
Atkinson, 1999	*London*	0.00555	0.00249	0.00234	0.00224	NA	NA	29	13	0.6–0.7	1992–1994	PM lag 3; BS lag2
Atkinson, 2001	**Netherlands**	0.00040	0.0006	-0.00040	0.00093	23	9	40	13	0.5–0.8[e]	1992/89–1995[f]	Lag 0–1
Atkinson, 2001	**Paris**	0.00119	0.00097	0.00080	0.00076	13	15	23	23	0.5–0.8[e]	1992–1996	Lag 0–1
Anderson, 2001	**West Midlands**	-0.00233	0.00419	-0.00284	0.00432	*NA*	*NA*	23	13	0.64	1994–1996	Lag 0–1
Percentage change per 10 $\mu g/m^3$ increase		*Percentage*	*95% CI*	*Percentage*	*95% CI*							
Pooled fixed effects		0.77	(-0.05–1.61)	0.52	(-0.50–1.55)							
Pooled random effects		0.77	(-0.05–1.61)	0.52	(-0.50–1.55)							
Heterogeneity chi-squared (df=4)		Q=2.2	P=0.697	Q=3.1	P=0.549							

[a] Locations in italics occur more than once; locations in bold included in meta-analysis.
[b] Mean or median ($\mu g/m^3$).
[c] Coefficient of the correlation between PM_{10} and BS concentrations.
[d] In cases where multiple lags are reported in Chapter 3, the estimate discussed by the author was used, as indicated in the APED as "selected lag".
[e] Range in correlation coefficient for all eight cities described in Atkinson et al. (2001) (three cities were not included in this review as no data were available on BS).
[f] 1992–1995 for PM_{10}; 1989–1995 for BS.

Table 3B 8. Hospital admissions, cardiac diseases, ages ≥65 years[a]

Reference	Location	Estimate PM_{10}		Estimate BS		IQR (μg/m^3)		Concentration[b]		Correlation (R) PM–BS[c]	Period	Selected lag[d]
		Beta	Standard error	Beta	Standard error	PM_{10}	BS	PM_{10}	BS			
Le Tertre et al., 2002	**Barcelona**	0.00050	0.00046	0.00066	0.00064	24	18	56	39	0.5–0.8[e]	1994–1996	Lag 0–1
Le Tertre et al., 2002	Birmingham	-0.00014	0.00039	0.00114	0.00078	15	7	25	13	0.5–0.8[e]	1992–1994	Lag 0–1
Le Tertre et al., 2002	**London**	0.00104	0.00027	0.00214	0.00049	14	8	28	13	0.5–0.8[e]	1992–1994	Lag 0–1
Le Tertre et al., 2002	**Paris**	0.00020	0.00028	0.00057	0.00022	13	15	23	23	0.5–0.8[e]	1992–1996	Lag 0–1
Anderson et al., 2001	**West Midlands**	0.00030	0.00108	0.00169	0.00117	*NA*	*NA*	23	13	0.64	1994–1996	Lag 0–1
Percentage change per 10 μg/m^3 increase		*Percentage*	*95% CI*	*Percentage*	*95% CI*							
Pooled fixed effects		0.54	(0.21–0.87)	0.83	(0.47–1.19)							
Pooled random effects		0.51	(0.04–0.98)	1.07	(0.27–1.89)							
Heterogeneity chi-squared (df=3)		Q=5.7	P=0.129	Q=8.8	P=0.032							

[a] Locations in bold included in meta-analysis
[b] Mean or median (μg/m^3).
[c] Coefficient of the correlation between PM_{10} and BS concentrations.
[d] In cases where multiple lags are reported in Chapter 3, the estimate discussed by the author was used, as indicated in the APED as "selected lag".
[e] Range in correlation coefficient for all eight cities described in Le Tertre et al. (2002). No information on cardiac admissions is available for the Netherlands. Three other cities are not included in this review as no data were available on BS.

Table 3B 9. Hospital admissions, cardiac diseases, ages ≥65 years[a]

Reference	Location	Estimate PM_{10}		Estimate BS		IQR (µg/m^3)		Concentration[b]		Correlation (R) PM–BS[c]	Period	Selected lag[d]
		Beta	Standard error	Beta	Standard error	PM_{10}	BS	PM_{10}	BS			
Le Tertre et al., 2002	**Barcelona**	0.00068	0.00055	0.00130	0.00075	24	18	56	39	0.5–0.8[e]	1994–1996	Lag 0–1
Le Tertre et al., 2002	**Birmingham**	0.00031	0.00047	0.00168	0.00094	15	7	25	13	0.5–0.8[e]	1992–1994	Lag 0–1
Le Tertre et al., 2002	**London**	0.00096	0.00032	0.00227	0.00057	14	8	28	13	0.5–0.8[e]	1992–1994	Lag 0–1
Le Tertre et al., 2002	**Paris**	0.00053	0.00035	0.00042	0.00027	13	15	23	23	0.5–0.8[e]	1992–1996	Lag 0–1
Percentage change per 10 µg/m^3 increase		*Percentage*	*95% CI*	*Percentage*	*95% CI*							
Pooled fixed effects		0.67	(0.28–1.06)	0.86	(0.41–1.30)							
Pooled random effects		0.67	(0.28–1.06)	1.32	(0.28–2.38)							
Heterogeneity chi-squared (df=3)		Q=1.5	P=0.673	Q=9.9	P=0.019							

[a] Locations in bold included in meta-analysis.
[b] Mean or median (µg/m^3).
[c] Coefficient of the correlation between PM_{10} and BS concentrations.
[d] In cases where multiple lags are reported in Chapter 3, the estimate discussed by the author was used, as indicated in the APED as "selected lag".
[e] Range in correlation coefficient for all eight cities described in Le Tertre et al. (2002). No information on cardiac admissions is available for the Netherlands. Three other cities are not included in this review as no data were available on BS.

Table 3B 10. Hospital admissions, ischaemic heart disease, ages ≥65 years[a]

Reference	Location	Estimate PM_{10}		Estimate BS		IQR (μg/m^3)		Concentration[b]		Correlation (R) PM–BSc[c]	Period	Selected lag[d]
		Beta	Standard error	Beta	Standard error	PM_{10}	BS	PM_{10}	BS			
Le Tertre et al., 2002	**Barcelona**	-0.00087	0.00087	0.00061	0.00120	24	18	56	39	0.5–0.8[e]	1994–1996	Lag 0–1
Le Tertre et al., 2002	Birmingham	0.00033	0.00076	-0.00073	0.00150	15	7	25	13	0.5–0.8[e]	1992–1994	Lag 0–1
Le Tertre et al., 2002	***London***	0.00104	0.00049	0.00265	0.00086	14	8	28	13	0.5–0.8[e]	1992–1994	Lag 0–1
Atkinson et al., 1999	*London*	0.00298	0.00128	0.00288	0.00119	NA	NA	29	13	0.6–0.7	1992–1994	PM lag 0; BS lag 3
Le Tertre et al., 2002	**Netherlands**	0.00036	0.00018	0.00100	0.00026	23	9	40	13	0.5–0.8[e]	1992/89–1995[c]	Lag 0–1
Le Tertre et al., 2002	**Paris**	0.00168	0.00057	0.00116	0.00043	13	15	23	23	0.5–0.8[e]	1992–1996	Lag 0–1
Anderson et al., 2001	**West Midlands**	0.00208	0.00209	0.00198	0.00220	*NA*	*NA*	23	13	0.64	1994–1996	Lag 0–1
Percentage change per 10 μg/m^3 increase		*Percentage*	*95% CI*	*Percentage*	*95% CI*							
Pooled fixed effects		0.50	(0.20–0.81)	1.13	(0.72–1.54)							
Pooled random effects		0.68	(0.01–1.36)	1.13	(0.72–1.54)							
Heterogeneity chi-squared (df=4)		Q=8.8	P=0.066	Q=3.6	P=0.463							

[a] Locations in italics occur more than once; locations in bold included in meta-analysis.
[b] Mean or median (μg/m^3).
[c] Coefficient of the correlation between PM_{10} and BS concentrations.
[d] In cases where multiple lags are reported in Chapter 3, the estimate discussed by the author was used, as indicated in the APED as "selected lag".
[e] Range in correlation coefficient for all eight cities described in Le Tertre et al. (2002). No information on cardiac admissions is available for the Netherlands. Three other cities were not included in this review as no data were available on BS.

Annex 3C. Study-specific effects estimates for mortality in studies that include both $PM_{2.5}$ and EC

Table 3C 1. Effects estimates for $PM_{2.5}$ and EC for all-cause mortality

Reference	Location	Estimate $PM_{2.5}$		Estimate EC		IQR (µg/m^3)		Concentration[a]		Correlation (R) PM–EC[b]	Period	Selected lag[c]
		Beta	Standard error	Beta	Standard error	$PM_{2.5}$	EC	$PM_{2.5}$	EC			
Klemm et al., 2004[d]	Atlanta, United States	0.00544	0.00184	0.01343	0.01072	11.6	1.1	19.6	2.0	NA	1998–2000	Lag 01
Ostro et al., 2007[d]	6 counties in California, United States	0.00056	0.00037	0.00829	0.00776	14.6	0.8	19.3	1.0	0.53	2000–2003	Lag 3
Cakmak, Dales & Blanco Vida, 2009	Santiago, Chile	0.00212	0.00025	0.01440	0.00063	35.8	5.3	NA	3.3	NA	1998–2006	PM NA; EC lag 1
Percentage change per 1 µg/m^3 increase[e]		*Percentage*	*95% CI*	*Percentage*	*95% CI*							
Pooled fixed effects		0.17	(0.13–0.21)	1.45	(1.32–1.57)							
Pooled random effects		0.19	(0.03–0.35)	1.45	(1.32–1.57)							

[a] Mean or median.
[b] Coefficient of the correlation between $PM_{2.5}$ and EC concentrations.
[c] In cases where multiple lags are reported in Chapter 3, the estimate discussed by the author was used, as indicated in the APED as "selected lag".
[d] Age >65 years ; not all ages.
[e] In supplement B of Janssen et al. (2011), the percentage change was calculated per 10 µg/m^3.

Source: Janssen et al., 2011 (supplemental material, Tables C1–3).

Table 3C 2. Effects estimates for $PM_{2.5}$ and EC for cardiovascular mortality

Reference	Location	Estimate PM2.5		Estimate EC		IQR (μg/m^3)		Concentration[a]		Correlation (R) PM–EC[b]	Period	Selected lag[c]
		Beta	Standard error	Beta	Standard error	$PM_{2.5}$	EC	$PM_{2.5}$	EC			
Mar et al., 2000	Phoenix, United States	0.00685	0.00236	0.04400	0.01820	8.5	1.2	12.0	1.3	0.84	1995–1997	Lag 1
Ostro et al., 2007[d]	6 counties in California, United States	0.00105	0.00054	0.02574	0.01129	14.6	0.8	19.3	1.0	0.53	2000–2003	Lag 3
Cakmak, Dales & Blanco Vida, 2009	Santiago, Chile	0.00327	0.00037	0.01736	0.00097	35.8	5.3	NA	3.3	NA	1998–2006	PM NA; EC lag1
Percentage change per 1 μg/m^3 increase[e]		*Percentage*	*95% CI*	*Percentage*	*95% CI*							
Pooled fixed effects		0.26	(0.20–0.32)	1.76	(1.57–1.96)							
Pooled random effects		0.29	(0.07–0.50)	1.77	(1.08–3.08)							

[a] Mean or median.
[b] Coefficient of the correlation between $PM_{2.5}$ and EC concentrations.
[c] In cases where multiple lags are reported in Chapter 3, the estimate discussed by the author was used, as indicated in the APED as "selected lag".
[d] Age >65 years, not all ages.
[e] In supplement B of Janssen et al. (2011), the percentage change was calculated per 10 μg/m^3.

Table 3C 3. Effects estimates for $PM_{2.5}$ and EC on respiratory mortality

Reference	Location	Estimate $PM_{2.5}$		Estimate EC		IQR (μg/m^3)		Concentration[a]		Correlation (R) PM–EC[b]	Period	Selected lag[c]
		Beta	Standard error	Beta	Standard error	$PM_{2.5}$	EC	$PM_{2.5}$	EC			
Ostro et al., 2007[d]	6 counties in California, United States	0.00098	0.00103	-0.03298	0.02219	14.6	0.8	19.3	1.0	0.53	2000–2003	Lag 3
Cakmak, Dales & Blanco Vida, 2009	Santiago, Chile	0.00648	0.00058	0.03453	0.00146	35.8	5.3	NA	3.3	NA	1998–2006	PM NA; EC lag 2

[a] Mean or median.
[b] Coefficient of the correlation between $PM_{2.5}$ and EC concentrations.
[c] In cases where multiple lags are reported in Chapter 3, the estimate discussed by the author was used, as indicated in the APED as "selected lag".
[d] Age >65 years, not all ages.

Annex 3D. Study-specific effects estimates for hospital admissions and emergency department visits in studies that include both $PM_{2.5}$ and EC

Table 3D 1. Effects estimates for $PM_{2.5}$, EC and sulfate on hospital admissions and emergency department visits[a]

Reference	Location	Endpoint	$PM_{2.5}$		EC		Sulfate		IQR (μg/m^3)			Concentration (μg/m^3)		
			Beta	Standard error	Beta	Standard error	Beta	Standard error	$PM_{2.5}$	EC	Sulfate	$PM_{2.5}$	EC	Sulfate
Hospital admissions														
Zanobetti & Schwartz, 2006	Boston, United States; elderly people	Pneumonia Acute myocardial infarction	**0.0037** **0.0048**	0.0015 0.0021	**0.0540** **0.0391**	0.0159 0.0194			8.9	1.0		11.1	1.2	
Ostro et al., 2009	6 counties in California, United States; children	All respiratory diseases Asthma Bronchitis Pneumonia	**0.0027** 0.0023 **0.0034** 0.0022	0.0008 0.0015 0.0015 0.0014	**0.0640** 0.0628 0.0525 0.0623	0.0277 0.0339 0.0341 0.0476	**0.0199** 0.0026 **0.0449** 0.0182	0.0089 0.0199 0.0211 0.0224	14.6	0.8	1.5	19.4	1.0	2.0
Peng et al., 2009	119 counties in United States; elderly people	CVD Respiratory diseases	**0.00068** 0.00031	0.00021 0.00035	**0.01794** 0.00998	0.00375 0.00599	**0.00140** 0.00266	0.00075 0.00140	9.5	0.4	3.1	12.2	0.6	2.6
Emergency department visits														
Tolbert et al., 2007	Atlanta, United States; all ages[b]	CVD Respiratory diseases	0.00046 0.00046	0.00056 0.00047	**0.01295** -0.00349	0.00439 0.00313	-0.00026 0.00183	0.00161 0.00147	11.0	1.2	3.8	17.1	1.6	4.9
Cakmak et al., 2009[c]	Santiago, Chile; all ages	All non-accidental Respiratory diseases	**0.00152** **0.00241**	0.00018 0.00028	**0.02287** **0.03531**	0.00184 0.00247	**0.02232** **0.03185**	0.00804 0.01092	40.3	4.8	2.3	NA	2.8	2.6

[a] Significant effects (P<0.05) in bold.
[b] Also estimates from additional endpoints available from three older papers that included a shorter study period (Metzger et al., 2004; Sarnat et al., 2008; Tolbert et al., 2000).
[c] Sulfate estimated from sulfur.

Source: Janssen et al., 2011 (supplemental material, Table D1).

Annex 3E. Effects estimates for EC and other particle components

Table 3E 1. Effects estimates for EC and other particle components[a]

Reference	Location	Endpoint	Percentage increase per IQR							IQR (μg/m^3)						
			EC	OC	Sulfate	Nitrate	Zinc	Potassium	Silicon	EC	OC	Sulfate	Nitrate	Zinc	Potassium	Silicon
Mortality																
Mar et al., 2000[b]	Phoenix, United States	All causes	NS	NS	**-3.0**		NS	NS		1.2	3.0	0.8		NA	0.06	
		Cardiovascular	**5.2**	**4.4**	NS		NS	**3.2**								
Klemm et al., 2004	Atlanta, United States	All causes	1.5	1.3	3.4	-0.1				1.1	2.4	3.9	1.3			
Ostro et al., 2007	6 counties in California, United States	All causes	0.7	0.6	0.2	0.1	0.6	0.2	0.0	0.8	4.6	1.5	5.5	0.01	0.08	0.15
		Cardiovascular	**2.1**	1.6	0.6	1.5	**2.2**	**0.5**	0.6							
		Respiratory	-2.6	-2.9	1.1	1.0	-0.5	0.5	1.5							
Maynard et al., 2007	Boston, United States	All causes	**2.3**		**1.1**					0.2		2.3				
		Respiratory	**3.7**		2.1											
		Cardiovascular	1.5		-0.2											
		Stroke	4.4		2.0											
		Diabetes	5.7		2.9											
Cakmak, Dales & Blanco Vida, 2009[b]	Santiago, Chile	All causes	**7.9**	**6.6**	**3.2**		**5.3**	**3.5**	1.7	5.3	7.4	2.8		0.08	0.23	0.20
		Cardiac	**9.6**	**8.3**	**5.1**		**5.9**	**5.1**	**4.2**							
		Respiratory	**20.0**	**17.9**	**6.9**		**13.6**	**11.7**	**8.1**							
Hospital admission																
Ostro et al., 2009	6 counties in California, United States; children	All respiratory	**5.4**	**3.4**	**3.0**	**3.3**	1.6	0.8	**2.8**							
		Asthma	5.3	4.0	0.4	2.4	1.8	0.3	2.9	0.8	4.5	1.5	5.6	0.01	0.08	0.15
		Bronchitis	4.4	**4.8**	**6.9**	3.9	1.7	2.1	**6.1**							
		Pneumonia	5.3	**4.5**	2.8	2.2	**2.0**	0.7	4.3							
Peng et al., 2009	119 counties, United States; elderly people	Cardiovascular	**0.7**	**0.7**	0.4	**0.5**			**0.2**	0.4	3.2	3.1	1.6			0.07
		Respiratory	0.4	**0.8**	-0.3	0.0			0.1							
Emergency department visits																
Sarnat et al., 2008[c]	Atlanta, United States; all ages	CVD	**2.5**	**2.4**	0.7	0.2	**1.3**	**3.0**	0.8	NA	NA	NA	NA	NA	NA	NA
		Respiratory	-0.4	-0.3	**2.0**	-0.1	-0.3	0.2	-0.4							
Cakmak et al., 2009[d]	Santiago, Chile; all ages	All non-accidental	**11.5**	**9.3**	**5.2**		**5.2**	**5.8**	**5.8**	4.8	8.5	2.3		0.07	0.21	0.18
		Respiratory	**18.3**	**14.3**	**7.5**		**7.5**	**9.8**	**11.4**							

[a] Percentage increase per IQR (significant effects (P<0.05) in bold).
[b] NS=non-significant (effect estimates not reported in Chapter 3).
[c] Estimates from Sarnat et al. (2008) used instead of Tolbert et al. (2007), despite shorter period (four instead of six years), as the Sarnat paper included more elements.
[d] Sulfate estimated from sulfur.

Source: Janssen et al., 2011 (supplemental material, Table E1).

Table 3E 2. Results from single- and multipollutant models including BC and sulfate

Reference/location	Health endpoint	BCP metric	R Sulfate-BCP[a]	Percentage change in RR[b]			
				Sulfate single-pollutant	Sulfate multipollutant	BCP single-pollutant	BCP multipollutant
Hoek et al., 2000	Total mortality CVD mortality	BS	0.65	3.2 (0.6–5.9) 2.1 (-1.9–6.3)	2.7 (-0.3–5.8) 0.8 (-3.7–5.4)	2.8 (1.7–3.8) 3.2 (1.6–4.8)	1.2 (-1.5–4.1) 2.9 (-1.3–7.4)
Anderson et al., 2001; West Midlands, United Kingdom	Respiratory admissions	BS	0.30	0.8 (-1.3–2.9)	NA	2.1 (-0.1–4.2)	2.4 (0.1–4.7)
Maynard et al., 2007	Total mortality	BC	0.44	1.1 (0.01–2.0)	0.5 (-0.45–1.6)	2.3 (1.2–3.4)	2.2 (0.2–4.2)
Peng et al., 2009; 119 counties, United States[c]	Respiratory admissions Cardiovascular admissions	EC	0.18	-0.3 (-1.1–0.5) 0.4 (-0.0–0.9)	-0.6 (-1.1–0.3) 0.0 (-0.5–0.6)	0.4 (-0.1–0.9) 0.7 (0.4–1.0)	0.0 (-0.1–0.8) 0.8 (0.3–1.3)
Cakmak, Dales & Blanco Vida, 2009; Santiago, Chile[d]	Total mortality Cardiac mortality Respiratory mortality	EC	0.33	3.2 (1.4–5.0) 5.1 (2.4–8.0) 6.9 (1.9–12.1)	Lost significance	7.9 (7.2–8.6) 9.6 (8.5–10.8) 20.0 (18.2–21.9)	Remained significantly associated
Cakmak et al., 2009 Santiago, Chile[c]	All non-accidental admissions Respiratory admissions	EC	0.20	5.2 (1.5–9.1) 7.5 (2.4–12.8)	Lost significance	11.5 (9.6–13.5) 18.3 (15.6–21.2)	Remained significant

[a] Coefficient of the correlation between sulfate and BCP concentrations.
[b] RRs expressed as reported in Chapter 3: IQR for Maynard (2007); Peng (2009); Cakmak, Dales & Blanco Vida (2009) and Cakmak et al. (2009); 1st–199th percentile for Hoek (2000); 10–1990th percentile for Anderson (2001).
[c] Multipollutant estimates also adjusted for OC matter, nitrate, silicon, and sodium and ammonium ions.
[d] Multipollutant estimates also adjusted for 16 other PM components and 3 gases; quantitative estimates for multipollutant models requested from the authors but not received.

Source: Janssen et al., 2011 (supplemental material, Table E2).

Annex 3F. Study-specific estimates for asthma and cough in panel studies among asthmatic and symptomatic children

Table 3F 1. Effects estimates for asthma, lag 1

Reference	Location	Population (N, age, asthmatic/ symptomatic)	Year; duration	Estimate PM_{10}		Estimate BCP		Concentration[a]		Correlation (R) PM–BCP
				Beta	Standard error	Beta	Standard error	PM_{10}	BCP	
Single-location estimates										
Roemer, Hoek & Brunekreef, 1993	Rural, Netherlands	N=73; 6–12 years; symptomatic	1990; 3 months	0.033	0.010	0.382	0.127	NA	NA	0.81
Gielen et al., 1997	Amsterdam, Netherlands	N=61; 7–13 years; asthmatic	1995; 2.5 months	0.000	0.004	0.018	0.056	30.5	1.5[b]	NA
Segala et al., 1998	Paris, France	N=84; 7–15 years; asthmatic	1992; 6 months	0.005	0.008	0.055	0.073	34.2	3.5[b]	0.82
van der Zee et al., 1999	Urban, Netherlands	N=142; 7–11 years; symptomatic	1993–1995; 3 months	0.004	0.001	0.034	0.029	38	1.2[b]	NA
	Rural, Netherlands	N=178; 7–11 years; symptomatic	1993–1995; 3 months	0.000	0.001	0.005	0.023	31	0.9[b]	NA
Just et al., 2002	Paris, France	N=82; 7–15 years; asthmatic	1996; 3 months	0.006	0.028	0.196	0.155	23.5	1.8[b]	0.59
Delfino et al., 2003	Los Angeles, United States	N=22; 10–16 years; asthmatic	1999; 3 months	0.002	0.007	0.003	0.075	59.9	5.1	0.82
Gent et al., 2009	New Haven, United States	N=149; 4–12 years; asthmatic	2000–2004; 12 months	-0.0020	0.0020	0.010	0.026	17.0	1.9	NA
Multicentre studies										
Roemer et al., 1998	PEACE study, Europe; 14 urban and 14 rural panels	N=2010; 6–12 years; symptomatic	1993–1994; 2 months	0.0000	0.0005	-0.008	0.006	11.2–1998.8	0.5–12.0[b]	
Percentage change per 1 µg/m^3 increase; single-location estimates				*Percentage*	*95% CI*	*Percentage*	*95% CI*			
Pooled fixed effects				0.13	(0.00–0.26)	2.11	(-0.58–4.86)			
Pooled random effects				0.19	(-0.13–0.51)	2.85	(-1.01–6.86)			
Heterogeneity chi-squared (df=7)				Q=18.0	P=0.012	Q=10.5	P=0.16			
Percentage change per 1 µg/m^3 increase; including PEACE study										
Pooled fixed effects				0.05	(-0.03–0.12)	-0.32	(-1.36–0.73)			
Pooled random effects				0.12	(-0.09–0.32)	1.53	(-1.39–4.53)			
Heterogeneity chi-squared (df=8)				Q=20.3	P=0.009	Q=14.2	P=0.077			

[a] Mean or median (µg/m^3).
[b] Derived from BS as 10 BS=1.1 EC.

Table 3F 2. Effects estimates for asthma, lag 0

Reference	Location	Population (N, age, asthmatic/ symptomatic)	Year; duration	Estimate PM_{10}		Estimate BCP		Concentration[a]		Correlation (R) PM–BCP
				Beta	Standard error	Beta	Standard error	PM_{10}	BCP	
Single-location estimates										
Roemer, Hoek & Brunekreef, 1993	Rural, Netherlands	N=73; 6–12 years; symptomatic	1990; 3 months	0.027	0.010	0.273	0.145	NA	NA	0.81
Gielen et al., 1997	Amsterdam, Netherlands	N=61; 7–13 years; asthmatic	1995; 2.5 months	0.005	0.003	0.005	0.055	30.5	1.5[b]	NA
Segala et al., 1998	Paris, France	N=84; 7–15 years; asthmatic	1992; 6 months	0.013	0.008	0.082	0.064	34.2	3.5[b]	0.82
van der Zee et al., 1999	Urban, Netherlands	N=142; 7–11 years; symptomatic	1993–1995; 3 months	0.003	0.001	0.036	0.032	38	1.2[b]	NA
	Rural, Netherlands	N=178; 7–11 years; symptomatic	1993–1995; 3 months	0.000	0.001	-0.009	0.026	31	0.9[b]	NA
Just et al., 2002	Paris, France	N=82; 7–15 years; asthmatic	1996; 3 months	0.006	0.028	0.196	0.155	23.5	1.8[b]	0.59
Delfino et al., 2003	Los Angeles, United States	N=22; 10–16 years; asthmatic	1999; 3 months	0.010	0.004	0.211	0.089	59.9	5.1	0.82
Gent et al., 2009	New Haven, United States	N=149; 4–12 years; asthmatic	2000–2004; 12 months	0.0001	0.0004	0.039	0.019	17.0	1.9	NA
Multicentre studies										
Roemer et al., 1998	PEACE study, Europe; 14 urban and 14 rural panels	N=2010; 6–12 years; symptomatic	1993–1994; 2 months	-0.0007	0.0004	-0.007	0.004	11.2–1998.8	0.5–12.0[b]	
Percentage change per 1 µg/m^3 increase; single-location estimates				*Percentage*	*95% CI*	*Percentage*	*95% CI*			
Pooled fixed effects				0.05	(-0.02–0.12)	3.40	(0.81–6.05)			
Pooled random effects				0.27	(0.03–0.51)	4.27	(0.19–8.52)			
Heterogeneity chi-squared (df=7)				Q=22.4	P=0.002	Q=11.4	P=0.123			
Percentage change per 1 µg/m^3 increase; including PEACE study										
Pooled fixed effects				0.00	(-0.05–0.06)	-0.32	(-1.05–0.41)			
Pooled random effects				0.13	(-0.03–0.29)	2.89	(-0.56–6.46)			
Heterogeneity chi-squared (df=8)				Q=27.2	P=0.001	Q=20.2	P=0.010			

[a] Mean or median (µg/m^3).
[b] Derived from BS as 10 BS=1.1 EC.

Table 3F 3. Effects estimates for cough, lag 1

Reference	Location	Population (N, age, asthmatic/ symptomatic)	Year(s), duration	Estimate PM_{10}		Estimate BCP		Concentration[a]		Correlation (R) PM–BCP
				Beta	Standard error	Beta	Standard error	PM_{10}	BCP	
Single-location estimates										
Roemer, Hoek & Brunekreef, 1993	Rural, Netherlands	N=73; 6–12 years; symptomatic	1990; 3 months	0.005	0.018	0.182	0.227	NA	NA	0.81
Gielen et al., 1997	Amsterdam, Netherlands	N=61; 7–13 years; asthmatic	1995; 2.5 months	0.004	0.002	-0.008	0.043	30.5	1.5[b]	NA
Tiitanen et al., 1999	Kuopio, Finland	N=49; 8–13 years; symptomatic	1995; 6 weeks	-0.001	0.002	0.054	0.082	28	0.8	0.74
van der Zee et al., 1999	Urban, Netherlands	N=142; 7–11 years; symptomatic	1993–1995; 3 months	-0.001	0.001	-0.016	0.017	38	1.2[b]	NA
	Rural, Netherlands	N=178; 7–11 years; symptomatic	1993–1995; 3 months	0.001	0.001	0.024	0.012	31	0.9[b]	NA
Just et al., 2002	Paris, France	N=82; 7–15 years; asthmatic	1996; 3 months	0.010	0.011	0.181	0.097	23.5	1.8[b]	0.59
Gent et al., 2009	New Haven, United States	N=149; 4–12 years; asthmatic	2000–2004; 12 months	0.0001	0.0010	0.010	0.011	17.0	1.9	Na
Multicentre studies										
Roemer et al., 1998	PEACE study, Europe; 14 urban and 14 rural panels	N=2010; 6–12 years; symptomatic	1993–1994; 2 months	-0.0003	0.0003	0.002	0.003	11.2–1998.8	0.5–12.0[b]	
Percentage change per 1 µg/m^3 increase; single-location estimates				*Percentage*	*95% CI*	*Percentage*	*95% CI*			
Pooled fixed effects				0.04	(-0.03–0.12)	1.15	(-0.28–2.66)			
Pooled random effects				0.04	(-0.03–0.12)	1.07	(-1.01–3.04)			
Heterogeneity chi-squared (df=6)				Q=5.8	P=0.44	Q=7.8	P=0.25			
Percentage change per 1 µg/m^3 increase; including PEACE study										
Pooled fixed effects				-0.00	(-0.05–0.04)	0.34	(-0.24–0.91)			
Pooled random effects				0.01	(-0.06–0.07)	0.64	(-0.56–1.86)			
Heterogeneity chi-squared (df=7)				Q=8.4	P=0.30	Q=9.3	P=0.23			

[a] Mean or median (µg/m^3).
[b] Derived from BS as 10 B=1.1 EC.

Table 3F 4. Effects estimates for cough, lag 0

Reference	Location	Population (N, age, asthmatic/ symptomatic)	Year(s), duration	Estimate PM_{10}		Estimate BCP		Concentration[a]		Correlation (R) PM–BCP
				Beta	Standard error	Beta	Standard error	PM_{10}	BCP	
Single-location estimates										
Roemer, Hoek & Brunekreef, 1993	Rural, Netherlands	N=73; 6–12 years; symptomatic	1990; 3 months	0.000	0.019	0.127	0.245	NA	NA	0.81
Gielen et al., 1997	Amsterdam, Netherlands	N=61; 7–13 years; asthmatic	1995; 2.5 months	-0.001	0.003	-0.003	0.055	30.5	1.5[b]	NA
Tiitanen et al., 1999	Kuopio, Finland	N=49; 8–13 years; symptomatic	1995; 6 weeks	0.000	0.002	-0.018	0.082	28	0.8	0.74
van der Zee et al., 1999	Urban, Netherlands	N=142; 7–11 years; symptomatic	1993–1995; 3 months	0.000	0.001	0.024	0.018	38	1.2[b]	NA
	Rural, Netherlands	N=178; 7–11 years; symptomatic	1993–1995; 3 months	0.001	0.001	0.009	0.015	31	0.9[b]	NA
Just et al., 2002	Paris, France	N=82; 7–15 years; asthmatic	1996; 3 months	0.010	0.011	0.181	0.097	23.5	1.8[b]	0.59
Gent et al., 2009	New Haven, United States	N=149; 4–12 years; asthmatic	2000–2004; 12 months	0.0001	0.0004	0.010	0.012	17.0	1.9	NA
Multicentre studies										
Roemer et al., 1998	PEACE study, Europe; 14 urban and 14 rural panels	N=2010; 6–12 years; symptomatic	1993–1994; 2 months	-0.0004	0.0002	-0.004	0.002	11.2–1998.8	0.5–12.0[b]	
Percentage change per 1 µg/m^3 increase; single-location estimates				*Percentage*	*95% CI*	*Percentage*	*95% CI*			
Pooled fixed effects				0.03	(-0.02–0.09)	1.33	(-0.28–2.96)			
Pooled random effects				0.03	(-0.02–0.09)	1.33	(-0.28–2.96)			
Heterogeneity chi-squared (df=6)				Q=1.6	P=0.26	Q=3.9	P=0.69			
Percentage change per 1 µg/m^3 increase; including PEACE study										
Pooled fixed effects				-0.02	(-0.05–0.01)	-0.26	(-0.67–0.15)			
Pooled random effects				-0.02	(-0.05–0.01)	0.17	(-0.79–1.15)			
Heterogeneity chi-squared (df=7)				Q=6.5	P=0.48	Q=7.9	P=0.34			

[a] Mean or median (µg/m^3).
[b] Derived from BS as 10 BS=1.1 EC.

Annex 3G. Effects of $PM_{2.5}$ and BC in cohort studies of respiratory health in children

Table 3G 1. Effects of $PM_{2.5}$ and BCP in birth cohort studies

Reference	Cohort	R PM–BCP[a]	RR expressed per IQR	Health endpoint[b]	RR PM	RR BCP
Gehring et al., 2002	Birth cohort (GINI/LISA); 1756 children born in the Munich metropolitan area, Germany; age 2 years	0.96	$PM_{2.5}$: 1.5 µg/m^3 Abs: 0.4 m^{-1} ×10^{-5}	Wheeze	0.96 (0.83–1.12)	0.98 (0.84–1.14)
				Dry cough at night	**1.20** (1.02–1.42)	1.16 (0.98–1.37)
				DD[b] asthmoid/spastic/obstructive bronchitis	0.92 (0.78–1.09)	0.94 (0.79–1.12)
				Respiratory infections	0.98 (0.80–1.20)	0.99 (0.80–1.22)
				Sneezing/runny stuffed nose	0.96 (0.82–1.12)	0.92 (0.78–1.09)
Brauer et al., 2002	Piama cohort; 3000 children throughout the Netherlands; symptoms at age 2 years	0.99	$PM_{2.5}$: 3.2 µg/m^3 Abs: 0.54 m^{-1} ×10^{-5}	Wheeze	1.14 (0.98–1.34)	1.11 (0.97–1.26)
				DD asthma	1.12 (0.84–1.50)	1.12 (0.88–1.43)
				Dry cough at night	1.04 (0.88–1.23)	1.02 (0.88–1.17)
				DD bronchitis	1.04 (0.85–1.26)	0.99 (0.84–1.17)
				Ear, nose, throat infections	**1.20** (1.01–1.42)	**1.15** (1.00–1.33)
				DD influenza/serious colds	**1.12** (1.00–1.27)	1.09 (0.98–1.21)
				Itchy rash	1.01 (0.88–1.16)	1.02 (0.91–1.15)
				DD eczema	0.95 (0.83–1.10)	0.96 (0.85–1.08)
Brauer et al., 2006	Birth cohort (Piama); 3000 children throughout the Netherlands	0.99	$PM_{2.5}$: 3 µg/m^3 EC: 0.5 µg/m^3	Otitis media at age:		
				1 year	1.13 (0.98– 1.32)	1.11 (0.98–1.26)
				2 years	**1.13** (1.00–1.27)	**1.10** (1.00–1.22
	Birth cohort (LISA); 600 children from Munich, Germany		$PM_{2.5}$: 3 µg/m^3 EC: 0.5 µg/m^3	Otitis media at age:		
				1 year	1.19 (0.73– 1.92)	1.12 (0.83–1.51)
				2 years	1.24 (0.84–1.83)	1.10 (0.86–1.41)
Brauer et al., 2007	PIAMA cohort; 3000 children throughout the Netherlands; symptoms at age 4 years	0.99	$PM_{2.5}$: 3.3 µg/m^3 Abs: 0.58 m^{-1} ×10^{-5}	Wheeze	1.20 (0.99–1.46)	**1.18** (1.00–1.40)
				DD asthma	1.32 (0.98–1.71)	1.30 (0.98–1.71)
				Dry cough at night	1.14 (0.98–1.33)	**1.14** (1.00–1.31)
				DD bronchitis	0.86 (0.66–1.11)	0.88 (0.69–1.11)
				Ear, nose, throat infections	**1.17** (1.02–1.34)	**1.16** (1.03–1.31)
				DD influenza/serious colds	**1.25** (1.07–1.46)	**1.19** (1.04–1.37)
				Itchy rash	0.98 (0.85–1.14)	0.97 (0.85–1.10)
				DD eczema	0.98 (0.82–1.17)	0.97 (0.83–1.14)
Morgenstern et al., 2007[c]	GINI/LISA cohort; 3577 children living in the Munich metropolitan area, Germany; age 2 years	0.49	$PM_{2.5}$: 1.0 µg/m^3 Abs: 0.22 m^{-1} ×10^{-5}	Wheeze	1.10 (0.96–1.25)	1.09 (0.90–1.33)
				Dry cough at night	1.03 (0.89–1.19)	1.18 (0.93–1.50)
				DD asthmoid/spastic/obstructive bronchitis	1.05 (0.92–1.20)	0.85 (0.31–2.34)
				Respiratory infections	1.09 (0.94–1.27)	1.05 (0.79–1.39)
				Sneezing/runny stuffed nose	**1.19** (1.04–1.36)	**1.27** (1.04–1.56)

Reference	Cohort	R PM–BCP[a]	RR expressed per IQR	Health endpoint[b]	RR PM	RR BCP
Morgenstern et al., 2008[c]	GINI/LISA cohort; ±3000 children living in the Munich metropolitan area, Germany; ages 4 and 6 years	0.49	$PM_{2.5}$: 1.0 µg/m^3 Abs: 0.22 m^{-1} ×10^{-5}	DD asthmoid/spastic/obstructive bronchitis	1.12 (0.94–1.29)	**1.56** (1.03–2.37)
				DD hayfever	1.01 (0.91–1.12)	**1.59** (1.11–2.27)
				DD eczema	1.00 (0.86–1.24)	1.03 (0.86–1.24)
				PR[b] asthmoid/spastic/obstructive bronchitis	0.97 (0.91–1.02)	0.96 (0.83–1.11)
				DD hayfever	1.02 (0.96–1.08)	1.11 (0.93–1.31)
				DD eczema	1.05 (0.90–1.37)	1.05 (0.93–1.47)
				Prevalent asthma	**1.26** (1.04–1.51)	**1.20** (1.02–1.42)
				Incident asthma	**1.28** (1.10–1.49)	**1.21** (1.06–1.38)
				Asthma symptoms	**1.15** (1.02–1.28)	**1.12** (1.01–1.24)
				Wheeze	**1.20** (1.08–1.33)	**1.16** (1.06–1.27)
				Sneezing, runny/blocked nose	**1.12** (1.01–1.24)	**1.11** (1.01–1.21)
				Hayfever	1.05 (0.83–1.32)	1.04 (0.85–1.27)
				Atopic eczema	1.00 (0.90–1.11)	1.00 (0.91–1.10)
Gehring et al., 2010	GINI/LISA cohort; ±3000 children living in the Munich metropolitan area, Germany; age 8 years			Wheeze	**1.29** (1.04–1.62)	**1.22** (1.00–1.48)
				Bronchial hyperactivity	0.98 (0.76–1.24)	1.04 (0.84–1.29)
				Allergic sensitization:	1.16 (0.96–1.39)	1.12 (0.95–1.32)
				– in utero exposure	**1.02** (1.00–1.03)	**1.08** (1.02–1.15)
				– first year exposure	1.01 (0.99–1.03)	**1.14** (1.01–1.29)

[a] Coefficient of the correlation between $PM_{2.5}$ and BCP concentrations.
[b] DD=diagnosed by doctor; PR=parental report.
[c] Further analyses by Gehring et al. (2002). Here, the study population was expanded by including subjects who lived outside the Munich area. Although this resulted in a lower correlation between $PM_{2.5}$ and BCP (R=0.49), the performance of the land use regression model used to assign exposure to individual participants was poorer than that of the smaller population (Morgenstern et al., 2007).

Source: Janssen et al., 2011 (supplemental material, Tables F1, F2).

Table 3G 2. Effects of $PM_{2.5}$ and BCP in cohort studies on lung function growth

Reference	Cohort	R PM–BCP[a]	RR expressed for concentration range (maximum–minimum)	Health endpoint	RR PM	RR BCP
Gauderman et al., 2002	Results from 2 cohorts: (1) 1457 children recruited in 1993; 4-year follow-up	0.91	$PM_{2.5}$: 22.2 µg/m^3 EC: 1.1 µg/m^3	Growth rate FVC (%) Growth rate FEV1 (%) Growth rate MMEF (%)	-0.42 (-0.86 – 0.03) -0.63 (-1.28 – 0.02) -0.94 (-1.88 – 0.01)	**-0.49** (-0.88 – -0.09) **-0.71** (-1.30 – -0.12) **-1.07** (-1.94 – -0.19)
	(2) 1678 children recruited in 1996; 4-year follow-up	0.93		Growth rate FVC (%) Growth rate FEV1 (%) Growth rate MMEF (%)	-0.14 (-0.67 – 0.40) -0.39 (-1.06 – 0.28) **-0.94** (-1.87 – 0.00)	-0.17 (-0.67 – 0.33) -0.40 (-1.02 – 0.23) **-0.92** (-1.78 – -0.05)
Gauderman et al., 2004	Cohort (1); 8-year follow-up	0.91	$PM_{2.5}$: 22.8 µg/m^3 EC: 1.1 µg/m^3	Growth rate FVC (ml) Growth rate FEV1 (ml) Growth rate MMEF (ml)	-60.1 (-166.1 – 45.9) **-79.7** (-153.0 – -6.4) -168.9 (-345.5 – 7.8)	-77.7 (-166.7 – 11.3) **-87.9** (-146.4 – -29.4) **-165.5** (-323.4 – -7.6)

[a] Coefficient of the correlation between $PM_{2.5}$ and BCP concentrations.

Annex 3H. Comparison of calculated health benefits of traffic abatement measures using $PM_{2.5}$ or BCP

As an illustration of the potential implications of using BCP as an air quality indicator, the health benefits of a traffic abatement measure for the population living along busy roads were calculated using $PM_{2.5}$ mass and BCP. Given the relatively large proportion of BCP in the roadside increment of $PM_{2.5}$ mass, it can be expected that traffic abatement measures will result in larger reductions in BCP, relative to reductions in PM mass. There are, however, few empirical data to support larger impacts on BCP than on $PM_{2.5}$ mass. In an evaluation of the effects on air quality of retrofitting trucks in southern California with diesel engine particle filters, Millstein & Harley (2010), using an Eulerian photochemical air quality model, estimated a decrease in EC concentrations in 2014 of 12–14%. The estimated effect on $PM_{2.5}$ mass concentrations was much smaller (<1%). In a modelling study of the effect of a speed limit reduction (from 120 to 90 km/hour) on air quality in Flanders, the EC concentrations decreased up to 30% just next to the busiest highways, compared to an estimated reduction of at most 8.5% for $PM_{2.5}$. For buffer zones of 0–100m distance from the highways, EC concentrations decreased by 9–10% (Lefebvre et al., 2011). A small monitoring study of the effects of road closures associated with the 2004 Democratic National Convention in Boston suggested slightly lower concentrations of EC and NO_2 during the road closure periods at monitoring sites proximate to the closed highway segments. This decrease was not observed for $PM_{2.5}$ or further from major highways (Levy, Baxter & Clougherty, 2006).

Based on a review of studies that have looked at roadside increments of PM mass compared to BCP, it is estimated that the percentage of EC in the roadside increment in $PM_{2.5}$ will be on average 55% (Table 3H 1). This result is in line with two modelling studies that estimated the percentage of EC in traffic emissions mentioned above (Millstein & Harley, 2010; Lefebvre et al., 2011) and also in the range provided by the European emission inventory database COPERT4 for the EC fraction in $PM_{2.5}$ in exhaust emissions for different vehicle categories (such as passenger cars, vans and trucks) (Ntziachristos & Samaras, 2009). The estimated percentage of EC in the roadside increment in $PM_{2.5}$ of 0.4–0.7 $\mu g/m^3$ implies that every 1 $\mu g/m^3$ reduction in traffic-related $PM_{2.5}$ will result in a 0.4–0.7 $\mu g/m^3$ reduction in EC. This range was used to estimate the health benefits of a hypothetical traffic abatement policy measure resulting in a 1 $\mu g/m^3$ reduction in $PM_{2.5}$ mass. This approach assumes that the reduction in BCP resulting from traffic abatement will be proportional to the decrease in PM mass by the percentage of EC in the roadside increment for PM mass, an assumption that will not hold for all policies. A comparison of the effect on life expectancy of the population living along major roads, using the calculated RRs for $PM_{2.5}$ mass and EC (Table 4), is given in Table 3H 2. When the average conversion factor of 10 $\mu g/m^3$ BS=1.1 $\mu g/m^3$ EC is used to derive the RR for a 1 $\mu g/m^3$ increase in EC (Table 4) and the percentage of EC in a roadside increment of $PM_{2.5}$ (Table 3H 1), the increase in life expectancy per person is five times higher for EC compared to $PM_{2.5}$ (3.6 months compared with 21 days, Table 3H 2). When the maximum and minimum conversion factors of 1.8 and 0.5 $\mu g/m^3$ EC per 10 $\mu g/m^3$ BS are used, the increase in estimated life expectancy is four to nine times higher. The estimated health benefits are, therefore, much greater when expressed in EC compared to an equivalent change in PM mass.

Table 3H 1. Estimated percentage of EC in roadside increment in $PM_{2.5}$

Reference	Location, period	Measurement method of BCP	Difference traffic–background (µg/m³)		Percentage of EC in roadside increment		
			PM	BCP			
Kinney et al., 2000	New York, United States; sidewalk; 1996	EC; sunset	4.4	2.6	58		
Funasaka et al., 2000	Osaka, Japan; outside homes; period N/A	EC	6.0	3.6	60		
Janssen et al., 2001; 2008	Netherlands; 50 m of highway; 1997/1998	EC from Abs;[a] VDI 2465	2.1	2.0	76[b]		
Lena et al., 2002	New York, United States; sidewalk; 1999	EC from Abs;[a] sunset	6.2	3.1	50		
Cyrys et al., 2003	Munich, Germany; 1999/2000	EC; VDI 2465	1.0	1.0	80[b]		
	Netherlands; 1999/2000	EC; VDI 2465	2.1	1.8	69[b]		
	Sweden; 1999 /2000	EC; VDI 2465	3.6	1.1	24[b]		
Harrison, Jones & Lawrence, 2004	London & Birmingham, United Kingdom; roadside; 2000/2002	EC	8.4	6.1	69		
					10 µg/m³ BS = 1.1µg/m³ EC	*10 µg/m³ BS = 0.5µg/m³ EC*	*10 µg/m³ BS = 1.8µg/m³ EC*
Janssen et al., 1997	Arnhem, Netherlands; curbside; October–November 1994	BS	7.9	28.3	39	18	64
Roorda-Knape et al., 1998	Netherlands; 15–32 m of highways; May–August 1995	BSBS	1.4	5.5	43	20	71
Roemer & van Wijnen, 2001b	Amsterdam, Netherlands; 7 m of busy street; 1998/1999	BS	2.0	11.0	61	28	99
Fischer et al., 2000	Amsterdam, Netherlands; outside homes; 1995	Abs of $PM_{2.5}$ filters[c]	3.0	1.3	47	22	77
Smargiassi et al., 2005	Montreal, Canada; curbside or on collector	Abs of $PM_{2.5}$ filters[c]	1.8	0.7	42	19	68
Janssen et al., 2008	Netherlands; 50 m of highway; 2001/2002	Abs of $PM_{2.5}$ filters[c]	2.4	0.9	43	19	70
Janssen et al., 2008	Munich, Germany; along highway; 2002	Abs of $PM_{2.5}$ filters[c]	3.6	1.2	38	17	62
Boogaard et al., 2010	Netherlands; 9–15 m of busy roads in large cities; 2008/2009	Abs of $PM_{2.5}$ filters[c]	2.2	1.6	77	35	127
				Average[d]	**55**	**41**	**70**
				95% CI	**46–63**	**29–54**	**59–82**

[a] Calculated using a study-specific calibration derived from co-located samples (see Table 3A 1).
[b] Results from studies that have used the VDI protocol were divided by 1.25, as this method has been shown to overestimate EC by, on average, 25% (Schmid et al., 2001).
[c] An increase in 1 unit of Abs is considered to equal an increase of 10 µg/m³ BS, according to Roorda-Knape et al (1998).
[d] Average includes all studies; average of studies that directly measured EC was 61%.

Source: Janssen et al., 2011 (supplemental material, Table G2).

Table 3H 2. Comparison of the estimated effect on life expectancy of reductions in $PM_{2.5}$ mass and EC resulting from a traffic management plan

Component	Conversion BS to EC[a]	RR[b]	Reduction (μg/m^3)[c]	Increase in life expectancy per person[d]
$PM_{2.5}$		1.007	1.00	21 days
EC	10 μg/m^3 BS=1.1 μg/m^3 EC	1.06	0.55 (0.46–0.63)	3.6 months (3.0–4.1 months)
	10 μg/m^3 BS=1.8 μg/m^3 EC	1.04	0.70 (0.59–0.82)	3.1 months (2.6–3.6 months)
	10 μg/m^3 BS=0.5 μg/m^3 EC	1.10	0.41 (0.29–0.54)	4.5 months (3.2–5.9 months)

[a] BS was converted to EC for 2 of the 4 studies that were used to calculate the RR for EC, and for 8 of 16 studies that were used to calculate the percentage EC in the roadside increment of $PM_{2.5}$ over background.
[b] RR per 1 μg/m^3.
[c] A traffic abatement measure is evaluated that reduces EC proportional to the percentage EC in the roadside increment of $PM_{2.5}$ over background. Values in brackets for the reduction correspond to the 95% CI of the percentage EC in the roadside increment of $PM_{2.5}$ (Janssen et al., 2011, supplemental material, Table G2).
[d] Values in brackets for the increase in life expectancy are based on the 95% CI of the reduction.

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Black carbon (BC) is an indicator of combustion-related air pollution and was recently recognized as one of the short-lived climate-forcers. This report presents the results of a systematic review of evidence of the health effects of BC in ambient air. It concludes that epidemiological studies provide sufficient evidence of the association of cardiopulmonary morbidity and mortality with BC exposure. The review of the toxicological studies suggested that BC may not be a major directly toxic component of fine particulate matter ($PM_{2.5}$), but it may operate as a universal carrier of a wide variety of chemicals of varying toxicity to the human body. A reduction in exposure to $PM_{2.5}$ containing BC and other combustion-related particulate material for which BC is an indirect indicator should lead to a reduction in the health effects associated with PM and simultaneously contribute to the mitigation of climate change.

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