

Health Impacts of Diesel, Based on Data from the National-Scale Air Toxics Assessment (NATA)

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Prepared by:
Donald McCubbin, Ph.D.

Prepared for:
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1. Introduction

Diesel particles cause widespread damage to human health. This report estimates the impact of onroad and offroad sources of diesel particles.

The contribution of diesel particles to ambient particle levels less than or equal to 2.5 microns ($PM_{2.5}$) in aerodynamic diameter are from the National-Scale Air Toxics Assessment (NATA) program. The epidemiological studies and methods used to estimate the health impacts of diesel are the same as those used by the U.S. Environmental Protection Agency (EPA) in recent regulatory impact assessments (e.g., U.S. EPA 2008a; 2008b; 2009).

In Section 2, I briefly describe the studies and methods that I used to estimate the health impacts of diesel. Additional details on the studies are provided in Appendix A. And in Section 3, I briefly summarize the national-level health impacts.

2. Methods

To estimate the diesel PM_{2.5}-related human health impacts and value these impacts, I use version 4.0 of the Environmental Benefits Mapping and Analysis Program (BenMAP).¹ The first step in using BenMAP is to change in ambient air quality, in this case the contribution of direct diesel particles to annual average ambient PM_{2.5} levels. (No impacts of diesel-related NO_x and VOC emissions on ambient PM_{2.5} are considered.) Given the annual change in diesel PM_{2.5} concentrations, BenMAP calculates the associated change in adverse health effects, such as premature mortality. To estimate the economic value of these health effects, I used EPA unit values and performed the calculations with SAS (version 9.2).

2.1 Annual Average Ambient Diesel Concentrations

The NATA program estimated tract-level direct diesel particle contributions to ambient PM_{2.5} concentrations for onroad and offroad sources. A tract-level file was accessed from the NATA website (<http://www.epa.gov/ttn/atw/nata2002/tables.html>) and then formatted for use in BenMAP.

2.2 Estimating Cases of Diesel-Related Human Health Impacts

The first step in estimating health impacts involves the specification of health impact functions, which quantify the relationship between changes in air pollution and adverse health impacts. A typical health impact function for PM_{2.5} has four components:

- **Effect estimate.** An effect estimate (“beta”) quantifies the change in health effects per unit of change in PM_{2.5}, and is derived from an epidemiological study.
- **PM_{2.5} change.** The estimated change in the concentration of ambient PM_{2.5}.
- **Incidence rate.** The baseline incidence rate for the health effect due to all causes.
- **Population.** The affected population; the age range included depends on the ages included in the epidemiological study.

The typical log-linear health impact function looks as follows:

$$\Delta\text{Health} = \left(1 - \frac{1}{\exp(\text{Beta} * \Delta\text{PM}_{2.5})}\right) * \text{Incidence} * \text{Population}$$

Another common form for health impact functions is the logistic, which appears as follows:

$$\Delta\text{Health} = \left(1 - \frac{1}{(1 - \text{Incidence}) * \exp(\text{Beta} * \Delta\text{PM}_{2.5}) + \text{Incidence}}\right) * \text{Incidence} * \text{Population}$$

¹ The key difference between BenMAP versions 3.0 and 4.0 is that version 4.0 has updated mortality incidence rates based on rates for the period 2004-2006 (as opposed to 1996-1998).

All of the health impact functions I use are in one of these two main forms. Both types have the same four elements. Appendix A derives these two forms and provides additional details on individual studies. Table 1 presents the PM_{2.5}-related health endpoints included in this analysis.

Table 1. Epidemiological Studies Used to Estimate Adverse Health Impacts of Diesel Particles

Endpoint	Author	Age
Mortality, All Cause	Laden <i>et al</i>	25-99
Mortality, All Cause	Pope <i>et al</i> (2002)	30-99
Mortality, All Cause	Woodruff <i>et al</i> (1997)	Infant
Chronic Bronchitis	Abbey <i>et al</i> (1995c)	27-99
Heart Attack, Nonfatal	Peters <i>et al</i> (2001)	18-99
HA, All Cardiovascular (less Myocardial Infarctions)	Moolgavkar (2000b)	18-64
HA, All Cardiovascular (less Myocardial Infarctions)	Moolgavkar (2003)	65-99
HA, Congestive Heart Failure	Ito (2003)	65-99
HA, Dysrhythmia	Ito (2003)	65-99
HA, Ischemic Heart Disease (less Myocardial Infarctions)	Ito (2003)	65-99
HA, Pneumonia	Ito (2003)	65-99
HA, Chronic Lung Disease (less Asthma)	Moolgavkar (2000a)	18-64
HA, Chronic Lung Disease	Ito (2003)	65-99
HA, Chronic Lung Disease	Moolgavkar (2003)	65-99
HA, Asthma	Sheppard (2003)	0-64
Emergency Room Visits, Asthma	Norris <i>et al</i> (1999)	0-17
Acute Bronchitis	Dockery <i>et al</i> (1996)	8-12
Lower Respiratory Symptoms	Schwartz and Neas (2000)	7-14
Upper Respiratory Symptoms	Pope <i>et al</i> (1991)	9-11
Asthma Exacerbation, Cough	Ostro <i>et al</i> (2001)	6-18
Asthma Exacerbation, Shortness of Breath	Ostro <i>et al</i> (2001)	6-18
Asthma Exacerbation, Wheeze	Ostro <i>et al</i> (2001)	6-18
Asthma Exacerbation, Cough	Vedal <i>et al</i> (1998)	6-18
Work Loss Days (WLD)	Ostro (1987)	18-64
Minor Restricted Activity Days (MRAD)	Ostro and Rothschild (1989)	18-64

Note: HA = hospital admissions.

2.3 Valuing Estimated Health Impacts

Estimating the economic benefit of the estimated change in health incidence, I multiplied the number of adverse cases of a specific type of effect (e.g., mortality) by its associated unit value and then adjusted for the estimated change in income between 1990 and 2002:

$$\text{\$Benefit} = \text{Cases Health Effects} * \text{Unit Value} * \text{Income Adjustment}$$

Table 2 presents the mean estimate of the unit values used in this analysis. As described in the next sub-section, the approach I use to adjust for income follows the approach used by EPA in recent regulatory analyses. In addition to adjusting for income, I also adjust the mortality estimate to account for an assumed distribution of deaths over time. This mortality adjustment (described below) is also an approach used by EPA in recent regulatory analyses.

Table 2. Unit Values for Economic Valuation of Health Endpoints (based on 2002 income and 2008\$)

Health Endpoint	Age Range	Unit Value
Mortality*	0 – 99	\$8,300,000
Chronic Bronchitis	27 - 99	\$450,000
Acute Myocardial Infarction, Nonfatal**	0 - 24	\$92,000
Acute Myocardial Infarction, Nonfatal	25 - 44	\$103,000
Acute Myocardial Infarction, Nonfatal	45 - 54	\$109,000
Acute Myocardial Infarction, Nonfatal	55 - 64	\$190,000
Acute Myocardial Infarction, Nonfatal	65 - 99	\$92,000
HA, All Cardiovascular (less AMI)	18 - 64	\$31,700
HA, All Cardiovascular (less AMI)	65 - 99	\$29,500
HA, Congestive Heart Failure	65 - 99	\$21,200
HA, Dysrhythmia	65 - 99	\$21,200
HA, Ischemic Heart Disease (less AMI)	65 - 99	\$36,100
HA, Pneumonia	65 - 99	\$24,800
HA, Chronic Lung Disease (less Asthma)	18-64	\$17,200
HA, Chronic Lung Disease	65 - 99	\$18,700
HA, Asthma	0 - 64	\$10,800
Asthma ER Visits***	0 - 17	\$399
Acute Bronchitis	8 - 12	\$470
Lower Resp. Symptoms	7 - 14	\$20
Upper Resp. Symptoms	9 - 11	\$31
Asthma Exacerbation, Cough	6 - 18	\$54
Asthma Exacerbation, Shortness of Breath	6 - 18	\$54
Asthma Exacerbation, Wheeze	6 - 18	\$54
Work Loss Days (WLD)	18 - 64	\$161
Minor Restricted Activity Days (MRAD)	18 - 64	\$67

NOTE: Numbers rounded to three significant digits. HA = hospital admissions. * Mortality value after adjustment for 20-year lag. **The age-specific acute myocardial infarction unit values are based on an average of two estimates: one based on Russell (1998) and one based on Wittels (1990). ** The asthma ER visit value is an average of two estimates: one based on Smith et al (1997) and the other based on Stanford et al (1999). **** County-specific median daily wage.

Income Adjustment

There is evidence that as people’s income increases, their willingness to pay (WTP) to avoid adverse health impacts also increases. Economists estimate “elasticities” to describe by what percent WTP goes up for a given percentage increase in income. As it turns out, these estimated elasticities are much less than one, however, there is considerable uncertainty over the precise value. I follow the approach used by EPA in recent regulatory analyses (U.S. EPA 2008b), which used elasticity estimates that vary by type of health effect, with relatively minor effects having a smaller elasticity than more severe effects.

Table 3. Elasticity of WTP by Type of Health Effect

Health Effect	Central Elasticity Estimate
Minor Health Effect	0.14
Severe & Chronic Health Effects	0.45
Premature Mortality	0.4

Source: EPA (2005, p. 4-18).

Multiplying these elasticities by historical and forecasted income data, EPA developed income adjustment factors which I use in this report. Table 4 presents the year 2002 income adjustment factors that I use, along with the intervening years between 1990 (the assumed income year for the valuation estimates) and 2002 (the year of interest).

Table 4. Income Adjustment Factors by Type of Health Effect

Year	Mortality	Severe	Minor
1990	1.000	1.000	1.000
1991	0.992	0.991	0.997
1992	0.998	0.998	0.999
1993	1.003	1.003	1.001
1994	1.013	1.014	1.004
1995	1.017	1.019	1.006
1996	1.024	1.027	1.008
1997	1.034	1.039	1.012
1998	1.039	1.044	1.013
1999	1.043	1.048	1.015
2000	1.039	1.043	1.013
2001	1.044	1.049	1.015
2002	1.050	1.056	1.017

Note that because of a lack of data on the dependence of COI on income, and a lack of data on projected growth in average wages, no adjustments are made to benefits estimates based on the COI approach or to work loss days and worker productivity benefits estimates. This lack of adjustment would tend to result in an under-prediction of benefits in future years, because it is likely that increases in real U.S. income would also result in increased COI (due, for example, to increases in wages paid to medical workers) and increased cost of work loss days and lost worker productivity (reflecting that if worker incomes are higher, the losses resulting from reduced worker production would also be higher).

Mortality Adjustment

The delay, or lag, between changes in PM exposures and changes in mortality rates is not precisely known. The current scientific literature on adverse health effects, such as those associated with PM

(e.g., smoking-related disease), and the difference in the estimated effect of chronic exposure studies versus daily mortality studies, suggests that it is likely that not all cases of avoided premature mortality associated with a given incremental reduction in PM exposure would occur in the same year as the exposure reduction.

Following recent EPA analyses (U.S. EPA 2006, p. 5-21), I assume a 20-year lag structure, with 30 percent of premature deaths occurring in the first year, 50 percent occurring evenly over years 2 to 5 after the reduction in PM_{2.5}, and 20 percent occurring evenly over years 6 to 20 after the reduction in PM_{2.5}. It should be noted that the selection of a 20-year lag structure is not directly supported by any PM-specific literature. Rather, it is intended to be a reasonable estimate of the appropriate time distribution of avoided cases of PM-related mortality. As noted by EPA, the distribution of deaths over the latency period is intended to reflect the contribution of short-term exposures in the first year, cardiopulmonary deaths in the 2- to 5-year period, and long-term lung disease and lung cancer in the 6- to 20-year period. Finally, it is important to keep in mind that changes in the lag assumptions do *not* change the total number of estimated deaths but rather the timing of those deaths.

Specifying the lag is important because people are generally willing to pay more for something now than for the same thing later. They would, for example, be willing to pay more for a reduction in the risk of premature death in the same year as exposure is reduced than for that same risk reduction to be received the following year. This time preference for receiving benefits now rather than later is expressed by discounting benefits received later. The exact discount rate that is appropriate (i.e., that represents people's time preference) is a topic of much debate. EPA has often used a discount rate of three percent, and I use a three percent rate for this analysis in conjunction with the 20-year lag structure described above.

3. Results

Table 5 summarizes the onroad and offroad diesel impacts. Details on the calculations are provided in Chapter 2 and Appendix A.

Table 5. Onroad & Offroad Diesel Health Impacts in 2002

Health Impact	OnRoad Diesel		OffRoad Diesel	
	Cases	Value (million 2008 \$)	Cases	Value (million 2008 \$)
Mortality, Adult (Laden et al, 2006)	11,200	\$84,200	23,900	\$180,000
Mortality, Adult (Pope et al, 2002)	4,360	\$32,800	9,350	\$70,300
Mortality, Infant	39	\$324	86	\$710
Chronic Bronchitis	2,880	\$1,300	6,250	\$2,810
Heart Attack, Nonfatal	6,330	\$727	13,500	\$1,550
HA, All Cardiovascular	2,020	\$58	4,360	\$126
HA, Respiratory	1,470	\$27	3,180	\$58
ER Visits, Asthma	3,920	\$2	8,500	\$3
Acute Bronchitis	7,870	\$4	16,900	\$8
Lower Resp. Symptoms	93,800	\$2	202,000	\$4
Upper Resp. Symptoms	71,300	\$2	156,000	\$5
Asthma Exacerbation	154,000	\$8	336,000	\$18
WLD	616,000	\$99	1,350,000	\$217
MRAD	3,590,000	\$240	7,850,000	\$526

Note: HA = hospital admissions. ER = emergency room. WLD = work loss days. MRAD = minor restricted activity days. Results rounded to three digits.

Appendix A. Human Health Impact Function Details

This appendix presents the derivation of the two main health impact functions used in this analysis (log-linear and logistic), as well as details on each function used.

A.1 Deriving Health Impact Functions

Below, I present a derivation of the mean coefficient estimates for log-linear and logistic health impact functions.

Log-Linear Derivation

$y_0 = \text{Incidence under baseline conditions}$

$y_c = \text{Incidence under control conditions}$

$$\Delta y = y_0 - y_c$$

$PM_0 = PM \text{ levels under baseline conditions}$

$PM_c = PM \text{ levels under control conditions}$

$$\Delta PM = PM_0 - PM_c$$

$$\ln(y) = \alpha + \beta PM$$

$$y = Be^{\beta PM}$$

$$y_0 = Be^{\beta PM_0}$$

$$y_c = Be^{\beta PM_c}$$

$$\Delta y = Be^{\beta PM_0} - Be^{\beta PM_c}$$

$$\Delta y = Be^{\beta PM_0} \cdot \left(1 - \frac{Be^{\beta PM_c}}{Be^{\beta PM_0}} \right)$$

$$\Delta y = Be^{\beta PM_0} \cdot \left(1 - e^{\beta \cdot (PM_c - PM_0)}\right)$$

$$\Delta y = Be^{\beta PM_0} \cdot \left(1 - e^{-\beta \Delta PM}\right)$$

$$\Delta y = y_0 \cdot \left(1 - e^{-\beta \Delta PM}\right)$$

$$\Delta y = y_0 \cdot \left(1 - \frac{1}{e^{\beta \Delta PM}}\right)$$

Logistic Derivation

y_0 = Incidence under baseline conditions

y_c = Incidence under control conditions

$$\Delta y = y_0 - y_c$$

PM_0 = PM levels under baseline conditions

PM_c = PM levels under control conditions

$$\Delta PM = PM_0 - PM_c$$

X = vector of explanatory variables

B = vector of coefficients

β = coefficient of the PM variable

$$y = \left(\frac{e^{XB}}{1 + e^{XB}}\right) = \frac{1}{1 + e^{-XB}}$$

$$odds = \frac{y}{1 - y} = \frac{\left(\frac{1}{1 + e^{-XB}}\right)}{1 - \left(\frac{1}{1 + e^{-XB}}\right)}$$

$$odds = \frac{\left(\frac{1}{1 + e^{-XB}}\right)}{\left(\frac{e^{-XB}}{1 + e^{-XB}}\right)} = \frac{1}{e^{-XB}} = e^{XB}$$

$$odds\ ratio = \frac{e^{X_0 B}}{e^{X_c B}} = \frac{e^{\gamma} \cdot e^{PM_0 \beta}}{e^{\gamma} \cdot e^{PM_c \beta}} = e^{\Delta PM \beta}$$

$$\left(\frac{y_0}{1-y_0}\right) = e^{\Delta PM\beta}$$

$$\left(\frac{y_c}{1-y_c}\right)$$

$$\left(\frac{y_0}{1-y_0}\right) \cdot e^{-\Delta PM\beta} = \frac{y_c}{1-y_c}$$

$$y_c = (1-y_c) \cdot \left(\frac{y_0}{1-y_0}\right) \cdot e^{-\Delta PM\beta}$$

$$y_c + y_c \cdot \left(\frac{y_0}{1-y_0}\right) \cdot e^{-\Delta PM\beta} = \left(\frac{y_0}{1-y_0}\right) \cdot e^{-\Delta PM\beta}$$

$$y_c \cdot \left[1 + \left(\frac{y_0}{1-y_0}\right) \cdot e^{-\Delta PM\beta}\right] = \left(\frac{y_0}{1-y_0}\right) \cdot e^{-\Delta PM\beta}$$

$$y_c = \frac{\left(\frac{y_0}{1-y_0}\right) \cdot e^{-\Delta PM\beta}}{1 + \left(\frac{y_0}{1-y_0}\right) \cdot e^{-\Delta PM\beta}}$$

$$y_c = \frac{y_0 \cdot e^{-\Delta PM\beta}}{1 - y_0 + y_0 \cdot e^{-\Delta PM\beta}}$$

$$y_c = \frac{y_0}{(1-y_0) \cdot e^{\Delta PM\beta} + y_0}$$

$$y_0 - y_c = y_0 - \frac{y_0}{(1-y_0) \cdot e^{\Delta PM\beta} + y_0}$$

$$\Delta y = y_0 \cdot \left(1 - \frac{1}{(1-y_0) \cdot e^{\Delta PM\beta} + y_0}\right)$$

A.2 PM_{2.5} Health Impact Functions

This analysis uses a range of health impact functions, including those to estimate premature mortality, chronic bronchitis, and hospital admissions. These health impact functions are the same ones used in recent EPA regulatory impact analyses (e.g., U.S. EPA 2008b).

Note that the input to BenMAP is the annual average contribution of diesel particles to population exposure of PM_{2.5}. To estimate the change in health effects associated with *daily* changes in PM_{2.5}, BenMAP assumes that the annual change is a reasonable proxy and multiplies the result by 365. Since the health impact functions are reasonably linear, the effect of this assumption is small, generally within a few percent, even for fairly extreme assumptions.

Below, I present a table with the health impact functions used to estimate PM_{2.5}-related adverse health effects. Following this table, I present a brief summary of each of the studies along with details not in the summary table.

Table 6. Details of PM_{2.5} Human Health Impact Functions

Endpoint Name	Study	Location	Age	Beta	Std Error	Functional Form
Adult mortality	Laden <i>et al</i> (2002)	6 cities	25-99	0.014842	0.004170	Log-linear
Adult mortality	Pope <i>et al</i> (2002)	51 cities	30-99	0.005827	0.002157	Log-linear
Infant mortality	Woodruff <i>et al</i> (1997)	86 cities	0-0	0.003922	0.001221	Logistic
Chronic Bronchitis	Abbey <i>et al</i> (1995c)	California	27-99	0.013185	0.006796	Logistic
Heart Attack, Nonfatal	Peters <i>et al</i> (2001)	Boston, MA	18-99	0.024121	0.009285	Logistic
Congestive Heart Failure	Ito (2003)	Detroit, MI	65-99	0.003074	0.001292	Log-linear
Dysrhythmia	Ito (2003)	Detroit, MI	65-99	0.001249	0.002033	Log-linear
Ischemic Heart Disease (less AMI)	Ito (2003)	Detroit, MI	65-99	0.001435	0.001156	Log-linear
Chronic Lung Disease	Ito (2003)	Detroit, MI	65-99	0.001169	0.002064	Log-linear
Pneumonia	Ito (2003)	Detroit, MI	65-99	0.003979	0.001659	Log-linear
All Cardiovascular (less AMI)	Moolgavkar (2000b)	Los Angeles, CA	18-64	0.001400	0.000341	Log-linear
Chronic Lung Disease (less Asthma)	Moolgavkar (2000b)	Los Angeles, CA	18-64	0.002200	0.000733	Log-linear
All Cardiovascular (less AMI)	Moolgavkar (2003)	Los Angeles, CA	65-99	0.001580	0.000344	Log-linear
Chronic Lung Disease	Moolgavkar (2003)	Los Angeles, CA	65-99	0.001850	0.000524	Log-linear
Asthma	Sheppard (2003)	Seattle, WA	0-64	0.003324	0.001045	Log-linear
Emergency Room Visits, Asthma	Norris <i>et al</i> (1999)	Seattle, WA	0-17	0.016527	0.004139	Log-linear
Minor Restricted Activity Days (MRAD)	Ostro & Rothschild (1989)	Nationwide	18-64	0.007410	0.000700	Log-linear
Acute Bronchitis	Dockery <i>et al</i> (1996)	24 communities	8-12	0.027212	0.017096	Logistic
Work Loss Days (WLD)	Ostro (1987)	Nationwide	18-64	0.004600	0.000360	Log-linear
Lower Respiratory Symptoms	Schwartz and Neas (2000)	6 U.S. cities	7-14	0.019012	0.006005	Logistic
Asthma Exacerbation, Cough	Ostro <i>et al</i> (2001)	Los Angeles, CA	6-18	0.000985	0.000747	Logistic
Asthma Exacerbation, Shortness of Breath	Ostro <i>et al</i>	Los Angeles, CA	6-18	0.002565	0.001335	Logistic
Asthma Exacerbation, Wheeze	Ostro <i>et al</i>	Los Angeles, CA	6-18	0.001942	0.000803	Logistic
Asthma Exacerbation, Cough	Vedal <i>et al</i> (1998)	Vancouver, CAN	6-18	0.007696	0.003786	Logistic
Upper Respiratory Symptoms	Pope <i>et al</i> (1991)	Utah Valley	9-11	0.0036	0.0015	Logistic

Mortality, All Cause (Laden *et al.* 2006)

The Laden *et al.* (2002) analysis is a longitudinal cohort tracking study that uses the same six city cohort as the original Dockery *et al.* (1993) study, and the Krewski *et al.* (2000) reanalysis. A key difference is that the Laden *et al.* study used a longer follow-up period.

The coefficient and standard error for PM_{2.5} are estimated from the relative risk (1.16) and 95% confidence interval (1.07-1.26) associated with a change in annual mean exposure of 10.0 µg/m³ (Laden *et al.* 2006, p. 667).

Functional Form: Log-linear

Coefficient: 0.014842

Standard Error: 0.004170

Incidence Rate: county-specific annual all cause mortality rate per person ages 25 and older

Population: population of ages 25 and older.

Mortality, All Cause (Pope *et al.* 2002)

The Pope *et al.* (2002) analysis is a longitudinal cohort tracking study that uses the same American Cancer Society cohort as the original Pope *et al.* (1995) study, and the Krewski *et al.* (2000) reanalysis. Pope *et al.* (2002) analyzed survival data for the cohort from 1982 through 1998, 9 years longer than the original Pope study. Pope *et al.* (2002) followed Krewski *et al.* (2000) and Pope *et al.* (1995, Table 2) and reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-519), and “all other” deaths.² Like the earlier studies, Pope *et al.* (2002) found that mean PM_{2.5} is significantly related to all-cause and cardiopulmonary mortality. In addition, Pope *et al.* (2002) found a significant relationship with lung cancer mortality, which was not found in the earlier studies. None of the three studies found a significant relationship with “all other” deaths.

The coefficient and standard error for PM_{2.5} using the average of '79-'83 and '99-'00 PM data are estimated from the relative risk (1.06) and 95% confidence interval (1.02-1.11) associated with a change in annual mean exposure of 10 µg/m³. Pope *et al.* (2002, Table 2).

Functional Form: Log-linear

Coefficient: 0.005827

Standard Error: 0.002157

Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older

Population: population of ages 30 and older.

Infant Mortality (Woodruff *et al.* 1997)

In a study of four million infants in 86 U.S. metropolitan areas conducted from 1989 to 1991, Woodruff *et al.* (1997) found a significant link between PM₁₀ exposure in the first two months of an infant's life with the probability of dying between the ages of 28 days and 364 days. PM₁₀ exposure was significant for all-cause mortality. PM₁₀ was also significant for respiratory mortality in average birth-weight infants, but not low birth-weight infants.

² All-cause mortality includes accidents, suicides, homicides and legal interventions. The category “all other” deaths is all-cause mortality less lung cancer and cardiopulmonary deaths.

The coefficient and standard error are based on the odds ratio (1.04) and 95% confidence interval (1.02-1.07) associated with a 10 $\mu\text{g}/\text{m}^3$ change in PM₁₀ (Woodruff et al. 1997, Table 3).

Functional Form: Logistic

Coefficient: 0.003922

Standard Error: 0.001221

Incidence Rate: county-specific annual post-neonatal³ infant deaths per infant under the age of one

Population: population of infants under one year old.

Chronic Bronchitis (Abbey et al. 1995b)

Abbey et al (1995b) examined the relationship between estimated PM_{2.5} (annual mean from 1966 to 1977), PM₁₀ (annual mean from 1973 to 1977) and TSP (annual mean from 1973 to 1977) and the same chronic respiratory symptoms in a sample population of 1,868 Californian Seventh Day Adventists. The initial survey was conducted in 1977 and the final survey in 1987. To ensure a better estimate of exposure, the study participants had to have been living in the same area for an extended period of time. In single-pollutant models, there was a statistically significant PM_{2.5} relationship with development of chronic bronchitis, but not for AOD or asthma; PM₁₀ was significantly associated with chronic bronchitis and AOD; and TSP was significantly associated with all cases of all three chronic symptoms. Other pollutants were not examined.

The estimated coefficient (0.0137) is presented for a one $\mu\text{g}/\text{m}^3$ change in PM_{2.5} (Abbey et al. 1995b, Table 2). The standard error is calculated from the reported relative risk (1.81) and 95% confidence interval (0.98-3.25) for a 45 $\mu\text{g}/\text{m}^3$ change in PM_{2.5} (Abbey et al. 1995b, Table 2).

Functional Form: Logistic

Coefficient: 0.0137

Standard Error: 0.00680

Incidence Rate: annual bronchitis incidence rate per person (Abbey et al. 1993, Table 3) = 0.00378

Population: population of ages 27 and older⁴ without chronic bronchitis = 95.57% of population 27+.⁵

Acute Myocardial Infarction (Heart Attacks), Nonfatal (Peters et al. 2001)

Peters et al (2001) studied the relationship between increased particulate air pollution and onset of heart attacks in the Boston area from 1995 to 1996. The authors used air quality data for PM₁₀, PM_{10-2.5}, PM_{2.5}, "black carbon", O₃, CO, NO₂, and SO₂ in a case-crossover analysis. For each subject, the case period was matched to three control periods, each 24 hours apart. In univariate analyses, the authors observed a positive association between heart attack occurrence and PM_{2.5} levels hours before and days before onset. The authors estimated multivariate conditional logistic models including two-hour and twenty-four hour pollutant concentrations for each pollutant. They found significant and independent associations between heart attack occurrence and both two-hour and twenty-four hour PM_{2.5} concentrations before onset. Significant associations were observed for PM₁₀ as well. None of the other

³ Post-neonatal refers to infants that are 28 days to 364 days old.

⁴ Using the same data set, Abbey et al (1995a, p.140) reported the respondents in 1977 ranged in age from 27 to 95.

⁵ The American Lung Association (2002b, Table 4) reports a chronic bronchitis prevalence rate for ages 18 and over of 4.43% (American Lung Association 2002b).

particle measures or gaseous pollutants were significantly associated with acute myocardial infarction for the two hour or twenty-four hour period before onset.

The mean age of participants was 62 years old, with 21% of the study population under the age of 50. In order to capture the full magnitude of heart attack occurrence potentially associated with air pollution and because age was not listed as an inclusion criteria for sample selection, BenMAP assumes an age range of 18 and over in the health impact function. According to the National Hospital Discharge Survey, there were no hospitalizations for heart attacks among children <15 years of age in 1999 and only 5.5% of all hospitalizations occurred in 15-44 year olds (Popovic 2001, Table 10).

The coefficient and standard error are calculated from an odds ratio of 1.62 (95% CI 1.13-2.34) for a 20 $\mu\text{g}/\text{m}^3$ increase in twenty-four hour average $\text{PM}_{2.5}$ (Peters et al. 2001, Table 4, p. 2813).

Functional Form: Logistic

Coefficient: 0.024121

Standard Error: 0.009285

Incidence Rate: region-specific daily nonfatal heart attack rate per person 18+ = 93% of region-specific daily heart attack hospitalization rate (ICD code 410)⁶

Population: population of ages 18 and older.

Hospital Admissions for Asthma (Sheppard *et al.* 1999; 2003)

Sheppard *et al.* (1999) studied the relation between air pollution in Seattle and nonelderly (ages <65) hospital admissions for asthma from 1987 to 1994. They used air quality data for PM_{10} , $\text{PM}_{2.5}$, coarse $\text{PM}_{10-2.5}$, SO_2 , ozone, and CO in a Poisson regression model with control for time trends, seasonal variations, and temperature-related weather effects. They found asthma hospital admissions associated with PM_{10} , $\text{PM}_{2.5}$, $\text{PM}_{10-2.5}$, CO, and ozone. They did not observe an association for SO_2 . They found PM and CO to be jointly associated with asthma admissions. The best fitting co-pollutant models were found using ozone. However, ozone data was only available April through October, so they did not consider ozone further. For the remaining pollutants, the best fitting models included $\text{PM}_{2.5}$ and CO. Results for other co-pollutant models were not reported.

In response to concerns that the work by Sheppard *et al.* (1999) may be biased because of the Splus issue, Sheppard (2003) reanalyzed some of this work, in particular Sheppard reanalyzed the original study's $\text{PM}_{2.5}$ single pollutant model.

The coefficient and standard error are based on the relative risk (1.04) and 95% confidence interval (1.01-1.06) for a 11.8 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ in the 1-day lag GAM stringent model (Sheppard 2003, pp. 228-299).

⁶ This estimate assumes that all heart attacks that are not instantly fatal will result in a hospitalization. In addition, Rosamond *et al.* (1999) report that approximately six percent of male and eight percent of female hospitalized heart attack patients die within 28 days (either in or outside of the hospital). I assume a factor of 0.93 to the number of hospitalizations to estimate the number of nonfatal heart attacks per year.

Functional Form: Log-linear

Coefficient: 0.003324

Standard Error: 0.001045

Incidence Rate: region-specific daily hospital admission rate for asthma admissions per person <65 (ICD code 493)

Population: population of ages 65 and under.

Hospital Admissions for Chronic Lung Disease (2000a; 2003)

Moolgavkar (2000a) examined the association between air pollution and COPD hospital admissions (ICD 490-496) in the Chicago, Los Angeles, and Phoenix metropolitan areas. He collected daily air pollution data for ozone, SO₂, NO₂, CO, and PM₁₀ in all three areas. PM_{2.5} data was available only in Los Angeles. The data were analyzed using a Poisson regression model with generalized additive models to adjust for temporal trends. Separate models were run for 0 to 5 day lags in each location. Among the 65+ age group in Chicago and Phoenix, weak associations were observed between the gaseous pollutants and admissions. No consistent associations were observed for PM₁₀. In Los Angeles, marginally significant associations were observed for PM_{2.5}, which were generally lower than for the gases. In co-pollutant models with CO, the PM_{2.5} effect was reduced. Similar results were observed in the 0-19 and 20-64 year old age groups.

In response to concerns with the Splus issue, Moolgavkar (2003) reanalyzed his earlier study. In the reanalysis, he reported that more generalized additive models with stringent convergence criteria and generalized linear models resulted in smaller relative risk estimates.

The PM_{2.5} C-R functions for the 65+ age group are based on the reanalysis in Moolgavkar (2003) of the single-pollutant model. The PM_{2.5} C-R functions for the 20-64 age group are based on the original study's single-pollutant model. Since the true PM effect is most likely best represented by a distributed lag model, then any single lag model should underestimate the total PM effect. As a result, the lag models with the greatest effect estimates was selected for use in the C-R functions.

Ages 18 to 64 (Moolgavkar 2000a)

The single pollutant coefficient and standard error are calculated from an estimated percent change of 2.2 and t-statistic of 3.0 for a 10 µg/m³ increase in PM_{2.5} in the two-day lag model (Moolgavkar 2000a, Table 4).

Functional Form: Log-linear

Coefficient: 0.0022

Standard Error: 0.000733

Incidence Rate: region-specific daily hospital admission rate for chronic lung disease admissions per person 18-64 (ICD codes 490-492, 494-496)⁷

Population: population of ages 18 to 64.⁸

⁷ Moolgavkar (2000a) reports results for ICD codes 490-496. In order to avoid double counting non-elderly asthma hospitalizations (ICD code 493), EPA excludes ICD code 493 from the baseline incidence rate used in this function.

Ages 65 and older (Moolgavkar 2003)

The coefficient and standard error are calculated from an estimated percentage change of 1.85 and t-statistic of 3.53 for a 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ in the 2-day lag GAM-30df stringent (10^{-8}) model (Moolgavkar 2003, Table 17).

Functional Form: Log-linear

Coefficient: 0.001833

Standard Error: 0.000519

Incidence Rate: region-specific daily hospital admission rate for chronic lung disease admissions per person 65+ (ICD codes 490-496)

Population: population of ages 65 and older.

Hospital Admissions for All Cardiovascular (Moolgavkar 2000b; 2003)

Moolgavkar (2000b) examined the association between air pollution and cardiovascular hospital admissions (ICD 390-448) in the Chicago, Los Angeles, and Phoenix metropolitan areas. He collected daily air pollution data for ozone, SO_2 , NO_2 , CO, and PM_{10} in all three areas. $\text{PM}_{2.5}$ data was available only in Los Angeles. The data were analyzed using a Poisson regression model with generalized additive models to adjust for temporal trends. Separate models were run for 0 to 5 day lags in each location. Among the 65+ age group, the gaseous pollutants generally exhibited stronger effects than PM_{10} or $\text{PM}_{2.5}$. The strongest overall effects were observed for SO_2 and CO. In a single pollutant model, $\text{PM}_{2.5}$ was statistically significant for lag 0 and lag 1. In co-pollutant models with CO, the $\text{PM}_{2.5}$ effect dropped out and CO remained significant. For ages 20-64, SO_2 and CO exhibited the strongest effect and any $\text{PM}_{2.5}$ effect dropped out in co-pollutant models with CO.

In response to concerns with the Splus issue, Moolgavkar (2003) reanalyzed his earlier study. In the reanalysis, he reported that more generalized additive models with stringent convergence criteria and generalized linear models resulted in smaller relative risk estimates. Not all of the original results were replicated, so BenMAP uses a mix of health impact functions from the reanalysis and from the original study (when the reanalyzed results were not available). The $\text{PM}_{2.5}$ C-R functions are based on single pollutant and co-pollutant ($\text{PM}_{2.5}$ and CO) models.

Note that Moolgavkar (2000b) reported results that include ICD code 410 (heart attack). I estimate avoided nonfatal heart attacks using the results reported by Peters *et al* (2001). In order to avoid double counting heart attack hospitalizations, ICD code 410 was excluded from the baseline incidence rate used in this function.

⁸ Although Moolgavkar (2000a) reports results for the 20-64 year old age range, for comparability to other studies, we apply the results to the population of ages 18 to 64.

Ages 18 to 64 (Moolgavkar 2000a)

The single pollutant coefficient and standard error are calculated from an estimated percent change of 1.4 and t-statistic of 4.1 for a 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ in the zero lag model (Moolgavkar 2000b, Table 4).

Functional Form: Log-linear

Coefficient: 0.0014

Standard Error: 0.000341

Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions per person ages 18 to 64 (ICD codes 390-409, 411-429)

Population: population of ages 18 to 64.⁹

Ages 65 and older (Moolgavkar 2003)

The single pollutant coefficient and standard error are calculated from an estimated percent change of 1.58 and t-statistic of 4.59 for a 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ in the 0-day lag GAM-30df stringent (10^{-8}) model (Moolgavkar 2003, Table 12).

Functional Form: Log-linear

Coefficient: 0.001568

Standard Error: 0.000342

Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions per person 65+ (ICD codes 390-409, 411-429)

Population: population of ages 65 and older.

Hospital Admissions for Respiratory & Cardiovascular Causes (Ito 2003)

Lippmann *et al* (2000) studied the association between particulate matter and daily mortality and hospitalizations among the elderly in Detroit, MI. Data were analyzed for two separate study periods, 1985-1990 and 1992-1994. The 1992-1994 study period had a greater variety of data on PM size and was the main focus of the report. The authors collected hospitalization data for a variety of cardiovascular and respiratory endpoints. They used daily air quality data for PM_{10} , $\text{PM}_{2.5}$, and $\text{PM}_{10-2.5}$ in a Poisson regression model with generalized additive models (GAM) to adjust for nonlinear relationships and temporal trends. In single pollutant models, all PM metrics were statistically significant for pneumonia (ICD codes 480-486), $\text{PM}_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $\text{PM}_{2.5}$ and PM_{10} were significant for heart failure (ICD code 428). There were positive, but not statistically significant associations, between the PM metrics and COPD (ICD codes 490-496) and dysrhythmia (ICD code 427). In separate co-pollutant models with PM and either ozone, SO_2 , NO_2 , or CO, the results were generally comparable. The $\text{PM}_{2.5}$ C-R functions are based on results of the single pollutant model and co-pollutant model with ozone.

⁹ Although Moolgavkar (2000a) reports results for the 20-64 year old age range, for comparability to other studies, we apply the results to the population of ages 18 to 64.

In response to concerns with the Splus issue, Ito (2003) reanalyzed the study by Lippmann *et al* (2000). The reanalysis by Ito reported that more generalized additive models with stringent convergence criteria and generalized linear models resulted in smaller relative risk estimates.

Chronic Lung Disease

The coefficient and standard error are based on the relative risk (1.043) and 95% confidence interval (0.902-1.207) for a 36 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ in the 3-day lag GAM stringent model (Ito 2003, Table 8).

Functional Form: Log-linear

Coefficient: 0.001169

Standard Error: 0.002064

Incidence Rate: region-specific daily hospital admission rate for chronic lung disease admissions per person 65+ (ICD codes 490-496)

Population: population of ages 65 and older.

Pneumonia

The estimated $\text{PM}_{2.5}$ coefficient and standard error are based on a relative risk of 1.154 (95% CI -1.027, 1.298) due to a $\text{PM}_{2.5}$ change of 36 $\mu\text{g}/\text{m}^3$ in the 1-day lag GAM stringent model (Ito 2003, Table 7).

Functional Form: Log-linear

Coefficient: 0.003979

Standard Error: 0.001659

Incidence Rate: region-specific daily hospital admission rate for pneumonia admissions per person 65+ (ICD codes 480-487)

Population: population of ages 65 and older.

Dysrhythmia

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.046 (95% CI 0.906-1.207) for a 36 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ in the 1-day lag GAM stringent model (Ito 2003, Table 10).

Functional Form: Log-linear

Coefficient: 0.001249

Standard Error: 0.002033

Incidence Rate: region-specific daily hospital admission rate for dysrhythmia admissions per person 65+ (ICD code 427)

Population: population of ages 65 and older.

Congestive Heart Failure

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.117 (95% CI 1.020-1.224) for a 36 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ in the 1-day lag GAM stringent model (Ito 2003, Table 11).

Functional Form: Log-linear

Coefficient: 0.003074

Standard Error: 0.001292

Incidence Rate: region-specific daily hospital admission rate for congestive heart failure admissions per person 65+ (ICD code 428)

Population: population of ages 65 and older.

Ischemic Heart Disease

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.053 (95% CI 0.971-1.143) for a 36 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ in the 1-day lag GAM stringent model (Ito 2003, Table 9).

Functional Form: Log-linear

Coefficient: 0.001435

Standard Error: 0.001156

Incidence Rate: region-specific daily hospital admission rate for ischemic heart disease admissions per person 65+ (ICD codes 411-414)¹⁰

Population: population of ages 65 and older.

Emergency Room Visits for Asthma (Norris et al. 1999)

Norris *et al* (1999) examined the relation between air pollution in Seattle and childhood (<18) hospital admissions for asthma from 1995 to 1996. The authors used air quality data for PM_{10} , light scattering (used to estimate fine PM), CO, SO_2 , NO_2 , and O_3 in a Poisson regression model with adjustments for day of the week, time trends, temperature, and dew point. They found significant associations between asthma ER visits and light scattering (converted to $\text{PM}_{2.5}$), PM_{10} , and CO. No association was found between O_3 , NO_2 , or SO_2 and asthma ER visits, although O_3 had a significant amount of missing data. In multipollutant models with either PM metric (light scattering or PM_{10}) and NO_2 and SO_2 , the PM coefficients remained significant while the gaseous pollutants were not associated with increased asthma ER visits.

In a model with NO_2 and SO_2 , the $\text{PM}_{2.5}$ coefficient and standard error are calculated from a relative risk of 1.17 (95% CI 1.08-1.26) for a 9.5 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ (Norris et al. 1999, p. 491).

Functional Form: Log-linear

Coefficient: 0.016527

Standard Error: 0.004139

Incidence Rate: region-specific daily emergency room rate for asthma admissions per person <18 (ICD code 493)

Population: population of ages under 18.

Acute Bronchitis (Dockery et al. 1996)

Dockery *et al* (1996) examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a study of 13,369 children ages 8-12

¹⁰ Lippmann *et al* (2000) reports results for ICD codes 410-414. EPA estimates avoided nonfatal heart attacks using the results reported by Peters *et al* (2001). In order to avoid double counting heart attack hospitalizations, ICD code 410 was excluded from the baseline incidence rate used in this function.

living in 24 communities in U.S. and Canada. Health data were collected in 1988-1991, and single-pollutant models were used in the analysis to test a number of measures of particulate air pollution. Dockery *et al* found that annual level of sulfates and particle acidity were significantly related to bronchitis, and $PM_{2.1}$ and PM_{10} were marginally significantly related to bronchitis.¹¹ They also found nitrates were linked to asthma, and sulfates linked to chronic phlegm. It is important to note that the study examined annual pollution exposures, and the authors did not rule out that acute (daily) exposures could be related to asthma attacks and other acute episodes.

Bronchitis was counted in the study only if there were “reports of symptoms in the past 12 months”(Dockery et al. 1996, p. 501). It is unclear, however, if the cases of bronchitis are acute and temporary, or if the bronchitis is a chronic condition. Dockery *et al* found no relationship between PM and chronic cough and chronic phlegm, which are important indicators of chronic bronchitis. I assume that the health impact function based on Dockery *et al* is measuring acute bronchitis.

The estimated logistic coefficient and standard error are based on the odds ratio (1.50) and 95% confidence interval (0.91-2.47) associated with being in the most polluted city ($PM_{2.1} = 20.7 \mu\text{g}/\text{m}^3$) versus the least polluted city ($PM_{2.1} = 5.8 \mu\text{g}/\text{m}^3$) (Dockery et al. 1996, Tables 1 and 4). The original study used $PM_{2.1}$, however, BenMAP uses the $PM_{2.1}$ coefficient and apply it to $PM_{2.5}$ data.

Functional Form: Logistic

Coefficient: 0.027212

Standard Error: 0.017096

Incidence Rate: annual bronchitis incidence rate per person = 0.043 (American Lung Association 2002c, Table 11)

Population: population of ages 8-12.

Lower Respiratory Symptoms (Schwartz and Neas 2000)

Schwartz and Neas (2000) used logistic regression to link lower respiratory symptoms and cough in children with coarse PM_{10} , $PM_{2.5}$, sulfate and H^+ (hydrogen ion). Children were selected for the study if they were exposed to indoor sources of air pollution: gas stoves and parental smoking. The study enrolled 1,844 children into a year-long study that was conducted in different years (1984 to 1988) in six cities. The students were in grades two through five at the time of enrollment in 1984. By the completion of the final study, the cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

The coefficient and standard error are calculated from the reported odds ratio (1.33) and 95% confidence interval (1.11-1.58) associated with a $15 \mu\text{g}/\text{m}^3$ change in $PM_{2.5}$ (Schwartz and Neas 2000, Table 2).

Functional Form: Logistic

¹¹ The original study measured $PM_{2.1}$, however when using the study's results we use $PM_{2.5}$. This makes only a negligible difference, assuming that the adverse effects of $PM_{2.1}$ and $PM_{2.5}$ are comparable.

Coefficient: 0.01901

Standard Error: 0.006005

Incidence Rate: daily lower respiratory symptom incidence rate per person = 0.0012 (Schwartz *et al.* 1994, Table 2).

Population: population of ages 7 to 14.

Minor Restricted Activity Days (Ostro 1989)

Ostro and Rothschild (1989) estimated the impact of PM_{2.5} and ozone on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for PM_{2.5}, two-week average ozone has highly variable association with RRADs and MRADs. Controlling for ozone, two-week average PM_{2.5} was significantly linked to both health endpoints in most years.¹² The C-R function for PM is based on this co-pollutant model.

Using the results of the two-pollutant model, separate coefficients were developed for each year in the analysis, which were then combined for use in this analysis. The coefficient is a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4) using the inverse of the variance as the weight. The standard error of the coefficient is calculated as follows, assuming that the estimated year-specific coefficients are independent.

Functional Form: Log-linear

Coefficient: 0.00741

Standard Error: 0.00070

Incidence Rate: daily incidence rate for minor restricted activity days (MRAD) = 0.02137 (Ostro and Rothschild 1989, p. 243)

Population: adult population ages 18 to 64.¹³

Work Loss Days (Ostro 1987)

Ostro (1987) estimated the impact of PM_{2.5} on the incidence of work-loss days (WLDs), restricted activity days (RADs), and respiratory-related RADs (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average PM_{2.5} levels were significantly linked to work-loss days, RADs, and RRADs, however there was some year-to-year variability in the results.¹⁴

¹² The study used a two-week average pollution concentration; the C-R function uses a daily average, which is assumed to be a reasonable approximation.

¹³ The study is based on a "convenience" sample of non-elderly individuals. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals under 65.

¹⁴ The study used a two-week average pollution concentration; the C-R function uses a daily average, which is assumed to be a reasonable approximation.

Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function presented here is a weighted average of the coefficients in Ostro (1987, Table 3) using the inverse of the variance as the weight.

The coefficient used in the C-R function is a weighted average of the coefficients in Ostro (1987, Table 3) using the inverse of the variance as the weight. The standard error of the coefficient is calculated as follows, assuming that the estimated year-specific coefficients are independent.

Functional Form: Log-linear

Coefficient: 0.0046

Standard Error: 0.00036

Incidence Rate: daily work-loss-day incidence rate per person ages 18 to 64 = 0.00595 (U.S. Bureau of the Census 1997, No. 22; Adams *et al.* 1999, Table 41)

Population: adult population ages 18 to 64.¹⁵

Asthma Exacerbation: Pooling Ostro *et al.* (2001) and Vedal *et al.* (1998)

I pool the results of studies by Ostro *et al.* (2001) and Vedal *et al.* (1998) to get an estimate of lower respiratory symptoms in asthmatics. I use a simple average of the results when I pool – unlike the analysis performed for Clean Air Interstate Rule (U.S. EPA 2005, Table 4-7). In addition to the lower respiratory estimate, I include an upper respiratory estimate based on a study by Pope *et al.* (1991).

To characterize asthma exacerbations in children, EPA uses two studies that followed panels of asthmatic children. Ostro *et al.* (2001) followed a group of 138 African-American children in Los Angeles for 13 weeks, recording daily occurrences of respiratory symptoms associated with asthma exacerbations (e.g., shortness of breath, wheeze, and cough). This study found a statistically significant association between PM_{2.5}, measured as a 12-hour average, and the daily prevalence of shortness of breath and wheeze endpoints. Although the association was not statistically significant for cough, the results were still positive and close to significance; consequently, EPA includes this endpoint, along with shortness of breath and wheeze, in generating incidence estimates.

Vedal *et al.* (1998) followed a group of elementary school children, including 74 asthmatics, located on the west coast of Vancouver Island for 18 months including measurements of daily peak expiratory flow (PEF) and the tracking of respiratory symptoms (e.g., cough, phlegm, wheeze, chest tightness) through the use of daily diaries. Association between PM₁₀ and respiratory symptoms for the asthmatic population was only reported for two endpoints: cough and PEF. Because it is difficult to translate PEF measures into clearly defined health endpoints that can be monetized, EPA only included the cough-related effect estimate from this study in quantifying asthma exacerbations.

¹⁵ The study is based on a “convenience” sample of non-elderly individuals. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals under 65.

EPA employed the following pooling approach in combining estimates generated using effect estimates from the two studies to produce a single asthma exacerbation incidence estimate. First, EPA pooled the separate incidence estimates for shortness of breath, wheeze, and cough generated using effect estimates from the Ostro *et al* (2001) study, because each of these endpoints is aimed at capturing the same overall endpoint (asthma exacerbations) and there could be overlap in their predictions. The pooled estimate from the Ostro *et al.* study is then pooled with the cough-related estimate generated using the Vedal *et al* study. The rationale for this second pooling step is similar to the first; both studies are attempting to quantify the same overall endpoint (asthma exacerbations).

To prevent double-counting, EPA (2005, p. 4-38) focused the estimation on asthma exacerbations occurring in children and excluded adults from the calculation. Asthma exacerbations occurring in adults are assumed to be captured in the general population endpoints such as work loss days and MRADs. Consequently, if EPA had included an adult-specific asthma exacerbation estimate, this would likely have double-counted incidence for this endpoint. However, because the general population endpoints do not cover children (with regard to asthmatic effects), an analysis focused specifically on asthma exacerbations for children (6 to 18 years of age) could be conducted without concern for double-counting.

Asthma Exacerbation: Cough, Wheeze, and Shortness of Breath (Ostro *et al.* 2001)

Ostro *et al.* (2001) studied the relation between air pollution in Los Angeles and asthma exacerbation in African-American children (8 to 13 years old) from August to November 1993. They used air quality data for PM₁₀, PM_{2.5}, NO₂, and O₃ in a logistic regression model with control for age, income, time trends, and temperature-related weather effects.¹ Asthma symptom endpoints were defined in two ways: “probability of a day with symptoms” and “onset of symptom episodes”. New onset of a symptom episode was defined as a day with symptoms followed by a symptom-free day. The authors found cough prevalence associated with PM₁₀ and PM_{2.5} and cough incidence associated with PM_{2.5}, PM₁₀, and NO₂. Ozone was not significantly associated with cough among asthmatics.

Note that the study focused on African-American children ages 8 to 13 years old. EPA applies the function based on this study to the general population ages 6 to 18 years old.

Asthma Exacerbation, Cough

The coefficient and standard error are based on an odds ratio of 1.03 (95% CI 0.98-1.07) for a 30 µg/m³ increase in 12-hour average PM_{2.5} concentration (Ostro *et al.* 2001, Table 4, p. 204).

Functional Form: Logistic

Coefficient: 0.000985

Standard Error: 0.000747

¹ The authors note that there were 26 days in which PM_{2.5} concentrations were reported higher than PM₁₀ concentrations. The majority of results the authors reported were based on the full dataset. These results were used for the basis for the C-R functions.

Incidence Rate: daily cough rate per person (Ostro et al. 2001, p. 202) = 0.145

Population: asthmatic population ages 6 to 18 = 5.67%.²

Asthma Exacerbation, Shortness of Breath

The coefficient and standard error are based on an odds ratio of 1.08 (95% CI 1.00-1.17) for a 30 µg/m³ increase in 12-hour average PM_{2.5} concentration (Ostro et al. 2001, Table 4, p. 204).

Functional Form: Logistic

Coefficient: 0.002565

Standard Error: 0.001335

Incidence Rate: daily shortness of breath rate per person (Ostro et al. 2001, p. 202) = 0.074

Population: asthmatic population ages 6 to 18 = 5.67%.

Asthma Exacerbation, Wheeze

The coefficient and standard error are based on an odds ratio of 1.06 (95% CI 1.01-1.11) for a 30 µg/m³ increase in 12-hour average PM_{2.5} concentration (Ostro et al. 2001, Table 4, p. 204).

Functional Form: Logistic

Coefficient: 0.001942

Standard Error: 0.000803

Incidence Rate: daily wheeze rate per person (Ostro et al. 2001, p. 202) = 0.173

Population: asthmatic population ages 6 to 18 = 5.67%.

Asthma Exacerbation, Cough (Vedal et al. 1998)

Vedal *et al.* (1998) studied the relationship between air pollution and respiratory symptoms among asthmatics and non-asthmatic children (ages 6 to 13) in Port Alberni, British Columbia, Canada. Four groups of elementary school children were sampled from a prior cross-sectional study: (1) all children with current asthma, (2) children without doctor diagnosed asthma who experienced a drop in FEV after exercise, (3) children not in groups 1 or 2 who had evidence of airway obstruction, and (4) a control group of children with matched by classroom.

The authors used logistic regression and generalized estimating equations to examine the association between daily PM₁₀ levels and daily increases in various respiratory symptoms among these groups. In the entire sample of children, PM₁₀ was significantly associated with cough, phlegm, nose symptoms, and throat soreness. Among children with diagnosed asthma, the authors report a significant association between PM₁₀ and cough symptoms, while no consistent effects were observed in the other groups. Since the study population has an over-representation of asthmatics, due to the sampling

² The American Lung Association (2002a, Table 7) estimates asthma prevalence for children 5-17 at 5.67% (based on data from the 1999 National Health Interview Survey).

strategy, the results from the full sample of children are not generalizable to the entire population. The C-R function presented below is based on results among asthmatics ages 6 to 18.

The PM₁₀ coefficient and standard error are based on an increase in odds of 8% (95% CI 0-16%) reported in the abstract for a 10 µg/m³ increase in daily average PM₁₀.

Functional Form: Logistic

Coefficient: 0.007696

Standard Error: 0.003786

Incidence Rate: daily cough rate per person (Vedal et al. 1998, Table 1, p. 1038) = 0.086

Population: asthmatic population ages 6 to 18 = 5.67%.³

Upper Respiratory Symptoms (Pope 1991)

Using logistic regression, Pope *et al.* (1991) estimated the impact of PM₁₀ on the incidence of a variety of minor symptoms in 55 subjects (34 “school-based” and 21 “patient-based”) living in the Utah Valley from December 1989 through March 1990. The children in the Pope *et al.* study were asked to record respiratory symptoms in a daily diary. With this information, the daily occurrences of upper respiratory symptoms (URS) and lower respiratory symptoms (LRS) were related to daily PM₁₀ concentrations. Pope *et al.* describe URS as consisting of one or more of the following symptoms: runny or stuffy nose; wet cough; and burning, aching, or red eyes. Levels of ozone, NO₂, and SO₂ were reported low during this period, and were not included in the analysis.

The sample in this study is relatively small and is most representative of the asthmatic population, rather than the general population. The school-based subjects (ranging in age from 9 to 11) were chosen based on “a positive response to one or more of three questions: ever wheezed without a cold, wheezed for 3 days or more out of the week for a month or longer, and/or had a doctor say the ‘child has asthma’ (Pope et al. 1991, p. 669).” The patient-based subjects (ranging in age from 8 to 72) were receiving treatment for asthma and were referred by local physicians. Regression results for the school-based sample (Pope et al. 1991, Table 5) show PM₁₀ significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM₁₀ effect. The results from the school-based sample are used here.

The coefficient and standard error for a one µg/m³ change in PM₁₀ is reported in Pope *et al.* (1991, Table 5).

Functional Form: Logistic

Coefficient: 0.0036

Standard Error: 0.0015

³ The American Lung Association (American Lung Association 2002a) estimates asthma prevalence for children 5-17 at 5.67% (based on data from the 1999 National Health Interview Survey).

Incidence Rate: daily upper respiratory symptom incidence rate per person = 0.3419 (Pope et al. 1991, Table 2)

Population: asthmatic population ages 9 to 11 = 5.67% of population ages 9 to 11.⁴

⁴ The American Lung Association (2002a, Table 7) estimates asthma prevalence for children ages 5 to 17 at 5.67% (based on data from the 1999 National Health Interview Survey).

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