Chapter 3

Black Carbon Effects on Public Health and the Environment

3.1 Summary of Key Messages

- BC is a component of both fine and coarse particulate matter (PM), though because of its small size, it is most strongly associated with the fine particle ($PM_{2.5}$) fraction. Most of the literature evaluating the potential impacts of BC on human health (and the health benefits of BC mitigation) has focused on BC as part of $PM_{2.5}$.

- Short-term and long-term exposures to $PM_{2.5}$ are associated with a broad range of adverse human health effects including respiratory and cardiovascular effects, as well as premature death.

- Over the past decade, the scientific community has focused increasingly on trying to identify the health impacts of particular $PM_{2.5}$ constituents, such as BC. However, EPA has determined that there is insufficient information at present to differentiate the health effects of the various constituents of $PM_{2.5}$; thus, EPA assumes that many constituents are associated with adverse health impacts.

  - The limited scientific evidence that is currently available about the health effects of BC is generally consistent with the general $PM_{2.5}$ health literature, with the most consistent evidence for cardiovascular effects. However, study results for BC are variable, and further research is needed to address remaining uncertainties.

- $PM_{2.5}$, both ambient and indoor, is estimated to result in millions of premature deaths worldwide, the majority of which occur in developing countries.

  - The World Health Organization (WHO) estimates that indoor smoke from solid fuels is among the top ten major risk factors globally, contributing to approximately 2 million deaths annually. Women and children are particularly at risk.

  - Ambient air pollution is also a significant health threat: according to the WHO, urban air pollution is among the top ten risk factors in medium- and high-income countries. Urban air pollution is not ranked in the top ten major risk factors in low-income countries since other risk factors (e.g. childhood underweight and unsafe water, sanitation and hygiene) are so substantial; however, a much larger portion of the total deaths related to ambient $PM_{2.5}$ globally are expected to occur in developing regions, partly due to the size of exposed populations in those regions. It is noteworthy that emissions and ambient concentrations of directly emitted $PM_{2.5}$ are often highest in urban areas, where large numbers of people live.

- $PM_{2.5}$, including BC, is linked to adverse impacts on ecosystems, to visibility impairment, to reduced agricultural production in some parts of the world, and to materials soiling and damage.

3.2 Introduction

This chapter assesses the current scientific knowledge relating to the public health and non-climate welfare effects associated with short-term and long-term exposure to BC. The magnitude of these impacts in the U.S. and globally is also addressed.

3.3 Health Effects Associated with BC

3.3.1 Key Health Endpoints Associated with Exposure to PM

BC is a component of both fine and coarse PM. Since 1997, EPA has recognized the need to regulate fine and coarse-fraction particles separately. Current national ambient air quality standards (NAAQS) use $PM_{2.5}$ as the indicator for fine particles, and PM_{10} as the indicator for thoracic coarse particles. At present, EPA is undertaking another periodic review of these standards. As part of this review, EPA has completed an Integrated Science Assessment for Particulate Matter (ISA) (U.S. EPA, 2009b) providing a concise evaluation and integration of the policy-relevant

Table 3-1. Summary of Causal Determinations for Exposure to $PM_{2.5}$ from 2009 PM ISA. (Source: U.S. EPA)

Exposure	Outcome	Causality Determination
Short-term exposure to $PM_{2.5}$	Cardiovascular Effects	Causal
	Respiratory Effects	Likely to be causal
	Mortality	Causal
Long-term exposure to $PM_{2.5}$	Cardiovascular Effects	Causal
	Respiratory Effects	Likely to be causal
	Mortality	Causal
	Reproductive and Developmental Effects	Suggestive
	Cancer, Mutagenicity, and Genotoxicity	Suggestive

science pertaining to the health and environmental effects of ambient particles. The ISA presents causal determinations by PM size fraction and exposure duration (i.e., short-term [days to weeks] or long-term [months to years]) for the health effects for which sufficient evidence was available to conclude a causal, likely to be causal, or suggestive relationship (Table 3-1). The discussion below is focused on the health effects with the strongest weight of evidence (i.e., cardiovascular effects, respiratory effects, and mortality) and conclusions drawn for these effects in the ISA. A more limited subset of studies has evaluated reproductive and developmental outcomes and cancer effects, but the weight of evidence for these effects is less substantial.[1]

A large body of scientific evidence links exposures to fine particles (i.e., ambient $PM_{2.5}$ mass concentrations) to an array of adverse effects, including premature mortality, increased hospital admissions and emergency department visits for cardiovascular and respiratory diseases, and development of chronic respiratory disease (U.S. EPA, 2009b). Recent evidence provides a greater understanding of the underlying mechanisms for $PM_{2.5}$-induced cardiovascular and respiratory effects for both short- and long-term exposures, providing biological plausibility for the effects observed in epidemiological studies. This evidence links exposure to $PM_{2.5}$ with cardiovascular outcomes that include the continuum of effects ranging from more subtle subclinical measures (e.g., changes in blood pressure, heart rate variability) to premature mortality. These health effects may occur over the full range of $PM_{2.5}$ concentrations observed in the long- and short-term epidemiological studies and EPA has concluded that no discernible threshold for any effects can be identified based on the currently available evidence.

In reviewing the studies regarding health effects of $PM_{2.5}$, EPA has recognized that it is highly plausible that the chemical composition of PM would be a better predictor of health effects than particle size alone (U.S. EPA, 2009b, 6-202). Differences in ambient concentrations of $PM_{2.5}$ constituents observed in different geographical regions as well as regional differences in $PM_{2.5}$-related health effects reported in a number of epidemiological studies are consistent with this hypothesis (U.S. EPA, 2009b, Section 6.6) . Over the past decade, the scientific community has focused increasingly on trying to identify the health impacts of particular $PM_{2.5}$ constituents or groups of constituents associated with specific source categories of fine particles. The growing body of evidence for the health impacts of specific $PM_{2.5}$ constituents includes evidence of effects associated with exposure to BC. However, the ISA concludes that the currently available scientific information continues to provide evidence that many different constituents of the fine particle mixture, as well as groups of constituents associated with specific source categories of fine particles, are linked to adverse health effects. While there is "some evidence for trends and patterns that link specific $PM_{2.5}$ constituents or sources with specific health outcomes... there is insufficient evidence to determine if these patterns are consistent or robust" (U.S. EPA, 2009b, 6-210). Consequently, research and data collection activities focused on particle composition could improve our understanding of the relative toxicity of different fine particle constituents or groups of constituents associated with specific sources of fine particles to inform future regulatory activities and benefits assessments.

The body of scientific evidence linking exposures to coarse particles (i.e., ambient $PM_{10-2.5}$ mass

[1] See Sections 7.4 and 7.5 of the PM ISA for an in-depth characterization of the evidence for an association between $PM_{2.5}$ and reproductive and developmental effects and cancer, respectively. (*http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546*)

concentrations) to health effects is much smaller than the body of evidence for $PM_{2.5}$ (U.S. EPA, 2009b). Similar to $PM_{2.5}$, the chemical composition of $PM_{10-2.5}$ can vary considerably by location, but city-specific speciated $PM_{10-2.5}$ data are limited. However, $PM_{10-2.5}$ may contain iron, silica, aluminum, and base cations from soil, plant and insect fragments, pollen, fungal spores, bacteria, and viruses, as well as fly ash, brake lining particles, debris, and automobile tire fragments. The last four of these components (fly ash, brake lining particles, debris, and automobile tire fragments) are associated with urban or industrial ambient mixes of coarse PM, which are often coated with BC (See Figure 2-3). Urban or industrial ambient mixes of coarse PM are dominated by high density vehicular, industrial, and construction emissions, and are likely to be associated with adverse health effects (U.S. EPA, 2006b). While there are no studies that specifically examine the association between BC as a component of $PM_{10-2.5}$ and health effects, the current evidence, primarily from epidemiological studies, indicates that short-term exposure to $PM_{10-2.5}$ is associated with effects on both the cardiovascular and respiratory systems. However, variability in the chemical and biological composition of $PM_{10-2.5}$, limited evidence regarding effects of the various components of $PM_{10-2.5}$, and lack of clearly defined biological mechanisms for $PM_{10-2.5}$-related effects are important sources of uncertainty (U.S. EPA, 2009b).

3.3.2 Health Effects Related to Ambient BC Concentrations

Some community epidemiological studies have included BC[2] as one of several indicators of fine particulate air pollution. Of $PM_{2.5}$ components, BC is one of the larger contributors to $PM_{2.5}$ total mass. For example, Bell et al. (2007) examined levels of PM components on a national basis, and identified EC as one of the seven main contributors. The effects observed with BC in health studies are similar to those observed for $PM_{2.5}$ and some other PM constituents (e.g., nickel, vanadium), suggesting that these effects are not attributable solely to BC. Indeed, it would be difficult to separate the contribution of BC to these associations from that of co-emitted OC and other correlated and co-emitted primary pollutants in such studies. Still, these studies provide generally consistent evidence for an association between cardiovascular morbidity and BC concentrations.

A number of studies have reported associations between short-term exposure to BC and cardiovascular effects (See Table 3-2). Telomere length attrition, an indication of biological age that is inversely associated with risk of cardiovascular disease, was associated with ambient BC concentrations in the Boston, MA, area (McCracken et al., 2010). A series of analyses found that changes in blood pressure (Delfino et al., 2010; Mordukhovich et al., 2009; Wilker et al., 2010) and heart rate variability (HRV) (Adar et al., 2007; Chuang et al., 2008; Gold et al., 2005; Huang et al., 2003; Park et al., 2005; Schwartz et al., 2005) were associated with increases in mean ambient BC concentration. The ST-segment of an electrocardiograph represents the period of slow repolarization of the ventricles and ST-segment depression can be associated with adverse cardiac outcomes, including ischemia. Delfino et al. (2011) found positive associations between ST-segment depression and BC concentrations. Homocysteine, a sulfur-containing amino acid formed during metabolism of methionine, is a risk factor for atherosclerosis, myocardial infarction (MI), stroke, and thrombosis. Similarly, lower blood DNA methylation content is found in processes related to cardiovascular outcomes, such as oxidative stress and atherosclerosis. Several studies observed an association between ambient BC concentration and elevated plasma total homocysteine (Park et al., 2008; Ren et al., 2010). An additional study (Baccarelli et al., 2009) observed an association between lower blood DNA methylation content and BC concentrations. Cardiac arrhythmia (a broad group of conditions where there is irregular electrical activity in the heart) was associated with increased concentrations of BC in studies conducted in Boston (Rich et al., 2005; Rich et al., 2006; Zanobetti et al., 2009; Baja et al., 2010; Dockery et al., 2005), but not in Vancouver, Canada (Rich et al., 2004). Another series of analyses has reported inconsistent associations between BC and blood markers of coagulation and inflammation, with some studies finding an effect (Dubowsky et al., 2006; Rückerl et al., 2006; Delfino et al., 2009; 2008; O'Neill et al., 2007), and others finding no effect for a blood marker with large intra-individual variability (i.e., B-type natriuretic peptide or BNP) (Wellenius et al., 2007) or no effects for acute lag periods (i.e., 48 hours or 1 week) (Zeka et al., 2006). Ambient concentrations of BC (Peters et al., 2001; Zanobetti and Schwartz, 2006) and EC (Bell et al., 2009; Peng et al., 2009; Sarnat et al., 2008; Tolbert et al., 2007; Ito et al., 2011) were also found to be associated with hospital admissions and emergency department visits for cardiovascular outcomes.

[2] The monitoring methods used to estimate BC vary, and include various surrogate measurements such as optical BC and thermal-optical EC (see Chapter 5 and Appendix 1). Categorization of studies according to the indicator measurements used should be the focus of future research.

Table 3-2. Summary of Epidemiological Studies of BC and Cardiovascular Health Outcomes. The study reference numbers listed in the left-hand column are for purposes of cross-reference with Figure 3-1. (Source: U.S. EPA)

Reference Number in Figure 3-1	Study	Location	Health Outcome	Metric	Representative Concentration (μg/m^3)	Exposure Assessment[a]	Selected Effect Estimates (95% CI)[b]
Short-term Exposure Studies							
—	McCracken et al. (2010)	Boston, MA	Telomere Length	BC	Mean (annual avg): 0.32	Annual outdoor home concentration estimates from spatiotemporal model	Association with leukocyte telomere length: -7.6% (-12.8%, -2.1%)
1.	Delfino et al. (2010)	Los Angeles, CA	Blood Pressure	BC	Mean (24-h avg): 1.67	Hourly outdoor home air-pollutant concentrations	Change in BP: SBP: 0.22 (-0.65, 1.09) DBP: 0.36 (-0.11, 0.83)
2.	Mordukhovich et al. (2009)	Boston, MA	Blood Pressure	BC	Mean (7-day moving average): 1.10	Continuous measurements from single monitor averaged by hour before BP measurement	Change in BP: SBP: 1.46 (0.10, 2.82) DBP: 0.87 (0.15, 1.59)
3.	Wilker et al. (2010)	Boston, MA	Blood Pressure	BC	Mean (7-day moving average): 0.98	Continuous measurements from single monitor averaged by hour before BP measurement	Change in BP: SBP: 3.52 (2.77, 4.26) DBP: 2.72 (2.31, 3.12)
4.	Adar et al. (2007)	St. Louis, MO	HRV	BC	Median (5-min avg periods): 0.285-2.911 (range across microenvironments)	Two portable carts containing continuous sampling instrumentation	Percent change in HRV: SDNN: -5.3 (-6.5, -4.1) RMSSD: -10.7 (-11.9, -9.5) PNN50+1: -13.2 (-15.0, -11.4) LF: -11.3 (-13.7, -8.8) HF: -18.8 (-21.1, -16.5) LF/HF: 9.3 (7.2, 11.4) HR: 1.0 (0.8, 1.3)
5.	Chuang et al. (2008)	Boston, MA	HRV	BC	Median (24-h avg): 0.79	Continuous measurements from single monitor	Relative risk for ST-segment Depression ≤1 mm: 1.50 (1.19-1.89)
6.	Gold et al. (2005)	Boston, MA	HRV	BC	Median (12-h avg): 1.14	Continuous measurements from single monitor	Relative risk for ST-segment Depression ≤0.5 mm: First rest: 3.8 (0.7, 21.3) Blood pressure: 5.7 (0.6, 56.3) Standing: 8.3 (0.8, 81.9) Exercise: 0.6 (0.1, 3.1) Second rest: 2.8 (0.5, 14.3) Paced breathing: 3.5 (0.5, 23.6)

Reference Number in Figure 3-1	Study	Location	Health Outcome	Metric	Representative Concentration (µg/m^3)	Exposure Assessment[a]	Selected Effect Estimates (95% CI)[b]
7.	Park et al. (2005)	Boston, MA	HRV	BC	Mean (24-h avg): 0.92	Continuous measurements from single monitor	Percent change in HRV: SDNN: -3.4 (-10.2, 3.9) HF: -13.8 (-28.9, 4.4) LF: -2.4 (-16.2, 13.6) LF/HF: 13.2 (-1.1, 29.6)
8.	Schwartz et al. (2005)	Boston, MA	HRV	BC	Mean (24-h avg): 1.2	Continuous measurements from single monitor	Percent change in HRV: SDNN: -5.1 (-1.5, -8.6) r-MSSD: -10.1 (-2.4, -17.2) PNN50: -16.9 (-6.0, -26.6) LF/HF: 7.2 (0.7-14.1)
9.	Delfino et al. (2011)	Los Angeles, CA	HRV	BC	Mean (24-h avg): 1.67	Hourly outdoor home air-pollutant concentrations	Odds ratio for ST-segment Depression ≤1 mm: 2.07 (1.30, 3.29)
10.	Park et al. (2008)	Boston, MA	Plasma Total Homocysteine	BC	Mean (24-h avg): 0.99	Continuous measurements from single monitor	Percent change in total homocysteine: 3.13 (0.76, 5.55)
11.	Ren et al. (2010)	Boston, MA	Plasma Total Homocysteine	BC	Mean (7-day moving average): 0.99	Continuous measurements from single monitor	Percent change in total homocysteine: 0.68 (-0.46, 1.81)
—	Baccarelli et al. (2009)	Boston, MA	Blood DNA Methylation	BC	Mean (24-h avg): 0.89	Continuous measurements from single monitor	Coefficient for effect on methylation: LINE-1: -0.09 (-0.15, -0.02) Alu: -0.02 (-0.08, 0.05)
12.	Baja et al. (2010)	Boston, MA	Ventricular Repolarization	BC	Mean (1-h avg): 1.08	Continuous measurements from single monitor	Change in mean QTc (msec): 1.89 (-0.16, 3.93)
13.	Dockery et al. (2005)	Boston, MA	Arrhythmia	BC	Median (2-day avg): 0.98	Continuous measurements from single monitor	Association with ventricular arrhythmias: Recent arrhythmia > 3 days: 1.02 (0.83, 1.24) Recent arrhythmia <3 days: 1.74 (1.28, 2.37)
14.	Rich et al. (2005)	Boston, MA	Arrhythmia	BC	Median (24-h avg): 0.94	Continuous measurements from single monitor	Odds ratio for ventricular arrhythmias: 0.93 (0.74, 1.18)
15.	Rich et al. (2006)	Boston, MA	Arrhythmia	BC	Median (24-h avg): 0.94	Continuous measurements from single monitor	Odds ratio for paroxysmal atrial fibrillation: 1.46 (0.67, 3.17)

Reference Number in Figure 3-1	Study	Location	Health Outcome	Metric	Representative Concentration (µg/m^3)	Exposure Assessment[a]	Selected Effect Estimates (95% CI)[b]
16.	Zanobetti et al. (2009)	Boston, MA	Arrhythmia	BC	Median (6-h avg): Ambient: 0.72 Indoor: 0.41 Outdoor: 0.50	Continuous ambient measurements from single monitor; indoor and outdoor measured continuously at participants' homes	Odds ratio for maximum T-wave alternans ≤ 26 µV: 1.42 (1.19, 1.69)
17.	Rich et al. (2004)	Vancouver, Canada	Arrhythmia	EC	Mean (24-h avg): 0.8	Continuous measurements from single monitor	Odds ratio for defibrillator discharge: 1.06 (0.87, 1.33)* *estimated from graph
18.	Sorensen et al. (2003)	Copenhagen, Denmark	Blood Markers of Coagulation and Inflammation	Carbon Black	Median (24-h avg): 8.1 (10-6/m)	Personal exposure	Association with plasma proteins: 4.1% increase in plasma proteins per 1 x 10^{-5}/m increase in personal CB exposure
19.	Dubowsky et al. (2006)	St. Louis, MO	Blood Markers of Coagulation and Inflammation	BC	Mean (24-h avg): 0.9	Continuous measurements from single monitor	Association with markers of inflammation: IL-6: -0.8 (-8.9, 8.0) CRP: 13 (-0.34, 28) WBC: 1.3 (-2.1, 4.8)
20.	Ruckerl et al. (2006)	Erfurt, Germany	Blood Markers of Coagulation and Inflammation	EC	Mean (24-h avg): 2..6	Continuous measurements from single monitor	Odds ratio for increase in blood marker above 90th percentile: CRP: 1.3 (0.7, 2.4) ICAM-1: 2.6 (1.7, 3.8)
21.	Delfino et al. (2008)	Los Angeles, CA	Blood Markers of Coagulation and Inflammation	BC	Mean (24-h avg): 2.00	Outdoor home measurements	Coefficient for association: CRP (ng/ml): 585.61 IL-6 (pg/ml): 0.48 sTNF-RII (pg/ml): 135.15 sP-selectin (ng/ml): 1.99 Cu, Zn-SOD (U/g Hb): -187.95
22.	Delfino et al. (2009)	Los Angeles, CA	Blood Markers of Coagulation and Inflammation	BC	Mean (24-h avg): 1.59 – 1.76	Hourly outdoor home air pollutants	Coefficient for association CRP (ng/ml): 252 (-54, 558) IL-6 (pg/ml): 0.16 (0.01, 0.31) sTNF-RII (pg/ml): 38 (-26, 102) sP-selectin (ng/ml): 1.19 (-0.52, 2.90) Cu, Zn-SOD (U/g Hb): -114 (-229, 1) TNF-α (pg/ml): 0.02 (-0.06, 0.10) GPx-1 (U/g Hb): -0.47 (-0.97, 0.03)
23.	O'Neill et al. (2007)	Boston, MA`	Blood Markers of Coagulation and Inflammation	BC	Mean (24-h avg): 1.1	Continuous measurements from single monitor	Percent change in inflammatory marker: ICAM-1 (ng/ml): 5.84 (0.87, 11.05) VCAM-1 (ng/ml): 9.26 (2.98, 15.91) vWF (proportion): 7.96 (-4.34, 21.84)

Reference Number in Figure 3-1	Study	Location	Health Outcome	Metric	Representative Concentration (μg/m^3)	Exposure Assessment[a]	Selected Effect Estimates (95% CI)[b]
24.	Wellenius et al. (2007)	Boston, MA	Blood Markers of Coagulation and Inflammation	BC	Mean (24-h avg): 0.73	Continuous measurements from single monitor	"No significant associations observed between [BC] and BNP levels at any of the lags examined."
25.	Zeka et al. (2006)	Boston, MA	Blood Markers of Coagulation and Inflammation	BC	Mean (2-day avg): 0.77	Continuous measurements from single monitor	Percent increase in inflammatory marker: Fibrinogen: 0.84 (-0.63, 2.31) CRP: 4.51 (-2.03, 11.06) Sediment rate: -4.56 (-25.55, 16.43) WBC count: -0.63 (-2.45, 1.19)
26.	Peters et al. (2001)	Boston, MA	ED visits and hospital admissions for CVD	BC	Mean (24-h avg): 1.35	Continuous measurements from single monitor	Odds ratio for MI: 1.21 (0.87, 1.70)
27.	Zanobetti and Schwartz (2006)	Boston, MA	ED visits and hospital admissions for CVD	BC	Median (24-h avg): 1.15	Continuous measurements from single monitor	Percent change in MI admissions: 8.34 (0.21, 15.82)
28.	Bell et al. (2009)	106 U.S. Counties	ED visits and hospital admissions for CVD	EC	Mean (24-h avg): 0.72	County-wide averages	Percent increase in health effect estimate: 25.8 (4.4, 47.2)
29.	Peng et al. (2009)	119 U.S. Counties	ED visits and hospital admissions for CVD	EC	Median (24-h avg): 0.58	County-wide averages	Percent increase in CVD hospital admissions: 0.72 (0.43, 1.01)
30.	Sarnat et al. (2008)	Atlanta, GA	ED visits and hospital admissions for CVD	EC	Mean (24-h avg): 1.4-1.7	Positive matrix factorization applied to measurements from single monitor	Relative risk of ED visit for CVD: 1.025 (1.014, 1.036)
31.	Tolbert et al. (2007)	Atlanta, GA	ED visits and hospital admissions for CVD	EC	Mean (24-h avg): 1.6	Continuous measurements from single monitor	Relative risk of ED visit for CVD: 1.015 (1.005, 1.025)
32.	Ito et al. (2011)	New York, NY	ED visits and hospital admissions for CVD	EC	Mean (24-h avg): 1.13	Average of continuous measurements from three monitors	Percent excess risk: 1.4 (0.1, 2.7)

Reference Number in Figure 3-1	Study	Location	Health Outcome	Metric	Representative Concentration (μg/m^3)	Exposure Assessment[a]	Selected Effect Estimates (95% CI)[b]
Long-term exposure studies							
33.	O'Neill et al. (2005)	Boston, MA	Endothelial dysfunction	BC	Mean (24-h avg): 1.0	Continuous measurements from single monitor	Percent change in vascular reactivity among those with (a) diabetes and (b) all subjects: Flow-mediated dilation: (a) -12.6 (-21.7, -2.4); (b) -9.3 (-17.8, 0.2) Nitroglycerin-mediated dilation: -(a) 6.6 (-14.0, 1.5); (b) -5.4 (-12.0, 1.7)
34.	Madrigano et al. (2010)	Boston, MA	Endothelial dysfunction	BC	Mean (24-h avg): 0.84	Continuous measurements from single monitor	Percent change in blood markers: sVCAM-1: 4.52 (1.09, 7.96) sICAM-1: -1.37 (-3.89, 1.15)
35.	Alexeeff et al. (2011)	Boston, MA	Markers of inflammation and endothelial response	BC	Mean (24-h avg): 0.42	Validated spatio-temporal (land use regression) model	Percent change in blood markers: 4 wk exposure, sVCAM-1: 1.00 (-0.65, 2.67) 4 wk exposure, sICAM-1: 1.50 (0.22, 2.80) 8 wk exposure, sVCAM-1: 1.20 (-0.58. 3.02) 8 wk exposure, sICAM-1: 1.58 (0.18, 3.00) 12 wk exposure, sVCAM-1: 1.26 (-0.58, 3.14) 12wk exposure, sICAM-1: 1.49 (0.04, 2.95)
36.	Gan et al. (2011)	Vancouver, BC	CHD hospitalizations	BC	Mean (24-h avg): 1.19	High resolution land use regression model	Relative Risk of CHD hospitalization: 1.01 (1.00, 1.03)

[a] A more complete description or evaluation of the BC monitoring method is beyond the scope of this summary, and may include surrogate measurements.

[b] SBP=systolic blood pressure; DBP=diastolic blood pressure; SDNN=standard deviation of normal intervals measured between consecutive sinus beats; RMSSD=root mean square successive difference, a measure of heart period variability; PNN50=number of times per hour in which the change in consecutive normal sinus intervals exceeds 50 milliseconds; LF=low frequency component of heart rate variability; HF=high frequency component of heart rate variability; HR=heart rate; Line-1=long interspersed nuclear element-1; Alu=short stretch of DNA; IL-6=interleukin 6; CRP=c-reactive protein; WBC=white blood cells; ICAM=intercellular adhesion molecule; sTNF-RII=extracellular domain of the tumor necrosis factor receptor; sp-Selectin=plasma soluble P-selectin; Cu, Zn-SOD=copper/zinc superoxide dismutase; TNF-α=tumor necrosis factor-alpha; GPx-1=glutathione peroxidase; VCAM=vascular cell adhesion molecule; vWF=vonWillebrand Factor; BNP=B-type natriuretic peptide; MI=myocardial infarction; CVD=cardiovascular disease; ED=emergency department; CHD=coronary heart disease.

The most noteworthy new cardiovascular-related revelation in recent years with regard to long-term PM exposure is that the systemic vasculature may be a target organ (U.S. EPA, 2009b). Endothelial dysfunction is a factor in many diseases and may contribute to the origin and/or exacerbation of MI or ischemic heart disease, as well as hypertension. Endothelial dysfunction is also a characteristic feature of early and advanced atherosclerosis. New evidence supports an association of ambient BC with decrements in the systemic vasculature. O'Neill et al. (2005) reported that increases in mean BC concentration were associated with decreased vascular reactivity among diabetics, but not among subjects at risk for diabetes. Several recent studies (Madrigano et al., 2010; Alexeeff et al., 2011; Gan et al., 2011) observed that ambient BC was associated with a marker of endothelial function and inflammation, and that genes related to oxidative defense might modify this association. Consistent with these findings, animal toxicological studies have shown that BC can affect heart rate variability (Tankersley et al., 2007; 2004), cardiac contractility (Tankersley et al., 2008) and oxidative stress response (Tankersley et al., 2008), providing biological plausibility for a long-term effect on cardiovascular health.

Overall, the limited body of evidence suggests that ambient BC may be associated with a continuum of effects ranging from more subtle subclinical measures (e.g. changes in blood pressure, heart rate variability) to emergency department visits and hospital admissions for cardiovascular outcomes (Figure 3-1). Generally, this is consistent with the association observed for $PM_{2.5}$ and cardiovascular outcomes (Janssen et al., 2011), as described above (Section 3.3.1).

Fewer studies have examined the effects of BC with respiratory effects (Table 3-3). Clark et al. (2010) investigated the effect of exposure to ambient air pollution in utero and during the first year of life on risk of subsequent asthma diagnosis (incident asthma diagnosis up to age 3-4) and reported that BC exposure was associated with a 14% (1-29%) increase in asthma risk. Delfino et al. (2006) found associations between airway inflammation and ambient EC concentrations among asthmatic children, while Jansen et al. (2005) reported an association with a marker of pulmonary inflammation and BC concentrations among older adults. These results are supported by toxicological studies reporting evidence of airway inflammation (Godleski et al., 2002; Saldiva et al., 2002). There is consistent evidence from a number of studies that report associations of respiratory symptoms among both asthmatic and non-asthmatic children and ambient BC or EC (Kim et al., 2004; Mann et al., 2010; McConnell et al., 2003; Patel et al., 2010; Spira-Cohen et al., 2011). Additionally, Suglia et al. (2008) reported that ambient BC was associated with decreased lung function among urban women. Recent studies evaluated the effect of ambient BC or EC on respiratory hospital admissions and found statistically significant associations between the county-average ambient concentrations of BC or EC and respiratory hospital admissions (Zanobetti and Schwartz, 2006; Bell et al., 2009; Ostro et al., 2009). However other studies found less consistent evidence (Peng et al., 2009; Mohr et al., 2008) or no evidence (Sarnat et al., 2008; Tolbert et al., 2007) for an association between ambient EC and respiratory emergency department visits. Overall, there is inconsistent evidence for an association between ambient BC concentrations and respiratory effects. Similar to what was observed in studies of $PM_{2.5}$, studies examining ambient BC report increased respiratory symptoms in asthmatic children, but less consistent evidence for an association with emergency department visits and hospital admissions.

Several recent epidemiological studies have examined the association between mortality and short-term ambient exposure to components of $PM_{2.5}$, including BC or EC (Table 3-4). Lippmann et al. (2006) reported that nickel, vanadium, and EC were the best predictors, respectively, of PM_{10} risk estimates for mortality. Cakmak et al. (2011) reported an association between increased exposure to concentrations of EC and increases in all cause mortality, while Ito et al. (2011) and Ostro et al. (2007) found positive associations between EC and cardiovascular mortality. These associations (Ostro et al., 2007) were higher in individuals with lower educational attainment and of Hispanic ethnicity (Ostro et al., 2008). Studies of long-term exposure to EC (Lipfert et al., 2006; 2009) and BC (Gan et al., 2011) also report associations with mortality. Overall, the limited body of evidence examining the association of ambient BC with mortality has reported associations with mortality, especially cardiovascular mortality. This association is consistent with the evidence for a causal relationship between $PM_{2.5}$ and mortality.

3.3.2.1 Health Effects Related to Indicators of Ambient BC Concentrations

Concentrations of many traffic-generated air pollutants are elevated for up to 300-500 meters downwind of roads with high traffic volumes (Zhou and Levy, 2007). Numerous sources on roads contribute to elevated roadside concentrations, including exhaust and evaporative emissions,

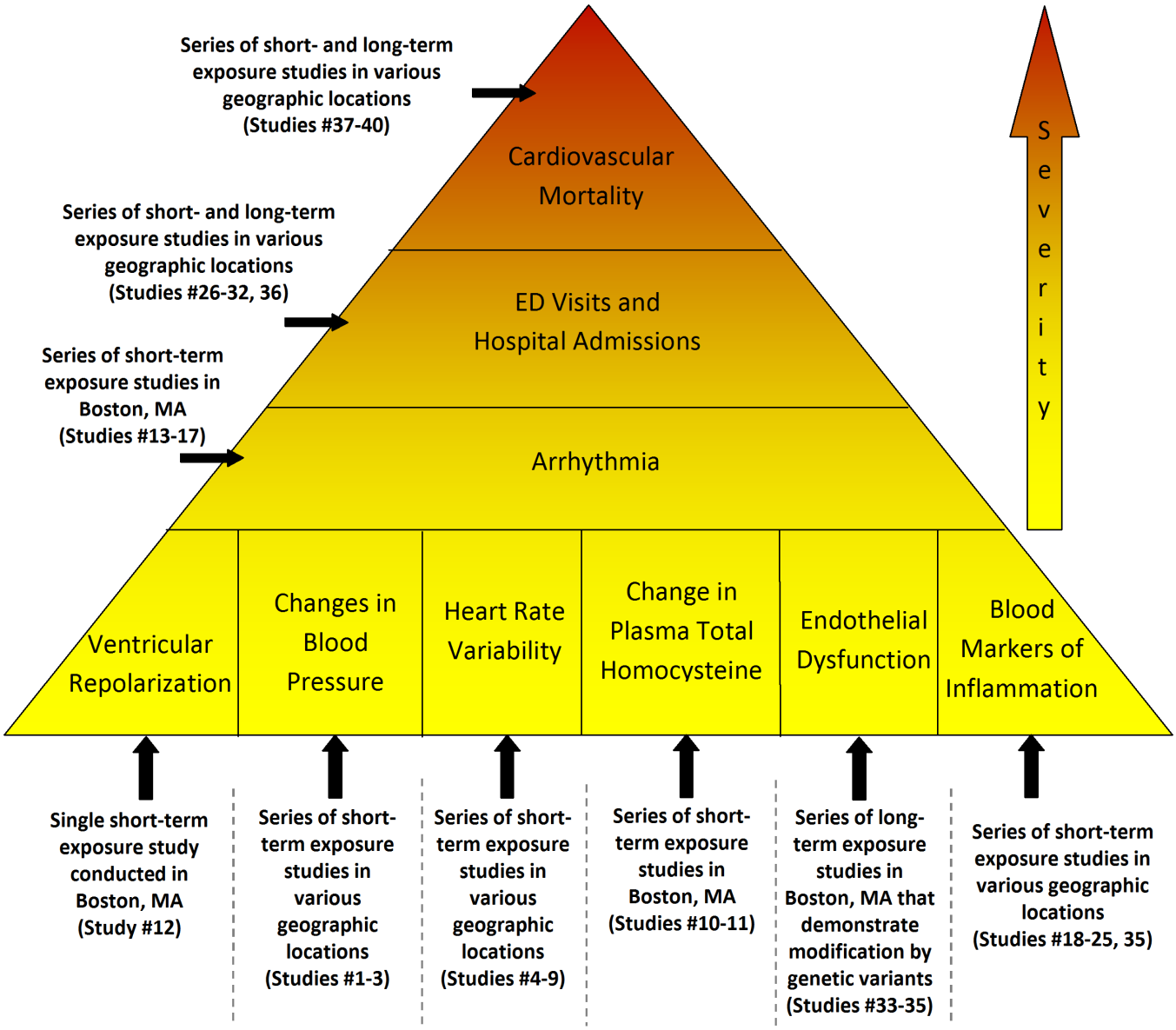

Figure 3-1. Conceptual Diagram of the Epidemiological Evidence for the Association of BC with the Continuum of Cardiovascular Effects, including sub-clinical effects (bottom level of the pyramid) and clinical effects, increasing in severity moving up the pyramid. It is important to note that the body of evidence describing the association between BC and cardiovascular effects is much smaller and less consistent than the one characterizing $PM_{2.5}$ and cardiovascular effects. The study reference numbers listed in parentheses correspond to the reference numbers assigned to individual studies in the left-hand column of Table 3-2 and Table 3-4. For study-specific details, please see Table 3-2 and Table 3-4. (Source: U.S. EPA)

and resuspension of road dust and tire and brake wear. Concentrations of several criteria and hazardous air pollutants are elevated near major roads. Furthermore, different semi-volatile organic compounds and chemical components of PM, including BC, organic material, and trace metals, have been reported at higher concentrations near major roads. While this document is focused on the health effects associated with BC specifically, this section discusses the mixture of different pollutants near major roadways, of which BC is a component. As such, this section emphasizes traffic-related air pollution, in general, as the relevant indicator of exposure to BC.

Table 3-3. Summary of Epidemiological Studies of BC and Respiratory Health Outcomes. (Source: U.S. EPA)

Study	Location	Health Outcome	Metric	Representative Concentration ($\mu g/m^3$)	Exposure Assessmenta	Selected Effect Estimates (95% CI)
Clark et al. (2010)	British Columbia	Development of childhood asthma	BC	Mean (24-h avg): 1.34	Land Use Regression (LUR) Model	Asthma risk due to average exposures In utero exposure: 1.08 (1.02, 1.15) First-year exposure: 1.14 (1.01, 1.29)
Delfino et al. (2006)	Los Angeles, CA	Exhaled nitric oxide (biomarker of airway inflammation)	EC	Mean (24-h avg): 0.71-1.61	Personal exposure and continuous measurements from central site monitors	Association between EC and exhaled NO: Personal exposure: 0.72 (0.32, 1.12) Central site exposure: 1.38 (0.15, 2.61)
Jansen et al. (2005)	Seattle, WA	Exhaled nitric oxide (biomarker of airway inflammation)	BC	Mean (24-h avg): Central site: 2.01 Indoor: 1.34 Personal: 1.64	Personal exposures, indoor monitors, and central outdoor monitoring site	Association between BC and exhaled NO among asthmatics: Central site exposure: 2.3 (1.1, 3.6) Indoor Exposure: 4.0 (2.0, 5.9) Personal exposure: 1.2 (0.2, 2.2)
Kim et al. (2004)	San Francisco, CA	Bronchitis symptoms and asthma	BC	Mean (24-h avg): 0.8	BC measured at 10 school sites	Odds ratios of respiratory illness by school-based BC concentration: Bronchitis: 1.04 (1.00, 1.08) Asthma: 1.07 (0.98, 1.17)
Mann et al. (2010)	Fresno, CA	Wheeze among asthmatic children	EC (estimated from BC)	Median (24-h avg): 1.3	Continuous measurements from single monitor	Odds ratio of EC and wheeze: 1.12 (0.97, 1.30)
McConnell et al. (2003)	12 Southern California communities	Bronchitic symptoms among asthmatic children	EC	Mean (24-h avg): 0.71	Annual averages computed from 2-week averages measured in each community	Bronchitic symptoms as a function of the 4-yr avg EC concentration: 1.64 (1.06, 2.54)
Patel et al. (2010)	New York City, NY	Respiratory symptoms among asthmatics and non-asthmatics	BC	Median (24-h avg): 0.49-2.4	BC measured at 4 high schools	Odds ratios for respiratory symptoms and use of asthma medication: Wheeze: 1.11 (1.00, 1.22) Cough: 0.95 (0.87, 1.03) Shortness of Breath: 1.26 (1.14, 1.38) Chest Tightness: 1.11 (1.01, 1.24) Use of Asthma Medication: 1.09 (0.89, 1.33)
Spira-Cohen et al. (2011)	South Bronx, NY	Lung function and respiratory symptoms among asthmatic children	EC	Mean (24-h avg): 1.9	Personal and outdoor school site monitoring at 4 elementary schools	Effect estimates for lung function decrements: Personal EC, PEF: -9.13 (-19.13, 0.86) Personal EC, FEV1: -0.02 (-0.09, 0.04) School-site EC, PEF: -4.58 (-14.01, 4.85) School-site EC, FEV1: 0.01 (-0.04, 0.07) Relative Risks for respiratory symptoms: Personal EC, cough: 1.61 (1.17, 2.21) Personal EC, wheeze: 1.67 (1.05, 2.66) Personal EC, shortness of breath: 1.41 (1.01, 1.99) Personal EC, total symptoms: 1.54 (1.13, 2.10)

Study	Location	Health Outcome	Metric	Representative Concentration (µg/m^3)	Exposure Assessmenta	Selected Effect Estimates (95% CI)
Suglia et al. (2008)	Boston, MA	Lung function among women	BC	Mean (predicted annual): 0.62	Local BC levels estimated by spatiotemporal land-use regression model	Effect estimates for change in lung function: FEV_1: -1.09 (-2.5, 0.3) FVC: -0.62 (-1.9, 0.6) $FEF_{25-75\%}$: -3.03 (-5.8, -0.3)
Bell et al. (2009)	106 U.S. Counties	ED visits and hospital admissions for respiratory effects	EC	Mean (24-h avg): 0.72	County-wide averages	Percent increase in health effect estimate: 511 (80.7, 941)
Ostro et al. (2009)	6 CA Counties	ED visits and hospital admissions for respiratory effects	EC	Mean (24-h avg): 1.0	County-wide averages	Excess risk per IQR: Ages <19: 5.4 (0.8, 10.3) Ages <19, Cool season only: 6.8 (-0.2, 14.2)
Peng et al. (2009)	119 U.S. Counties	ED visits and hospital admissions for respiratory effects	EC	Median (24-h avg): 0.58	County-wide averages	Percent increase in respiratory hospital admissions: 0.43 (-0.02, 0.85)
Mohr et al. (2008)	St. Louis, MO	ED visits and hospital admissions for respiratory effects	EC	Median (24-h avg): ~0.6-0.7	Continuous measurements from single monitor	Relative risk for asthma ED visits: Ages 2-5, summer: 1.01 (0.93, 1.09) Ages 6-10, summer: 0.98 (0.77, 1.26) Ages 11-17, summer: 1.09 (1.02, 1.17) Ages 2-17, summer: 1.05 (1.00, 1.11)
Sarnat et al. (2008)	Atlanta, GA	ED visits and hospital admissions for respiratory effects	EC	Mean (24-h avg): 1.4-1.7	Positive matrix factorization applied to measurements from single monitor	Relative risk of ED visit for Resp: 0.996 (0.988, 1.003)
Tolbert et al. (2007)	Atlanta, GA	ED visits and hospital admissions for respiratory effects	EC	Mean (24-h avg): 1.6	Continuous measurements from single monitor	Relative risk of ED visit for respiratory effects: 0.996 (0.989, 1.004)
Zanobetti and Schwartz (2006)	Boston, MA	ED visits and hospital admissions for respiratory effects	BC	Median (24-h avg): 1.15	Continuous measurements from single monitor	Percent change in pneumonia ED visits: 11.71 (4.79, 17.36)

[a] A more complete description or evaluation of the BC monitoring method is beyond the scope of this summary, and may include surrogate measurements.

[b] NO=nitric oxide; PEF=peak expiratory flow; FEV_1=forced expiratory volume in 1 second; FVC=forced vital capacity; $FEF_{25-75\%}$=difference between the 25th and 75th percentile of forced expiratory flow; ED=emergency department.

Table 3-4. Summary of Epidemiological Studies of BC and Mortality. The study reference numbers listed in the left-hand column are for purposes of cross-reference with Figure 3-1. (Source: U.S. EPA)

Reference Number in Figure 3-1	Study	Location	Health Outcome	Metric	Representative Concentration (μg/m³)	Exposure Assessment[1]	Selected Effect Estimates (95% CI)
Short-term Exposure Studies							
—	Cakmak et al. (2011)	3 urban areas in Chile	All-cause mortality	EC	Mean (24-h avg): 2.69 – 5.37	Continuous measurements from 3 monitors (one in each urban area)	Mortality risk ratio: All-cause: 1.084 (1.067, 1.100) ≤64 yrs old: 1.052 (1.019, 1.085) ≥85 yrs old: 1.137 (1.103, 1.173)
37.	Ito et al. (2011)	New York City, NY	CVD mortality	EC	Mean (24-h avg): 1.13	Continuous measurements from 3 monitors	Percent increase in mortality: All year: 2.0 (0.8, 3.3) Warm season: 2.3 (0.3, 4.3) Cold season: 1.6 (-0.1, 3.2)
—	Lippmann et al. (2006)	90 U.S. cities and Hong Kong	All-cause mortality	EC	(Not reported)	Speciation data from U.S. EPA AQS from 2000-2003	"Elevated but nonsignificant increases >0.21 [in health effect estimate] were associated with EC"
38.	Ostro et al. (2007)	6 California counties	All-cause, CVD and respiratory mortality	EC	Mean (24-h avg): 0.966	Continuous measurements across monitors	Percent excess risk: All-cause mortality: 0.7 (-0.6, 1.9) Cardiovascular mortality: 2.1 (0.3, 3.9) Respiratory mortality: 1.2% (-2.2, 4.7)
39.	Zhou et al. (2011)	Seattle, WA and Detroit, MI	All-cause, CVD and respiratory mortality	BC (as a surrogate index of EC in the filter samples)	Median (24-h avg): 0.52-0.71	Continuous measurements from 1 monitor in each city	Percent excess risk: Detroit, warm season: All-cause mortality: 1.359 (-1.027, 3.804) Cardiovascular mortality: 1.227 (-2.276, 8.499) Respiratory mortality: 0.426 (-7.046, 4.062) Seattle, warm season: All-cause mortality: -1.652 (-7.029, 4.035) Cardiovascular mortality: -0.539 (-9.764, 9.629) Respiratory mortality: 10.579 (-8.382, 33.465)
Long-term Exposure Studies							
—	Lipfert et al. (2006)	187 U.S. Counties	All-cause mortality	EC	Mean (24-h avg): 0.79	Speciation data from U.S. EPA AQS from 2002	Coefficient (SE) for association with mortality: 0.1664 (0.05884)
—	Lipfert et al. (2009)	3,065 U.S. Counties	All-cause mortality	EC	Mean (24-h avg): 0.82	Estimates from Atmospheric and Environmental Research, Inc (AER) plume-in-grid air quality model	Cumulative mortality risks: All subjects: 1.07 (1.05, 1.10) Subjects in counties with high traffic density: 1.15 (1.13, 1.16) Subjects in counties with low traffic density: 1.04 (1.01, 1.07)
40.	Gan et al. (2011)	Vancouver, BC	CHD mortality	BC	Mean (24-h avg): 1.19	High resolution land use regression model	Relative Risk of CHD mortality: 1.06 (1.03, 1.09)

1. A more complete description or evaluation of the BC monitoring method is beyond the scope of this summary, and may include surrogate measurements.
2. SE=standard error; CHD=coronary heart disease.

Populations near major roads experience greater risk of certain adverse health effects. The Health Effects Institute (HEI) published a report on the health effects of traffic-related air pollution (Health Effects Institute, 2010). It concluded that evidence is "sufficient to infer the presence of a causal association" between traffic exposure and exacerbation of childhood asthma symptoms. The HEI report also concludes that the evidence is either "sufficient" or "suggestive but not sufficient" for a causal association between traffic exposure and new childhood asthma cases. A review of asthma studies by Salam et al. (2008) reaches similar conclusions. The HEI report also concludes that there is "suggestive" evidence for pulmonary function deficits associated with traffic exposure, but concluded that there is "inadequate and insufficient" evidence for causal associations with respiratory health care utilization, adult-onset asthma, chronic obstructive pulmonary disease (COPD) symptoms, and allergy. A review by Holguin (2008) notes that the effects of traffic on asthma may be modified by nutrition status, medication use, and genetic factors.

The HEI report also concludes that evidence is "suggestive" of a causal association between traffic exposure and all-cause and cardiovascular mortality. There is also evidence of an association between traffic-related air pollutants and cardiovascular effects such as changes in heart rhythm, heart attack, and cardiovascular disease. The HEI report characterizes this evidence as "suggestive" of a causal association, and an independent epidemiological literature review by Adar and Kaufman (2007) concludes that there is "consistent evidence" linking traffic-related pollution and adverse cardiovascular health outcomes.

Some studies have reported associations between traffic exposure and other health effects, such as birth outcomes (e.g., low birth weight) and childhood cancer. The HEI report concludes that there is currently "inadequate and insufficient" evidence for a causal association between these effects and traffic exposure. A review by Raaschou-Nielsen and Reynolds (2006) concluded that evidence of an association between childhood cancer and traffic-related air pollutants is weak, but noted the inability to draw firm conclusions based on limited evidence.

Investigators have attempted to trace PM health effects back to specific sources (e.g., traffic) using source apportionment techniques. A number of these studies have linked BC-rich sources, including motor vehicles and traffic, with adverse cardiovascular and respiratory health outcomes (U.S. EPA, 2009b, Section 6.6.2). For example, Sarnat et al. (2008) found consistent positive associations between cardiorespiratory morbidity and sources related to biomass combustion and metal processing. However, in general there are uncertainties associated with source apportionment methods; these have been characterized in a recent review (Stanek et al., 2011). First, the number of components that comprise PM is not only large, but the correlations between them can be high. Some studies identify the resulting groups or factors with named sources of ambient PM (e.g., "traffic") or PM-related processes (e.g., "secondary organic aerosols"), but many do not draw explicit links between factors and actual sources or processes. Second, there is no well-established, objective method for conducting the various forms of factor analysis and source apportionment, leaving much of the model operation and assignment of factors to sources open to judgment by individual investigators. Because of this and differences in composition and correlations among components between studies, the factors identified vary considerably, thus complicating direct comparisons. Likewise, it cannot be ruled out that a seemingly comparable factor across studies may correspond to different sources depending on location. Despite these uncertainties, a number of studies (e.g., Hopke et al., 2006; Thurston et al., 2005; Mar et al., 2006; Ito et al., 2006; Sarnat et al., 2008) have found that effect estimates based on different source apportionment methods were generally in close agreement, and that the variability in relative risks across source apportionment methods was smaller than the variability across source types (Ito et al., 2006).

Overall, source apportionment studies report little agreement for a particular group of components or sources being responsible for cardiovascular or respiratory effects, which may be due in part to the limited number of studies evaluating these endpoints (Stanek et al., 2011). The results of source apportionment studies indicate that many grouped components can be linked with various health effects, but collectively they have not yielded a clear and consistent association with specific health outcomes.

Finally, it is important to note that a variety of hazardous air pollutants (HAPs) including polycyclic aromatic hydrocarbons (PAHs), dioxins and furans, are co-emitted with BC (Allen et al., 1996; Shih et al., 2008; Hedman et al., 2006; Yadav et al., 2010; Amador-Muñoz et al., 2010; Walgraeve et al., 2010). These HAPs are associated with adverse health effects including cancer and respiratory effects, among others. Reductions in HAP emissions occurring in conjunction with BC mitigation programs will help reduce these

health risks. Furthermore, these toxic pollutants are generally persistent once they are emitted into the environment, so these co-benefits can be expected to have long-lasting beneficial impacts (Quiroz et al., 2010; Chi et al., 2010).

3.3.2.2 Magnitude of Impacts of Ambient $PM_{2.5}$ in the United States and Globally

$PM_{2.5}$ is a serious detriment to public health, both in the United States and globally. Regulation of $PM_{2.5}$ concentrations in the United States has resulted in significant declines in $PM_{2.5}$ concentrations and $PM_{2.5}$-related mortality over time (Fann and Risley, 2011). However, many areas of the country remain in non-attainment for the $PM_{2.5}$ NAAQS, and 2005 ambient $PM_{2.5}$ concentrations have been associated with 130,000 premature deaths annually, corresponding to 1.1 million years of life lost (Fann et al., 2011). While a portion of these $PM_{2.5}$-related deaths will be reduced by the recently finalized Cross-State Air Pollution Rule aimed at controlling SO_2 and NO_X emissions (U.S. EPA, 2011e), $PM_{2.5}$ remains a significant risk factor for public health in the United States.

Globally, ambient air pollution concentrations are often much higher than those found in the United States, and the public health burden is correspondingly more severe. In 2004, the WHO estimated that ambient $PM_{2.5}$ in urban areas was associated with about 800,000 premature deaths each year globally, based on surface monitor observations which are limited in many locations around the world (Cohen et al., 2004). More recently, Anenberg et al. (2010) estimated about 3.7 million global premature deaths annually due to outdoor anthropogenic $PM_{2.5}$ using a global atmospheric model to isolate the total anthropogenic contribution to $PM_{2.5}$ concentrations (calculated as the difference between simulated present-day concentrations in 2000 and preindustrial concentrations in 1860) with full spatial coverage including both urban and rural populations. This estimate was still considered to be an underestimate since the resolution of the atmospheric model was too coarse to capture fine spatial gradients of both concentration and population, particularly in urban areas. Impacts of outdoor $PM_{2.5}$ were estimated to be an order of magnitude higher than the impacts of outdoor ozone, due both to high $PM_{2.5}$ concentrations, particularly in very populated areas, and a stronger mortality relationship for $PM_{2.5}$ relative to ozone (e.g., Jerrett et al., 2009; Krewski et al., 2009).

The WHO estimates that urban air pollution ranks as the 10th and 8th major risk factor in medium- and high-income countries, respectively (World Health Organization, 2009). Urban air pollution is not ranked in the top 10 of major risk factors in low-income countries since other risk factors (e.g. childhood underweight and unsafe water, sanitation, and hygiene) are so significant; however, a much larger portion of deaths related to ambient $PM_{2.5}$ are expected to occur in developing regions (Cohen et al., 2004; Anenberg et al., 2010). The ongoing Global Burden of Diseases, Injuries, and Risk Factors Study[3] is expected to update these burden estimates leveraging the advantages from air pollution monitors on the ground, satellite observations, and atmospheric models.

Since the literature on differential toxicity of $PM_{2.5}$ components is currently inconclusive, these studies all assume that all $PM_{2.5}$ components are equally toxic, and calculate premature deaths associated with total $PM_{2.5}$ concentrations from the epidemiology literature. Using the same assumption, Anenberg et al. (2011) estimated that halving anthropogenic BC emissions globally avoids 157,000 premature deaths annually. Multiplying this estimate by two for the total anthropogenic BC burden (using a reasonable assumption that $PM_{2.5}$ concentrations respond about linearly to BC emission changes) yields about 314,000 avoided premature deaths annually worldwide.

3.3.3 Health Effects Related to Indoor BC Exposures

BC is a component of indoor air pollution, which has been implicated in an array of adverse health effects for those who rely on solid fuels for everyday cooking and heating, mostly in the form of biomass (e.g., wood, animal dung, or crop wastes) but also coal (mainly in China) (Rehfuess et al., 2006). The use of solid fuels in poorly ventilated conditions results in high levels of indoor air pollution, most seriously affecting women and their youngest children (Bruce et al., 2000; Martin et al., 2011). Recent observational studies have suggested that indoor air pollution from biomass fuel is associated with respiratory morbidity, including acute lower respiratory tract infections in children (Smith et al., 2000a; 2011) and COPD in women (Orozco-Levi et al., 2006; Rinne et al., 2006; Liu et al., 2007; Kiraz et al., 2003; Regalado et al., 2006; Ramirez-Venegas et al., 2006; Ezzati et al., 2004; Smith et al., 2004). Exposure to biomass smoke in Guatemalan women has been shown to increase diastolic blood pressure (McCracken et al., 2007). Evidence also exists that implicates exposure to biomass fuel smoke in adverse effects on

[3] *http://www.who.int/healthinfo/global_burden_disease/GBD_2005_study/en/index.html.*

different birth outcomes, including low birth weight and stillbirth (Boy et al., 2002; Sram et al., 2005; Pope et al., 2010). Finally, exposure to indoor air pollution from solid fuel use has been linked to mortality (World Health Organization, 2009).

3.3.3.1 Magnitude of Impacts of Indoor Exposures to $PM_{2.5}$ Globally

Globally, more than half of the population burns solid fuels (e.g. coal, wood, straw, agricultural residue, dung, etc.) for cooking and heating, mainly in the developing world (World Health Organization, 2009). Indoor burning of solid fuels results in high exposure concentrations, as emissions are largely uncontrolled, the homes in which they are used often have poor ventilation, and women and children may spend long periods of time in direct exposure to the emissions during cooking activities. Solid fuel combustion emits a mixture of harmful substances, including $PM_{2.5}$. Consistent with the epidemiological literature on indoor air pollution, impact assessments generally relate risk of mortality with household use of solid fuel combustion, including the total mixture of emissions, rather than using a concentration-response function for individual pollutants (e.g., $PM_{2.5}$).

The WHO estimates that exposure to indoor burning of solid fuels is associated with 2 million annual premature deaths worldwide (World Health Organization, 2009; Smith et al., 2004). Globally, indoor smoke from solid fuels ranks as the 10th leading risk factor for premature death and contributes 3.3% of total deaths. In terms of overall disease burden, as measured in Disability Adjusted Life Years (DALYs), indoor smoke from solid fuels ranks as the 9th leading risk factor globally, associated with 2.7% of all DALYs. It is particularly a problem in low-income countries, where indoor smoke from solid fuels ranks as the 6th leading mortality risk factor (4.8% of total deaths) and the 5th leading disease risk factor (4% of all DALYs). Indoor smoke from solid fuels does not rank as a major risk factor for high-income countries, where use is relatively limited and ventilation is generally sufficient to maintain air quality indoors. As for ambient air pollution, the ongoing Global Burden of Diseases, Injuries, and Risk Factors Study[4] is expected to update these burden estimates with improved assumptions and more recent demographic information.

[4] *http://www.who.int/healthinfo/global_burden_disease/GBD_2005_study/en/index.html.*

3.4 Non-Climate Welfare Effects of $PM_{2.5}$, Including BC

Non-climate welfare effects resulting from BC emissions are discussed in terms of $PM_{2.5}$ exposure and deposition. Visibility impairment, which is caused by light scattering and absorption by suspended particles and gases, is the primary non-climate welfare effect of BC. Crop yields may also be adversely affected by exposure to and deposition from $PM_{2.5}$. $PM_{2.5}$ has been linked to adverse impacts on ecosystems, primarily through deposition of PM constituents. In addition, deposition of PM is associated with damages to materials and buildings.

3.4.1 Role of BC in Visibility Impairment

Particles are the dominant air pollutant responsible for visibility impairment, e.g. "haze," in both urban and remote areas. In the same way that particles influence the Earth's radiative balance, by scattering and/or absorbing solar radiation, they influence the quantity and quality of light received by the human eye and, therefore, one's ability to recognize and appreciate the form, contrast detail, and color of near and distant features. Aerosol-based light extinction can be estimated using the Interagency Monitoring of Protected Visual Environments (IMPROVE) algorithm that multiplies the ambient concentration of PM components by typical component-specific light extinction efficiencies.[5] BC and crustal minerals are the only included components that contribute to light absorption. Under low humidity conditions, BC and OC have the greatest effect on visibility among the major PM species. Per unit mass, the algorithm specifies that BC is 2.5 times more effective at absorbing light than organic carbon is at scattering.

Carbonaceous PM is responsible for a large fraction of regional haze, particularly in the Northwest, where annual average concentrations for 2000-2004 account for 40-60% of the aerosol based light extinction. Most of this average carbonaceous visibility impairment throughout the United States is associated with OC (in both rural and urban

[5] See *http://vista.cira.colostate.edu/improve*. For two major $PM_{2.5}$ components, sulfate and nitrate, water growth factors are included to account for enhanced light extinction due to relative humidity. The original IMPROVE equation included Rayleigh scattering (from natural atmospheric gasses) and factors for particulate sulfates, nitrates, organic carbon, elemental carbon, fine soil and coarse particles, with a hygroscopic growth function for enhanced light scattering from water associates with the sulfates and nitrates. A recently proposed revision to this equation (Pitchford et al., 2007) enhances the scattering from high concentrations of sulfates, nitrates or organics and adds terms for scattering and hygroscopic growth from sea salt and for light absorption from gaseous NO_2.

areas) because of relatively high OC concentrations compared to BC. Regional haze in the eastern United States generally contains even higher concentrations of carbonaceous PM and light-absorbing BC plays a relatively larger but still minor role compared to OC (DeBell, 2006).

As described further in Chapter 5, urban areas have more carbonaceous PM than nearby remote (rural) areas in the same region (U.S. EPA, 2004b). Western urban areas have more than twice the average concentrations of carbonaceous PM than remote areas in the same region (DeBell, 2006). As shown in Figure 5-6, average urban $PM_{2.5}$ is composed of roughly equal proportions of carbonaceous and sulfate components in some eastern areas. In conditions of high relative humidity common in the eastern United States, hydrated sulfate dominates as the constituent responsible for most urban haze on the haziest summer-time days (U.S. EPA, 2009b).

The 1977 Clean Air Act Amendments called for the development of regulations to address regional haze (visibility impairment) in 156 National Parks and wilderness areas in the United States. The EPA promulgated a Regional Haze Rule (RHR) in 1999 in response to this mandate. Implementation of the RHR entails planned emissions reductions to ensure that by 2064, the worst haze days in these protected areas will improve to natural conditions without degrading visibility conditions for the best haze days. In addition to the RHR aimed at achieving visibility improvements in protected National Park areas, the NAAQS program has been successful at achieving visibility improvements in rural areas, as well as in urban areas where people live and work.

3.4.2 Role of BC in Crop Damage and Other Environmental Impacts

Crop yields can be sensitive to the amount of sunlight received. As discussed in detail in Chapter 2, BC and other airborne particles contribute to surface dimming, and crop losses have been attributed to increased airborne particle concentrations in some areas of the world (Chameides et al., 1999). Auffhammer et al. (2006) found that fossil fuel and biomass burning contributes to reduced rice harvests in India. Decreases in rice and winter wheat yields have also been attributed to regional scale air pollution in China (Chameides et al., 1999).

Ecological effects of PM include direct effects to metabolic processes of plant foliage (Naidoo and Chirkoot, 2004; Kuki et al., 2008); contribution to total metal loading resulting in alteration of soil biogeochemistry (Burt et al., 2003; Ramos et al., 1994; Watmough et al., 2004), plant growth (Audet and Charest, 2007; Kucera et al., 2008; Strydom et al., 2006) and animal growth and reproduction (Gomot-de Vaufleury and Kerhoas, 2000; Regoli et al., 2006); and contribution to total organics loading resulting in bioaccumulation and biomagnification across trophic levels (Notten et al., 2005).

Building materials (metals, stones, cements, and paints) undergo natural weathering processes from exposure to environmental elements (wind, moisture, temperature fluctuations, sunlight, etc.). Deposition of PM is associated with both physical damage (materials damage effects) and impaired aesthetic qualities (soiling effects) for building materials. Wet and dry deposition of PM can physically affect materials, adding to the effects of natural weathering processes, by potentially promoting or accelerating corrosion of metals, by degrading paints and by deteriorating building materials (Haynie, 1986; Nazaroff and Cass, 1991). Fine particles may coat building materials, damaging the appearance of homes, public buildings, and historic landmarks (Hamilton and Mansfield, 1991). Studies have been conducted by a number of authors identifying the anthropogenic sources of soiling and materials damages to monuments and historical buildings (Sabbioni et al., 2003; Bonazza et al., 2005). For example, Bonazza evaluated deposition to the London Tower and found that "deposition of elemental carbon darkens surfaces and has importantly aesthetic implications for buildings." Reduction of PM deposition is beneficial in terms of reduced cleaning, maintenance, and restoration expenditures for buildings and structures.

3.5 Key Uncertainties Regarding Health/Environmental Impacts of BC

A review of the literature describing the health effects associated with ambient concentrations of BC indicates that the strongest relationship exists between BC and cardiovascular effects. This evidence includes support for a continuum of cardiovascular effects ranging from subtle subclinical measures to more severe effects on the cardiovascular system, such as emergency department visits and hospital admissions. These associations are generally consistent with the associations observed for $PM_{2.5}$ and cardiovascular effects (Janssen et al., 2011), though the body of evidence describing the association between BC and cardiovascular effects is much smaller and less consistent than the one characterizing $PM_{2.5}$ and cardiovascular effects. It is noteworthy that, among the studies that characterize the association between BC and cardiovascular effects, a large

majority have been conducted in the greater Boston, MA area, utilizing BC measurements from a single BC monitor (see Table 3-2). There can be substantial spatial variation in BC concentrations within a single city, and ambient concentrations of BC in any urban area can vary widely from location to location within the city. Thus, the reliance on this single monitor to estimate exposure for a number of studies across the entire greater Boston, MA area may contribute uncertainty to the reported associations. Similarly, the ambient concentration and composition of PM is geographically heterogeneous, with variations due to unique PM sources and from unique formation, transport, transformation, removal, and infiltration processes in different locations. Thus, a body of evidence that is focused on one geographic area, in this case Boston, MA, introduces uncertainty to the characterization of the association between ambient BC and cardiovascular effects, and the generalizability of this association to broader geographic areas.

An additional uncertainty regarding the health impacts of BC is the inconsistency between the results of studies examining ambient concentrations of BC and the results attributed to traffic in the HEI report (Health Effects Institute, 2010). In examining the body of evidence for health effects associated with BC, the strongest relationship was observed for BC and cardiovascular effects, while the evidence for an effect of BC on respiratory effects was observed to be inconsistent. Conversely, the HEI report on traffic (Health Effects Institute, 2010), concluded that evidence is "sufficient to infer the presence of a causal association" between traffic exposure and respiratory effects (i.e., exacerbation of childhood asthma symptoms), while the evidence for an association with cardiovascular effects was "suggestive." Thus, while BC is a known component of the air pollution mixture attributed to traffic sources, it may have a stronger association with some health effects attributed to traffic (i.e., respiratory effects) than others (i.e., cardiovascular effects). Furthermore, this line of reasoning indicates that there are likely additional components to the air pollution mixture attributed to traffic sources (other than BC) that contribute to the health effects associated with exposure to traffic. Additionally, BC could be serving as an indicator for a larger category of primary combustion particles, which, in addition to BC, can include trace metals and hydrocarbons such as PAHS, any or all of which could be acting to cause adverse health effects.