APPENDIX C

POPULATION FORECASTING FOR BENEFITS ANALYSIS

This appendix summarizes the steps used to estimate 2020 and 2030 population. In addition, we include a table with age-specific population estimates by state for the years 2000, 2020, and 2030.

Population Grid Cells

BenMAP calculates health impacts at the level of U.S. counties as well as for a variety of grid structures used in air quality modeling (e.g.,, REMSAD, and CAMx). In this description, we use the term "population grid-cells" to refer to counties or the cells within an air quality modeling grid. The foundation for calculating the population level in the population grid-cells is the 2000 Census block data.[1] A separate application developed by Abt Associates, called "PopGrid," combines the Census block data with any user-specified set of population grid-cells, so long as they are defined by a GIS shape file.

If the center of a Census block falls within a population grid-cell, PopGrid assigns the block population to this particular population grid-cell. Note that the grid-cells in air quality model, such as REMSAD and CAMx, may cross multiple county boundaries. To account for this, PopGrid keeps track of the total number of people by county within a particular populatin grid-cell. Keeping track of the total number of people in a county is useful in the estimation of adverse health effects, where the calculation of premature mortality depends on county-level mortality rates. It is also useful in the presentation of health benefits, when users may want estimates at the state- and county-level, as opposed to estimates by, say, the area covered by an air quality model.

Within any given population grid-cell, BenMAP has 256 demographic variables, including 180 unique racial-gender-age groups: 19 age groups by gender by 5 racial groups (19*2*5=180). In addition there is an Hispanic ethnicity variable, which includes a number of different racial groups, as well as a number of variables that aggregate the population by race and gender. Exhibit C-1 presents the 256 population variables available in BenMAP. As discussed below, these variables are available for use in developing age estimates in whatever grouping desired by the user.

[1] Geolytics (2001; 2002a) provided the 1990 and 2000 census data.

Exhibit C-1. Demographic Groups and Variables Available in BenMAP

Racial/Ethnic Group	Gender	Age	# Variables
White, African American, Asian, American Indian, Other, Hispanic	Female, Male	0-1, 1-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85+	228
All	–	0-1, 1-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85+	19
All	Female, Male	–	2
White, African American, Asian, American Indian, Other, Hispanic	–	–	6
All	–	–	1

Census Data 2000

In addition to forecasting post-2000 population levels based on the 2000 Census, BenMAP also allows the user to estimate the impacts for 1991-1999 by interpolating between the results of the 1990 and 2000 Census. As a result, we have developed a consistent set of demographic variables, based on the 1990 Census, which provides somewhat less detail than the 2000 Census.

The 2000 Census allows respondents to choose more than one racial category, unlike the 1990 Census, which allowed only one choice. As a result there are seven racial categories in the 2000 Census versus five in the 1990 Census (Exhibit C-2). To make the 2000 Census data consistent with the 1990 Census, we reduced the seven racial groups to the five used in the 1990 Census.

The initial data set at the block level includes 368 demographic groups: seven racial groups and Hispanic ethnicity, by 23 pre-defined age groups by gender (Exhibit C-2). Because the 2000 Census includes somewhat different age groupings than that for the final set generated for the 1990 Census. Age variables 15-17 and 18-19 are combined, 20, 21, and 22-24 are combined, 60-61 and 62-24 are combined, and 65-66 and 67-69 are combined at the block level. One variable, under 5 years, must be split into two variables (Under 1 and 1-4 years). Assuming that the population is uniformly distributed within age groups, we apply a factor of 1/5 to create the 0-1 age group and 4/5 to create the 1-4 age group.

Exhibit C-2. Race, Ethnicity and Age Variables in 2000 Census Block Data

	Race / Ethnicity	Gender	Age
Initial Variables	White Alone, Black Alone, Native American Alone, Asian Alone, Pacific Islander / Hawaiian Alone, Other Alone, Two or More Alone, Hispanic (Non-Exclusive)	Male, Female	0-5, 5-10, 10-14, 15-17, 18-19, 20, 21, 22-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-61, 62-64, 65-66, 67-69, 70-74, 75-79, 80-84 85+
Final Variables (identical to 1990 variables)	White, African American, Asian & Pacific Islander, American Indian, Other, Hispanic	Female, Male	0-1, 1-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85+

Source: Geolytics (2002a). Note: Some population values were errors in the original Census data (e.g., values of a billion or more). Following personal communication with Geolytics, these were set to zero.

Matching Racial Categories in the 1990 and 2000 Censuses

Unlike the 1990 Census, respondents in the 2000 Census respondents could check more than one box for race, so the reported results included a grouping of individuals that had checked two or more racial categories. In addition, the 2000 Census separately reported the categories "Pacific Islander / Hawaiian Along" and "Asian Alone." To make the racial groupings comparable with the 1990 Census, we first combined Pacific Islander / Hawaiian Alone with the Asian Alone category to form the category Asian and Pacific Islander category. Then we divided the category Two-or-More between the remaining five racial categories.

Exhibit C-3 presents the estimated percentage of the national population by five racial groups: (1) American Indian or Alaska Native, (2) Asian or Pacific Islander, (3) Black, (4) White, and (5) Other, as well as for four combinations: (1) American Indian or Alaska Native (AIAN)/White, (2) Asian or Pacific Islander (API)/White, (3) Black/White, and (4) Other combinations. Slightly over 98 percent of individuals chose a single racial category, with 1.45 percent choosing three AIAN/White, API/White, and Black/White, and 0.30 choosing other combinations (e.g., Black/Asian). Exhibit C-3 also presents the estimated primary racial affiliation of individuals in these subcategories if they were to choose a single racial affiliation.

Exhibit C-3. Distribution of Racial Groups

Racial Category	% of Total U.S. Population [a]	% of Population in Sub-Groups by Primary Racial Affiliation [b]					
		AIAN	API	Black	White	Other	All
American Indian or Alaska Native (AIAN)	0.85	100	–	–	–	–	100
Asian or Pacific Islander (API)	3.35	–	100	–	–	–	100
Black	12.07	–	–	100	–	–	100
White	79.72	–	–	–	100	–	100
Other race	2.25	–	–	–	–	100	100
AIAN/White	0.89	12.4	–	–	80.9	6.7	100
API/White	0.30	–	34.6	–	46.9	18.4	100
Black/White	0.26	–	–	48.2	25.2	26.6	100
Other combinations [c]	0.30	–	–	–	–	100.0	100
Two-or-More Sub-Total [d]	1.75	6.3	5.9	7.2	52.9	27.7	100

[a] All percentages weighted to be nationally representative. Percentages taken from Parker and Makuc (2001, Table 2), who cited the National Health Interview Survey 1993-1995, APPENDIX: Percent Distribution (Standard Error) of Primary Racial Identification for Selected Detailed Race Groups.

[b] Primary racial affiliation based on survey results from Parker and Makuc (2001, Appendix).

[c] Parker and Makuc (2001) did not provide an estimate of the primary racial affiliation for "Other combinations, so we assume that it belongs to the "Other" category. Note that they did provide the primary racial affiliation for a fourth group "Black/AIAN:" 85.4% Black, 7.0% AIAN, and 7.6% Other. However, we do not have an estimate of the relative abundance of Black/AIAN in the general population, so we have dropped it from further consideration.

[d] As described in the text below, we calculated the percentages in this row from the percentages in the previous four rows for AIAN/White, API/White, Black/White, and Other combinations.

To estimate how to assign a single racial group for individuals that chose two or more racial groups, we used the results of Exhibit C-3 for the three main categories for which we an estimate of the primary racial affiliation: AIAN/White, API/White, and Black/White. To account for the 0.30 percent of the population in other combinations, we For each Census block, we assume that .89 / (.89+.30+.26+.30) = 50.8% of respondents in the Two or More category will fall into the AIAN / White category, and of these, 80.9% would primarily identify themselves as White if they were to choose a single racial category, 12.4% would primarily identify themselves as American Indian or Alaska Native, and 6.7% would primarily identify themselves as Other. Thus 0.508 * .809 = 41% of Two or More we will call White, 10% we identify as Native American, and 5% as Other.

We did not attempt to predict what respondents in the 'Other Combinations' category would have selected if they were to choose a single racial category, so we assume they are part

of the "Other" category. To estimate the number of individuals in each of the five races, we performed the following calculations:

$$NativeAmerican = NativeAmericanAlone_{Pop} + TwoorMore_{Pop} \cdot \left(\frac{AIAN/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot AIAN\%_{AIAN/White} \right)$$

$$Asian = AsianAlone_{Pop} + PacificIslander/Hawaiian_{Pop} + TwoorMore_{Pop} \cdot \left(\frac{API/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot API\%_{API/White} \right)$$

$$Black = BlackAlone_{Pop} + TwoorMore_{Pop} \cdot \left(\frac{Black/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot Black\%_{Black/White} \right)$$

$$White = White\ Alone_{Pop} + Two\ \ or\ More_{Pop} \cdot \left(\begin{array}{l} \frac{AIAN/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot White\%_{AIAN/White} + \\ \frac{API/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot White\%_{API/White} + \\ \frac{Black/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot White\%_{Black/White} \end{array} \right)$$

$$Other = Other\ Alone_{Pop} + Two\ \ or\ More_{Pop} \cdot \left(\begin{array}{l} \frac{AIAN/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot Other\%_{AIAN/White} + \\ \frac{API/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot Other\%_{API/White} + \\ \frac{Black/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot Other\%_{Black/White} + \\ \frac{Other\ \ Combinations_{Nat\%}}{MultipleRace_{Nat\%}} \end{array} \right)$$

This then reduces to:

$$Native\ American_{Pop} = Native\ American\ Alone_{Pop} + (0.063)Two\ or\ More_{Pop}$$

$$Asian_{Pop} = Asian\ Alone_{Pop} + Pacific\ Islander / Hawaiian_{Pop} + (0.059)Two\ or\ More_{Pop}$$

$$Black_{Pop} = Black\ Alone_{Pop} + (0.072)Two\ or\ More_{Pop}$$

$$White_{Pop} = White\ Alone_{Pop} + (0.530)Two\ or\ More_{Pop}$$

$$Other_{Pop} = White\ Alone_{Pop} + (0.276)Two\ or\ More_{Pop} \quad .$$

Estimating Population Levels in Alternative Age Groups

In calculating the population in age groups that may include a portion of one of the pre-specified demographic groups in Exhibit C-1, BenMAP assumes the population is uniformly distributed in the age group. For example, to calculate the number of children ages 3 through 12, BenMAP calculates:

$$age_{3-12} = \frac{1}{4} \cdot age_{1-4} + age_{5-9} + \frac{3}{5} \cdot age_{10-14} \quad .$$

<u>Estimating Population Levels in Non-Census Years</u>

To forecast population levels beyond 2000, BenMAP scales the 2000 Census-based estimate with the ratio of the county-level forecast for the future year of interest over the 2000 county-level population level. Woods & Poole (2001) provides the county-level population forecasts used to calculate the scaling ratios.

In the simplest case, where one is forecasting a single population variable, say, children ages 4 to 9 in the year 2010, CAMPS calculates:

$$age_{4-9,\,g,\,2010} = age_{4-9,\,g,\,2000} \cdot \frac{age_{4-9,\,county,\,2010}}{age_{4-9,\,county,\,2000}}$$

where the g^{th} population grid-cell is wholly located within a given county.

In the case, where the g[th] grid-cell includes "n" counties in its boundary, the situation is somewhat more complicated. BenMAP first estimates the fraction of individuals in a given age group (e.g., ages 4 to 9) that reside in the part of each county within the g[th] grid-cell. BenMAP calculates this fraction by simply dividing the population all ages of a given county within the g[th] grid-cell by the total population in the g[th] grid-cell:

$$fraction\ of\ \ age_{4-9,\ g\ in\ county_c} = \frac{age_{all,\ g\ in\ county_c}}{age_{all,\ g}}$$

Multiplying this fraction with the number of individuals ages 4 to 9 in the year 2000 gives an estimate of the number of individuals ages 4 to 9 that reside in the fraction of the county within the g[th] grid-cell in the year 2000:

$$age_{4-9,\ g\ in\ county_c,\ 2000} = age_{4-9,\ g,\ 2000} \cdot fraction\ age_{4-9,\ g\ in\ county_c}$$

To then forecast the population in 2010, we scale the 2000 estimate with the ratio of the county projection for 2010 to the county projection for 2000:

$$age_{4-9,\ g\ in\ county_c,\ 2010} = age_{4-9,\ g\ in\ county_c,\ 2000} \cdot \frac{age_{4-9,\ county_c,\ 2010}}{age_{4-9,\ county_c,\ 2000}} \quad .$$

Combining all these steps for "c" counties within the g[th] grid-cell, we forecast the population of persons ages 4 to 9 in the year 2010 as follows:

$$age_{4-9,\ g,\ 2010} = \sum_{c=1}^{n} age_{4-9,\ g,\ 2000} \cdot \frac{total\ \ pop_{g\ in\ county_c}}{total\ \ pop_g} \cdot \frac{age_{4-9,\ county_c,\ 2010}}{age_{4-9,\ county_c,\ 2000}}$$

In the case where there are multiple age groups and multiple counties, BenMAP first calculates the forecasted population level for individual age groups, and then combines the forecasted age groups. In calculating the number of children ages 4 to 12, BenMAP calculates:

$$age_{4-9,\ g,\ 2010} = \sum_{c=1}^{n} age_{4-9,\ g,\ 2000} \cdot \frac{total\ \ pop_{g\ in\ county_c}}{total\ \ pop_g} \cdot \frac{age_{4-9,\ county_c,\ 2010}}{age_{4-9,\ county_c,\ 2000}}$$

$$age_{10-14,\ g,\ 2010} = \sum_{c=1}^{n} age_{10-14,\ g,\ 2000} \cdot \frac{total\ \ pop_{g\ in\ county_c}}{total\ \ pop_g} \cdot \frac{age_{10-14,\ county_c,\ 2010}}{age_{10-14,\ county_c,\ 2000}}$$

$$age_{4-12,\,g,\,2010} = age_{4-9,\,g,\,2010} + \frac{3}{5} \cdot age_{10-14,\,g,\,2010} \quad .$$

Since the Woods and Poole (2001) projections only extend through 2025, we used the existing projections and constant growth factors to provide additional projections. To estimate population levels beyond 2025, BenMAP linearly extrapolates from the final two years of data. For example, to forecast population in 2030, BenMAP calculates:

$$age_{4-9,\,2030} = age_{4-9,\,2025} + 5 \cdot \left(age_{4-9,\,2025} - age_{4-9,\,2024} \right) \quad .$$

Exhibit C-4 summarizes the forecasted age-stratified, state-level populations for 2020 and 2030. In addition, to provide a point of comparison, it includes population levels for year 2000.

Exhibit C-4. State-Level Population Estimates by Age Group

	2000			2020			2030		
State	***0-18***	***18-64***	***65+***	***0-18***	***18-64***	***65+***	***0-18***	***18-64***	***65+***
AL	1,126,337	2,740,965	579,798	1,212,702	3,057,540	909,065	1,284,828	3,069,518	1,195,921
AZ	1,371,099	3,091,693	667,839	1,832,737	4,351,254	1,309,943	2,117,749	4,708,843	1,812,966
AR	681,003	1,618,378	374,019	769,208	1,821,876	557,233	832,285	1,861,954	698,259
CA	9,254,212	21,021,768	3,595,658	10,105,474	25,308,061	5,717,329	10,895,067	26,084,324	7,681,464
CO	1,101,772	2,783,415	416,073	1,321,930	3,359,731	954,691	1,498,688	3,453,808	1,370,557
CT	839,051	2,096,330	470,183	821,773	2,110,504	588,222	845,210	1,991,695	728,973
DE	195,997	485,877	101,726	213,375	571,224	149,015	227,024	574,497	204,731
DC	120,659	381,502	69,898	95,389	337,146	103,401	93,833	305,565	123,728
FL	3,643,004	9,531,774	2,807,597	4,466,384	12,098,406	4,472,647	5,026,785	12,483,019	5,933,620
GA	2,176,259	5,224,918	785,275	2,600,100	6,296,967	1,328,722	2,851,139	6,620,751	1,778,194
ID	369,522	778,515	145,916	451,473	980,346	282,616	507,776	1,045,592	374,826
IL	3,247,904	7,671,362	1,500,025	3,286,653	8,148,579	2,069,429	3,425,612	7,962,757	2,669,430
IN	1,581,993	3,745,661	752,831	1,691,800	4,113,510	1,087,932	1,800,717	4,096,828	1,421,006
IA	737,415	1,752,696	436,213	734,433	1,820,333	593,034	766,374	1,750,358	755,945
KS	714,371	1,617,818	356,229	760,573	1,795,227	499,065	814,382	1,778,859	653,139
KY	998,042	2,538,933	504,793	1,077,101	2,762,379	801,696	1,154,120	2,750,564	1,052,988
LA	1,221,651	2,730,396	516,929	1,247,161	2,952,038	850,018	1,318,748	2,917,899	1,116,293
ME	299,691	791,830	183,402	284,880	852,466	289,399	297,507	807,626	394,873
MD	1,350,517	3,346,661	599,307	1,453,726	3,868,715	926,465	1,559,338	3,877,266	1,256,566
MA	1,508,818	3,980,116	860,162	1,533,618	4,071,543	1,144,857	1,604,543	3,871,104	1,469,089
MI	2,596,118	6,123,307	1,219,018	2,587,563	6,590,540	1,798,905	2,703,858	6,417,627	2,366,125
MN	1,285,100	3,040,113	594,266	1,413,120	3,525,458	948,035	1,547,597	3,524,435	1,298,319
MS	779,939	1,721,196	343,523	826,142	1,912,067	513,412	867,469	1,926,497	667,632
MO	1,428,853	3,410,978	755,379	1,526,846	3,830,433	1,062,471	1,631,969	3,798,554	1,404,065

Exhibit C-4. State-Level Population Estimates by Age Group (continued) *May 12, 2003*

	2000			2020			2030		
State	***0-18***	***18-64***	***65+***	***0-18***	***18-64***	***65+***	***0-18***	***18-64***	***65+***
MT	228,916	552,330	120,949	234,129	612,736	241,971	258,376	604,179	322,696
NE	450,372	1,028,696	232,195	483,340	1,148,129	329,112	522,703	1,142,368	426,556
NV	510,633	1,268,694	218,929	757,488	1,921,749	477,249	904,840	2,150,250	654,589
NH	309,490	778,326	147,970	321,958	906,776	236,489	346,320	897,774	329,510
NJ	2,074,020	5,227,192	1,113,136	2,126,538	5,560,594	1,507,553	2,222,228	5,402,892	1,949,786
NM	506,558	1,100,262	212,225	583,389	1,372,898	412,394	644,935	1,420,580	572,907
NY	4,696,232	11,831,869	2,448,352	4,487,417	11,815,310	3,179,326	4,540,245	11,246,710	3,953,934
NC	1,977,387	5,102,877	969,048	2,399,345	6,081,807	1,588,246	2,646,039	6,337,401	2,079,430
ND	162,017	385,705	94,478	152,979	407,052	152,185	160,056	387,072	203,240
OH	2,889,207	6,956,175	1,507,757	2,894,902	7,316,549	2,031,922	3,021,197	7,084,923	2,597,112
OK	894,531	2,100,173	455,950	968,204	2,255,616	685,395	1,037,634	2,249,445	865,166
OR	846,233	2,136,988	438,177	1,028,841	2,593,792	824,166	1,161,142	2,731,400	1,065,084
PA	2,930,189	7,431,699	1,919,165	2,807,320	7,589,422	2,473,482	2,879,828	7,112,827	3,152,928
RI	252,438	643,479	152,402	248,650	664,840	185,270	253,697	626,436	236,588
SC	1,017,627	2,509,052	485,333	1,125,147	2,936,359	879,310	1,217,702	2,989,589	1,188,398
SD	202,496	444,217	108,131	209,379	498,258	159,468	222,092	487,168	215,460
TN	1,402,958	3,583,013	703,311	1,614,405	4,118,556	1,147,546	1,759,007	4,213,846	1,513,183
TX	5,891,741	12,887,542	2,072,532	7,108,830	15,994,222	3,802,007	7,929,363	16,840,990	5,236,651
UT	724,466	1,318,481	190,222	989,440	1,826,327	368,454	1,120,100	2,046,412	497,421
VT	147,949	383,368	77,510	137,590	414,505	138,315	144,053	397,442	190,941
VA	1,743,459	4,542,721	792,333	1,955,331	5,201,333	1,308,689	2,132,729	5,295,036	1,734,954
WA	1,511,831	3,720,140	662,148	1,785,937	4,644,371	1,174,213	2,006,978	4,857,761	1,581,410
WV	404,484	1,126,965	276,895	388,379	1,094,529	403,851	404,280	1,038,496	488,364
WI	1,369,215	3,291,907	702,553	1,413,693	3,680,062	1,070,942	1,511,982	3,612,218	1,450,806
WY	128,585	307,504	57,693	124,005	314,574	117,862	132,595	301,356	154,460

Woods & Poole Data

Woods & Poole (2001) developed county-level forecasts for each year from 2000 through 2025, for three racial groups "Black," "White," and "Other," and by age and by gender. For the Hispanic ethnic group, Woods and Poole developed forecasts just for the total population, and not by age and gender. As discussed in the section on population forecasts, BenMAP uses these forecasts to simply scale the 2000 Census block data, in order to estimate the population in the population grid-cells for any given year after 2000.

Aligning Woods & Poole FIPS Codes with BenMAP FIPS Codes

The county geographic boundaries used by Woods & Poole are somewhat more aggregated than the county definitions used in the 2000 Census (and BenMAP), and the FIPS codes used by Woods and Poole are not always the standard codes used in the Census. To make the Woods and Poole data consistent with the county definitions in BenMAP, we disaggregated the Woods and Poole data and changed some of the FIPS codes. Exhibit C-5 lists the discrepancies in the county definitions between Woods & Poole and those used in BenMAP.

To assign the population in the more aggregated Woods & Poole county definitions to the more disaggregated definitions used in BenMAP (and the U.S. Census), we used the total county population from the 2000 U.S. Census. We then assumed that the age and racial groups were distributed uniformly across the BenMAP counties contained within a Woods & Poole county definition. For example, in estimating the population of children ages 4-9 in county "c" contained within a more broadly defined Woods & Poole county, we would do the following:

$$age_{4-9,\,county_c} = age_{4-9,\,W\&P\ county} \cdot \frac{age_{all,\,county_c}}{age_{all,\,W\&P\ county}}$$

After this factor was applied, we rounded the estimates to the nearest integer so as to avoid having data with "partial people."

Exhibit C-5. Linkage Between Woods & Poole County Definitions and BenMAP County Definitions

Woods and Poole Counties (FIPS)	Counties in BenMAP (FIPS)
Northwest Arctic Borough, AK (02188)	Kobuk, AK (02140)
Remainder of Alaska, AK (02999)	Aleutian Islands, AK (02010), Aleutian Islands East Borough, AK (02013), Aleutian Islands West Census Area, AK (02016), Bethel Census Area, AK (02050), Denali Borough, AK (02068), Dillingham Census Area, AK (02070), Haines Borough, AK (02100), Kenai Peninsula Borough, AK (02122), Lake and Peninsula Borough, AK (02164), North Slope Borough, AK (02185), Prince of Wales-Outer Ketchikan, AK (02201), Sitka Borough, AK (02220), Skagway-Yukatat-Angoon, AK (02231), Skagway-Hoonah-Angoon Census Area, AK (02232), Southeast Fairbanks Census Area, AK (02240), Valdez-Cordova Census Area, AK (02261), Wrangell-Petersburg Census Area, AK (02280), Yakutat Borough, AK (02282), Yukon-Koyukuk, AK (02290)
Yuma + La Paz, AZ (04027)	La Paz, AZ (04012), Yuma, AZ (04027)
Miami-Dade, FL (12086)	Dade, FL (12025)
Maui + Kalawao, HI (15901)	Kalawao, HI (15005), Maui, HI (15009)
Fremont, ID (16043)	Fremont, ID (16043), Yellowstone Park, ID
Park, MT (30067)	Park, MT (30067), Yellowstone Park, MT (30113)
Valencia + Cibola, NM (35061)	Cibola, NM (35006), Valencia, NM (35061)
Halifax, VA (51083)	Halifax, VA (51083), South Boston City, VA (51780)
Albemarle + Charlottesville, VA (51901)	Albemarle, VA (51003), Charlottesville City, VA (51540)
Alleghany + Clifton Forge + Covington, VA (51903)	Alleghany, VA (51005), Clifton Forge City, VA (51560), Covington City, VA (51580)
Augusta + Staunton + Waynesboro, VA (51907)	Augusta, VA (51015), Staunton City, VA (51790), Waynesboro City, VA (51820)
Bedford + Bedford City, VA (51909)	Bedford, VA (51019), Bedford City, VA (51515)
Campbell + Lynchburg, VA (51911)	Campbell, VA (51031), Lynchburg City, VA (51680)
Carroll + Galax, VA (51913)	Carroll, VA (51035), Galax City, VA (51640)
Dinwiddie + Colonial Heights + Petersburg, VA (51918)	Dinwiddie, VA (51053), Colonial Heights City, VA (51570), Petersburg City, VA (51730)
Fairfax + Fairfax City + Falls Church City, VA (51919)	Fairfax, VA (51059), Fairfax City, VA (51600), Falls Church City, VA (51610)
Frederick + Winchester, VA (51921)	Frederick, VA (51069), Winchester City, VA (51840)
Greensville + Emporia, VA (51923)	Greensville, VA (51081), Emporia City, VA (51595)
Henry + Martinsville, VA (51929)	Henry, VA (51089), Martinsville City, VA (51690)
James City + Williamsburg, VA (51931)	James City County, VA (51095), Williamsburg City, VA (51830)

Woods and Poole Counties (FIPS)	Counties in BenMAP (FIPS)
Montgomery + Radford, VA (51933)	Montgomery, VA (51121), Radford City, VA (51750)
Pittsylvania + Danville, VA (51939)	Pittsylvania, VA (51143), Danville City, VA (51590)
Prince George + Hopewell, VA (51941)	Prince George, VA (51149), Hopewell City, VA (51670)
Prince William + Manassas + Manassas Park, VA (51942)	Prince William, VA (51153), Manassas City, VA (51683), Manassas Park City, VA (51685)
Roanoke + Salem, VA (51944)	Roanoke, VA (51161), Salem City, VA (51775)
Rockbridge + Buena Vista + Lexington, VA (51945)	Rockbridge, VA (51163), Buena Vista City, VA (51530), Lexington City, VA (51678)
Rockingham + Harrisonburg, VA (51947)	Rockingham, VA (51165), Harrisonburg City, VA (51660)
Southampton + Franklin, VA (51949)	Southampton, VA (51175), Franklin City, VA (51620)
Spotsylvania + Fredericksburg, VA (51951)	Spotsylvania, VA (51177), Fredericksburg City, VA (51630)
Washington + Bristol, VA (51953)	Washington, VA (51191), Bristol City, VA (51520)
Wise + Norton, VA (51955)	Wise, VA (51195), Norton City, VA (51720)
York + Poquoson, VA (51958)	York, VA (51199), Poquoson City, VA (51735)
Shawano (includes Menominee), WI (55901)	Menominee, WI (55078), Shawano, WI (55115)

Age, Gender, Race, and Ethnicity

We generated the same 38 age and gender categories developed from the 1990 and 2000 Census data. Since these projections are available for every year of age, it is a simple matter to sum the individual years to get the same age categories used by BenMAP.

However, the only racial categories available are "White," "Black," and "Other." Since we do not have an Asian or Native American group, or an Other group which is consistent with the definition used by the 1990 and 2000 Census data, we assume that projection data's Other category is representative of all 3 groups, and that they move together over time.

The county projections only forecast the Hispanic population of all ages, and does not have separate gender and age forecasts. Lacking further information, we use the ratio of future-year all age population to the year 2000 all age population when forecasting any particular age group of Hispanics. In effect, we assume for all forecast years the same distribution of age and gender as found in the 2000 Census.

Creating Growth Ratios from Absolute Population Values

For each year from 2000 through 2025 and for each of the 256 demographic groups listed in Exhibit C-1, BenMAP stores the ratio of the future-year to year 2000 county-level population projections. As described below, these ratios are used to forecast population levels in the population grid-cells used by BenMAP to health effects.

Note that there are a small number of cases were the 2000 county population for a specific demographic group is zero, so the ratio of any future year to the year 2000 data is undefined. In these relatively rare cases, we set the year 2000 ratio and all subsequent ratios to 1, assuming no growth.

There are an even smaller number of cases where a total population variable dwindles from some non-zero number to zero, creating ratios of zero. Variables which represent a subpopulation of the first variable may not be zero, however. In these cases, we set all subset population variables for that year to zero.

For instance, if a county only had one person in it for the year 2000 - a 79 year old black male - we set all variables (excluding total variables and BlackMale75to79) to a ratio of 1, because their 2000 values of 0 produce undefined ratios. If the man dies at age 82, the total black population variable for years 2003 and beyond is calculated as 0/1 = 0. Thus for each of those years where the total black population is listed as zero, we go back and set all black population variables to zero, to reflect the knowledge that the block is empty. For all variables except the BlackMale75to79 age group (already zero), 1 becomes 0.

APPENDIX D

PARTICULATE MATTER AND OZONE CONCENTRATION-RESPONSE FUNCTIONS

In this Appendix, we present the concentration-response (C-R) functions used to estimate adverse health effects related to PM and ozone. First, we discuss the concentration response functions for particulate matter, then we discuss then concentration response functions for ozone. Each sub-section has an Exhibit with a brief description of the C-R function and the underlying parameters. Following each Exhibit, we present a brief summary of each of the studies and any items that are unique to the study. Also, note that each citation in the text includes a numbered reference to a database that facilitates updating the citations.

Particulate Matter Concentration Response Functions

Long-term Mortality

There are two types of exposure to PM that may result in premature mortality. Short-term exposure may result in excess mortality on the same day or within a few days of exposure. Long-term exposure over, say, a year or more, may result in mortality in excess of what it would be if PM levels were generally lower, although the excess mortality that occurs will not necessarily be associated with any particular episode of elevated air pollution levels. In other words, long-term exposure may capture a facet of the association between PM and mortality that is not captured by short-term exposure.

Exhibit D-1 Concentration-Response (C-R) Functions for Particulate Matter and Long-Term Mortality

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time	Beta	Std Error	Functional Form	Notes
All Cause	$PM_{2.5}$	Krewski et al.	2000	63 cities	30+	All	All	None	Annual Avg	0.004626	0.001205	Log-linear	ACS reanalysis
All Cause	$PM_{2.5}$	Krewski et al.	2000	50 cities	30+	All	All	None	Annual Median	0.005348	0.001464	Log-linear	ACS reanalysis
All Cause	$PM_{2.5}$	Krewski et al.	2000	nationwide	30+	All	All	None	Annual Median	0.010394	0.002902	Log-linear	ACS reanalysis; RE Ind Cities
All Cause	$PM_{2.5}$	Krewski et al.	2000	nationwide	30+	All	All	None	Annual Median	0.006058	0.003383	Log-linear	ACS reanalysis; RE Reg Adj
All Cause	$PM_{2.5}$	Krewski et al.	2000	6 cities	25+	All	All	None	Annual Avg	0.013272	0.004070	Log-linear	Six Cities reanalysis
All Cause	$PM_{2.5}$	Pope et al.	1995	50 cities	30+	All	All	None	Annual Median	0.006408	0.001509	Log-linear	
All Cause	$PM_{2.5}$	Dockery et al.	1993	6 cities	25+	All	All	None	Annual Avg	0.012425	0.004228	Log-linear	
All Cause	$PM_{2.5}$	Pope et al.	2002	61 cities	30+	All	All	None	Annual Avg	0.004018	0.001642	Log-linear	'79-'83 exposure
Cardiopulmonary	$PM_{2.5}$	Pope et al.	2002	61 cities	30+	All	All	None	Annual Avg	0.005733	0.002167	Log-linear	'79-'83 exposure
Lung Cancer	$PM_{2.5}$	Pope et al.	2002	61 cities	30+	All	All	None	Annual Avg	0.007881	0.003463	Log-linear	'79-'83 exposure
Infant	PM_{10}	Woodruff et al.	1997	86 cities	<1	All	All	None	Annual Avg	0.003922	0.001221	Logistic	

Mortality - Mean, All Cause [Krewski, 2000 #1805] - Reanalysis of Pope et al. [, 1995 #81]

The Krewski et al. [2000 #1805] reanalysis of Pope et al. [1995 #81] used a Cox proportional hazard model to estimate the impact of long-term PM exposure. The original investigation followed 295,223 individuals[1] ages 30 and over in 50 cities from September 1, 1982 to December 31, 1989, and related their survival to median $PM_{2.5}$ concentrations for 1979 to 1983. Krewski et al. [2000 #1805] independently estimated city-specific annual *mean* values from EPA's Inhalable Particle Monitoring Network (IPMN) for the same years (1979-1983). Krewski et al. [2000 #1805] followed Pope et al. [1995 #81, 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] and found that mean $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. Krewski et al. included only PM, so it is unclear to what extent it may be including the impacts of ozone or other gaseous pollutants.

Pope et al. [1995 #81] is the better of the two published prospective cohort studies: it has a larger population and includes more cities than the prospective cohort study by Dockery et al. [1993 #20]. Pope et al.'s study has several further advantages. The population followed in this study was largely Caucasian and middle class, decreasing the likelihood that interlocational differences in premature mortality were due in part to differences in race, socioeconomic status, or related factors. In addition, the PM coefficient in Pope et al. is likely to be biased downward, counteracting a possible upward bias associated with historical air quality trends discussed earlier. One source of this downward bias is the generally healthier and study population, in comparison to poorer minority populations. Krewski et al. [2000 #1805, Part II - Table 52] found that educational status was a strong effect modifier of the PM - mortality relationship in both studies, with the strongest effect seen among the less educated. In fact, much of the differences in magnitude of effect between the studies was made up when assessing risk across comparable levels of educational attainment.

Another source of downward bias is that intercity movement of cohort members was not considered in the original study and therefore could not be evaluated in the reanalysis. Migration across study cities would result in exposures of cohort members being more similar than would be indicated by assigning city-specific annual average pollution levels to each member of the cohort. The more intercity migration there is, the more exposure will tend toward an intercity mean. If this is ignored, differences in exposure levels, that are proxied by differences in city-specific annual average PM levels, will be exaggerated, and will result in a downward bias of the PM coefficient (because a given difference in mortality rates is being associated with a larger difference in PM levels than is actually the case).

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[1]The total study population was 552,138 in 151 cities, however, only 295,223 individuals resided in 50 cities with fine particle data.

[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.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.12) and 95% confidence interval (1.06-1.19) associated with a change in *annual mean* $PM_{2.5}$ exposure of 24.5 $\mu g/m^3$ (based on the range from the original ACS study) [Krewski, 2000 #1805, Part II - Table 31].

Functional Form: Log-linear
Coefficient: 0.004626
Standard Error: 0.001205
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

Mortality - Median, All Cause [Krewski, 2000 #1805] - Reanalysis of Pope et al. [1995 #81]

Krewski et al. [2000 #1805] performed an analysis of Pope et al. [2000 #1805] using independently estimated city-specific annual *median* values as well. Fine particle estimates were obtained from EPA's Inhalable Particle Monitoring Network (IPMN) for the years 1979-1983 for the same 50 cities. Overall, the estimates showed good agreement with the median values used in the original investigation with one exception. The median fine particle concentration for Denver dropped from 16.1 to 7.8 $\mu g/m^3$, resulting in a larger range between the least and most polluted cities and a reduced relative risk. Since the original estimate could not be audited, Denver is included in the subsequent C-R function as there is no reason to believe that the monitoring data is invalid.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.14) and 95% confidence interval (1.06-1.22) associated with a change in *annual median* $PM_{2.5}$ exposure of 24.5 $\mu g/m^3$ (based on the range from the original ACS study) [Krewski, 2000 #1805, Part II - Table 31].

Functional Form: Log-linear
Coefficient: 0.005348
Standard Error: 0.001464
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

Mortality - Median, Random Effects with Regional Adjustment [Krewski, 2000 #1805] - Reanalysis of Pope et al. [1995 #81]

Krewski et al. [2000 #1805] also performed an analysis of Pope et al. [2000 #1805] using a random effects model to estimate a regionally-adjusted relative risk. The authors used an

indicator variable representing seven regions of the U.S. The regionally-adjusted estimate was comparable with the results from the standard Cox Proportional Hazards Model, which assumes that all observations are statistically independent.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.16) and 95% confidence interval (0.99-1.37) associated with a change in *annual median* $PM_{2.5}$ exposure of 24.5 $\mu g/m^3$ (based on the range from the original ACS study) [Krewski, 2000 #1805, Part II - Table 46].

Functional Form: Log-linear
Coefficient: 0.006058
Standard Error: 0.003383
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

Mortality - Median, Random Effects with Independent Cities [Krewski, 2000 #1805] - Reanalysis of Pope et al. [1995 #81]

Krewski et al. [2000 #1805] also performed an analysis of Pope et al. [2000 #1805] using a random effects approach to estimate an independent cities model. This approach incorporates between-city variation into second-stage modeling weights, thereby avoiding the assumption of independent observations. However, potential regional patterns in mortality may be overlooked, because the approach assumes that city-specific mortality rates are statistically independent. The independent cities estimate is considerably larger than the standard Cox Proportional Hazards Model, which assumes that all observations are statistically independent.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.29) and 95% confidence interval (1.12-1.48) associated with a change in *annual median* $PM_{2.5}$ exposure of 24.5 $\mu g/m^3$ (based on the range from the original ACS study) [Krewski, 2000 #1805, Part II - Table 46].

Functional Form: Log-linear
Coefficient: 0.010394
Standard Error: 0.002902
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

Mortality [Krewski, 2000 #1805] - Reanalysis of Dockery et al. [1993 #20]

Krewski et al. [2000 #1805] performed a validation and replication analysis of Dockery et al. [1993 #20]. The originial investigators examined the relationship between PM exposure and mortality in a cohort of 8,111 individuals aged 25 and older, living in six U.S. cities. They surveyed these individuals in 1974-1977 and followed their health status until 1991. While they used a smaller sample of individuals from fewer cities than the study by Pope et al., they used improved exposure estimates, a slightly broader study population (adults aged 25 and older; a higher proportion without a high school education), and a follow-up period nearly twice as long as that of Pope et al. [1995 #81]. Krewski et al. [2000 #1805, Part II - Table 52] found that educational status was a strong effect modifier of the PM - mortality relationship in both studies, with the strongest effect seen among the less educated. Perhaps because of these differences, Dockery et al. study found a larger effect of PM on premature mortality than that found by Pope et al.

After an audit of the air pollution data, demographic variables, and cohort selection process, Krewski et al. [2000 #1805] noted that a small portion of study participants were mistakenly censored early. The following C-R function is based on the risk estimate from the audited data, with the inclusion of those person-years mistakenly censored early.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.28) and 95% confidence interval (1.10-1.48) associated with a change in *annual mean* $PM_{2.5}$ exposure of 18.6 $\mu g/m^3$ to 29.6 $\mu g/m^3$ [Krewski et al.,2000 #1805, Part I - Table 19c].

Functional Form: Log-linear
Coefficient: 0.013272
Standard Error: 0.004070
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, 1995 #81]

Pope et al. [1995 #81] used a Cox proportional hazard model to estimate the impact of long-term PM exposure. They followed 295,223 individuals[3] ages 30 and over in 50 cities from September 1, 1982 to December 31, 1989, and related their survival to median $PM_{2.5}$ concentrations for 1979 to 1983. Pope et al. [1995 #81, Table 2] reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-

[3]The total study population was 552,138 in 151 cities, however, only 295,223 individuals resided in 50 cities with fine particle data.

519), and "all other" deaths,[4] and found that median $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. Pope et al. included only PM, so it is unclear to what extent it may be including the impacts of ozone or other gaseous pollutants.

Pope et al. [1995 #81] is the better of the two published prospective cohort studies: it has a larger population and includes more cities than the prospective cohort study by Dockery et al. [1993 #20]. Pope et al.'s study has several further advantages. The population followed in this study was largely Caucasian and middle class, decreasing the likelihood that interlocational differences in premature mortality were due in part to differences in race, socioeconomic status, or related factors. In addition, the PM coefficient in Pope et al. is likely to be biased downward, counteracting a possible upward bias associated with historical air quality trends discussed earlier. One source of this downward bias is the generally healthier study population, in comparison to poorer minority populations. Another source of downward bias is that intercity movement of cohort members was not considered in this study. Migration across study cities would result in exposures of cohort members being more similar than would be indicated by assigning city-specific annual average pollution levels to each member of the cohort. The more intercity migration there is, the more exposure will tend toward an intercity mean. If this is ignored, differences in exposure levels, that are proxied by differences in city-specific annual average PM levels, will be exaggerated, and will result in a downward bias of the PM coefficient (because a given difference in mortality rates is being associated with a larger difference in PM levels than is actually the case).

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.17) and 95% confidence interval (1.09-1.26) associated with a change in *annual median* $PM_{2.5}$ exposure of 24.5 $\mu g/m^3$ [Pope, 1995 #81, Table 2].

Functional Form: Log-linear
Coefficient: 0.006408
Standard Error: 0.001509
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

Mortality, All Cause [Dockery, 1993 #20]

Dockery et al. [1993 #20] examined the relationship between PM exposure and mortality in a cohort of 8,111 individuals aged 25 and older, living in six U.S. cities. They surveyed these individuals in 1974-1977 and followed their health status until 1991. While they used a smaller sample of individuals from fewer cities than the study by Pope et al., they used improved

[4]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.

exposure estimates, a slightly broader study population (adults aged 25 and older), and a follow-up period nearly twice as long as that of Pope et al. [1995 #81]. Perhaps because of these differences, Dockery et al. study found a larger effect of PM on premature mortality than that found by Pope et al.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.26) and 95% confidence interval associated (1.08-1.47) with a change in *annual mean* $PM_{2.5}$ exposure of 18.6 $\mu g/m^3$ [Dockery, 1993 #20, Tables 1 and 5].

Functional Form: Log-linear
Coefficient: 0.012425
Standard Error: 0.004228
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, 2002 #2240] - Based on ACS Cohort: Mean $PM_{2.5}$

The Pope et al. [2002 #2240] analysis is a longitudinal cohort tracking study that uses the same American Cancer Society (ACS) cohort as the original Pope et al. [1995 #81] study, and the Krewski et al. [2000 #1805] reanalysis. Pope et al. [2002 #2240] analyzed survival data for the cohort from 1982 through 1998, 9 years longer than the original Pope study. Pope et al. [2002 #2240] also obtained $PM_{2.5}$ data in 116 metropolitan areas collected in 1999, and the first three quarters of 2000. This is more metropolitan areas with $PM_{2.5}$ data than was available in the Krewski reanalysis (61 areas), or the original Pope study (50 areas), providing a larger size cohort.

They used a Cox proportional hazard model to estimate the impact of long-term PM exposure using three alternative measures of $PM_{2.5}$ exposure; metropolitan area-wide annual mean PM levels from the beginning of tracking period (’79-’83 PM data, conducted for 61 metropolitan areas with 359,000 individuals), annual mean PM from the end of the tracking period (’99-’00, for 116 areas with 500,000 individuals), and the average annual mean PM levels of the two periods (for 51 metropolitan areas, with 319,000 individuals). PM levels were lower in ’99-00 than in ’79 - ’83 in most cities, with the largest improvements occurring in cities with the highest original levels.

Pope et al. [2002 #2240] followed Krewski et al. [2000 #1805] and Pope et al. [1995 #81, 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.[5] Like the

[5]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.

earlier studies, Pope et al. [2002 #2240] found that mean $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. In addition, Pope et al. [2002 #2240] 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.

Pope et al. [2002 #2240] obtained ambient data on gaseous pollutants routinely monitored by EPA during the 1982-1998 observation period, including SO_2, NO_2, CO, and ozone. They did not find significant relationships between NO_2, CO, and ozone and premature mortality, but there were significant relationships between SO_2, and all-cause, cardiopulmonary, lung cancer and "all other" mortality.

'79-'83 Exposure

The coefficient and standard error for $PM_{2.5}$ using the '79-'83 PM data are estimated from the relative risk (1.041) and 95% confidence interval (1.008-1.075) associated with a change in *annual mean* exposure of 10.0 $\mu g/m^3$. Pope et al. [2002 #2240, Table 2].[6]

Functional Form: Log-linear
Coefficient: 0.004018
Standard Error: 0.001642
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

Mortality, Cardiopulmonary [Pope, 2002 #2240] - Based on ACS Cohort: Mean $PM_{2.5}$

Pope et al. [2002 #2240] followed Krewski et al. [2000 #1805] and Pope et al. [1995 #81, 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.[7] Like the earlier studies, Pope et al. [2002 #2240] found that mean $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. In addition, Pope et al. [2002 #2240] 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.

'79-'83 Exposure

The coefficient and standard error for $PM_{2.5}$ using the '79-'83 PM data are estimated from the relative risk (1.059) and 95% confidence interval (1.015-1.105) associated with a change in

[6]Note that we used an unpublished, final version of the paper that presents the relative risks with one more significant digit than that found in the published version. We chose to use this extra information to increase the precision of our estimates.

[7]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.

annual mean exposure of 10.0 μg/m^3. Pope et al. [2002 #2240, Table 2].[8]

Functional Form: Log-linear
Coefficient: 0.005733
Standard Error: 0.002167
Incidence Rate: county-specific annual cardiopulmonary mortality rate (ICD codes 401-440, 460-519) per person ages 30 and older
Population: population of ages 30 and older

Mortality, Lung Cancer [Pope, 2002 #2240] - Based on ACS Cohort: Mean $PM_{2.5}$

Pope et al. [2002 #2240] followed Krewski et al. [2000 #1805] and Pope et al. [1995 #81, 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.[9] Like the earlier studies, Pope et al. [2002 #2240] found that mean $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. In addition, Pope et al. [2002 #2240] 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.

'79-'83 Exposure

The coefficient and standard error for $PM_{2.5}$ using the '79-'83 PM data are estimated from the relative risk (1.082) and 95% confidence interval (1.011-1.158) associated with a change in *annual mean* exposure of 10.0 μg/m^3. Pope et al. [2002 #2240, Table 2].[10]

Functional Form: Log-linear
Coefficient: 0.007881
Standard Error: 0.003463
Incidence Rate: county-specific annual lung cancer mortality rate (ICD code 162) per person ages 30 and older
Population: population of ages 30 and older

[8]Note that we used an unpublished, final version of the paper that presents the relative risks with one more significant digit than that found in the published version. We chose to use this extra information to increase the precision of our estimates.

[9]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.

[10]Note that we used an unpublished, final version of the paper that presents the relative risks with one more significant digit than that found in the published version. We chose to use this extra information to increase the precision of our estimates.

Infant Mortality [Woodruff, 1997 #210]

In a study of four million infants in 86 U.S. metropolitan areas conducted from 1989 to 1991, Woodruff et al. [1997 #210] found a significant link between PM_{10} 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. PM_{10} exposure was significant for all-cause mortality. PM_{10} was also significant for respiratory mortality in average birth-weight infants, but not low birth-weight infants.

In addition to the work by Woodruff et al., work in Mexico City [Loomis, 1999 #756], the Czech Republic [Bobak, 1992 #1130], Sao Paulo [Saldiva, 1994 #167; Pereira, 1998 #164], and Beijing [Wang, 1997 #1132] provides additional evidence that particulate levels are significantly related to infant or child mortality, low birth weight or intrauterine mortality.

Conceptually, neonatal or child mortality could be added to the premature mortality predicted by Pope et al. [1995 #81], because the Pope function covers only the population over 30 years old.[11] However, the EPA Science Advisory Board recently advised the Agency not to include post-neonatal mortality in this analysis because the study is of a new endpoint and the results have not been replicated in other studies [U.S. EPA, 1999 #930, p. 12]. The estimated avoided incidences of neonatal mortality are estimated and presented as a sensitivity analysis, and are not included in the primary analysis.

Single Pollutant Model

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 $\mu g/m^3$ change in PM_{10} [Woodruff, 1997 #210, Table 3].

Functional Form: Logistic
Coefficient: 0.003922
Standard Error: 0.001221
Incidence Rate: county-specific annual postneonatal[12] infant deaths per infant under the age of one
Population: population of infants under one year old

[11] Predicted neonatal mortality could not be added to the premature mortality predicted by the daily (short-term exposure) mortality studies, however, because these studies cover all ages.

[12] Post-neonatal refers to infants that are 28 days to 364 days old.

Exhibit D-2 Concentration-Response (C-R) Functions for Particulate Matter and Short-Term Mortality

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Beta	Std Error	Functional Form	Notes
Non-Accidental	$PM_{2.5}$	Schwartz et al.	1996	6 cities	All	All	All	None	24-hr avg	0.001433	0.00013	Log-linear	
Non-Accidental	$PM_{2.5}$	Schwartz et al.	1996	6 cities	All	All	All	None	24-hr avg	0.002835	--	Log-linear	Lag Adjusted[2]
Chronic Lung	$PM_{2.5}$	Schwartz et al.	1996	6 cities	All	All	All	None	24-hr avg	0.006423	--	Log-linear	Lag Adjusted[2]

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.
2. Refer to the study summaries below for a discussion of the lag adjustment used for these functions.

Short-term Mortality

Short-term mortality studies are those that typically link daily air pollution levels with daily changes in mortality rates.

Short-Term Mortality, Non-Accidental [Schwartz, 1996 #98]

Schwartz et al. [1996 #98] pooled the results from six cities in the U.S. and found a significant relationship between daily $PM_{2.5}$ concentration and non-accidental mortality.[13] Abt Associates Inc. [1996 #239, p. 52] used the six $PM_{2.5}$ relative risks reported by Schwartz et al. in a three-step procedure to estimate a pooled $PM_{2.5}$ coefficient and its standard error. The first step estimates a random-effects pooled estimate of β; the second step uses an "empirical Bayes" procedure to reestimate the β for each study as a weighted average of the β reported for that location and the random effects pooled estimate; the third step estimates the underlying distribution of β, and uses a Monte Carlo procedure to estimate the standard error [Abt Associates Inc., 1996 #238, p. 65].

Single Pollutant Model

Abt Associates Inc. [1996 #239, p. 52] used the six $PM_{2.5}$ relative risks reported by Schwartz et al. in a three-step procedure to estimate a pooled $PM_{2.5}$ coefficient [Abt Associates Inc., 1996 #238, Exhibit 7.2] and its standard error [Abt Associates Inc., 1996 #238, Exhibit 7.2].

Functional Form: Log-linear
Coefficient: 0.001433
Standard Error: 0.000129
Incidence Rate: county-specific annual daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

Short-Term Mortality, Non-Accidental - Lag Adjusted [Schwartz, 1996 #98]

Schwartz et al. [1996 #98] pooled the results from six cities in the U.S. and found a significant relationship between daily $PM_{2.5}$ concentration and non-accidental mortality.[14] Abt Associates Inc. [1996 #239, p. 52] used the six $PM_{2.5}$ relative risks reported by Schwartz et al. in

[13]Schwartz et al. [1996 #98, p. 929] defined non-accidental mortality as all-cause mortality less deaths due to accidents and other external causes (ICD-9 codes: 800-999). Other external causes includes suicide, homicide, and legal intervention (National Center for Health Statistics, 1994).

[14]Schwartz et al. [1996 #98, p. 929] defined non-accidental mortality as all-cause mortality less deaths due to accidents and other external causes (ICD-9 codes: 800-999). Other external causes includes suicide, homicide, and legal intervention (National Center for Health Statistics, 1994).

a three-step procedure to estimate a pooled $PM_{2.5}$ coefficient and its standard error. The first step estimates a random-effects pooled estimate of β; the second step uses an "empirical Bayes" procedure to reestimate the β for each study as a weighted average of the β reported for that location and the random effects pooled estimate; the third step estimates the underlying distribution of β, and uses a Monte Carlo procedure to estimate the standard error [Abt Associates Inc., 1996 #238, p. 65]. In order to estimate the impact of daily $PM_{2.5}$ levels on daily mortality if a distributed lag model had been fit, the $PM_{2.5}$ coefficient is adjusted as described below.

Recent studies have found that an increase in PM levels on a given day can elevate mortality for several days following the exposure [Schwartz, 2000 #1550; Samet, 2000 #1810]. These studies have reported the results of distributed lag models for the relationship between PM_{10} and daily mortality. Schwartz [2000 #1550] examined the relationship between PM_{10} and daily mortality and reported results both for a single day lag model and an unconstrained distributed lag model. The unconstrained distributed lag model coefficient estimate is 0.0012818 and the single-lag model coefficient estimate is 0.0006479. A distributed lag adjustment factor can be constructed as the ratio of the estimated coefficient from the unconstrained distributed lag model to the estimated coefficient from the single-lag model reported in Schwartz (2000). The ratio of these estimates is 1.9784. In order to estimate the full impact of daily PM levels on daily mortality, we applied this ratio to the coefficient obtained from Schwartz et al. [1996 #98] for the association between $PM_{2.5}$ and daily mortality.

In applying the ratio derived from a PM_{10} study to $PM_{2.5}$, we assume that the same relationship between the distributed lag and single day estimates would hold for $PM_{2.5}$. Effect estimates for the PM_{10}-daily mortality relationship tend to be lower in magnitude than for $PM_{2.5}$, because fine particles are believed to be more closely associated with mortality than the coarse fraction of PM. If most of the increase in mortality is expected to be associated with the fine fraction of PM_{10}, then it is reasonable to assume that the same proportional increase in risk would be observed if a distributed lag model were applied to the $PM_{2.5}$ data.

Single Pollutant Model

The distributed lag model coefficient is estimated by applying the distributed lag adjustment factor of 1.9784 to the pooled $PM_{2.5}$ coefficient (0.001433) estimated by Abt Associates Inc. [1996 #238, Exhibit 7.2] from the six $PM_{2.5}$ relative risks reported by Schwartz et al. [1996 #98].[15]

Functional Form: Log-linear
Coefficient: 0.002835
Incidence Rate: county-specific annual daily non-accidental mortality rate (ICD codes <800) per person

[15] The distributed lag adjustment C-R function is only run for the point estimate. The standard error of this coefficient has not been estimated.

Population: population of all ages

Short-Term Mortality, Chronic Lung Disease - Lag Adjusted [Schwartz, 1996 #98]

Schwartz et al. [1996 #98] evaluated the relationship between daily $PM_{2.5}$ levels and short-term mortality in six U.S. cities. Schwartz pooled results across the six cities and found statistically significant associations between daily $PM_{2.5}$ levels and non-accidental mortality (ICD codes <800), along with mortality for ischemic heart disease (ICD codes 410-414), COPD (ICD codes 490-496), and pneumonia (ICD codes 480-486). A smaller association was found for PM_{10} and no significant associations were reported for $PM_{10-2.5}$. The C-R function for chronic lung disease mortality is based on the results of a single pollutant model using a two-day average of $PM_{2.5}$ [Schwartz et al., 1996 #98, Table 7]. In order to estimate the impact of daily $PM_{2.5}$ levels on daily mortality if a distributed lag model had been fit, the $PM_{2.5}$ coefficient is adjusted as described below.

Recent studies have found that an increase in PM levels on a given day can elevate mortality for several days following the exposure [Schwartz, 2000 #1550; Samet, 2000 #1810]. These studies have reported the results of distributed lag models for the relationship between PM_{10} and daily mortality. Schwartz [2000 #1550] examined the relationship between PM_{10} and daily mortality and reported results both for a single day lag model and an unconstrained distributed lag model. The unconstrained distributed lag model coefficient estimate is 0.0012818 and the single-lag model coefficient estimate is 0.0006479. A distributed lag adjustment factor can be constructed as the ratio of the estimated coefficient from the unconstrained distributed lag model to the estimated coefficient from the single-lag model reported in Schwartz (2000). The ratio of these estimates is 1.9784. In order to estimate the full impact of daily PM levels on daily mortality, we applied this ratio to the coefficient obtained from Schwartz et al. [1996 #98] for the association between $PM_{2.5}$ and daily mortality.

In applying the ratio derived from a PM_{10} study to $PM_{2.5}$, we assume that the same relationship between the distributed lag and single day estimates would hold for $PM_{2.5}$. Effect estimates for the PM_{10}-daily mortality relationship tend to be lower in magnitude than for $PM_{2.5}$, because fine particles are believed to be more closely associated with mortality than the coarse fraction of PM. If most of the increase in mortality is expected to be associated with the fine fraction of PM_{10}, then it is reasonable to assume that the same proportional increase in risk would be observed if a distributed lag model were applied to the $PM_{2.5}$ data.

Single Pollutant Model

The $PM_{2.5}$ coefficient is based on a reported 3.3% increase in COPD mortality associated with a 10 $\mu g/m^3$ change in two-day average $PM_{2.5}$ levels [Schwartz, 1996 #98, Table 7]. This coefficient (0.003247) is then multiplied by the distributed lag adjustment factor of 1.9784 to estimate a distributed lag model coefficient.

Functional Form: Log-linear
Coefficient: 0.006423
Incidence Rate: county-specific annual daily chronic lung disease mortality rate (ICD codes 490-496)
Population: population of all ages

Exhibit D-3 Concentration-Response (C-R) Functions for Particulate Matter and Chronic Illness

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time	Beta	Std Error	Functional Form
Chronic Bronchitis	$PM_{2.5}$	Abbey et al.	1995	SF, SD, South Coast Air Basin	27+	All	All	None	Annual Avg	0.0137	0.00680	Logistic
Chronic Bronchitis	PM_{10}	Schwartz	1993	53 cities	30+	All	All	None	Annual Avg	0.0123	0.00434	Logistic

Chronic Illness

Schwartz [1993 #240] and Abbey et al. [1993 #245;, 1995 #452] provide evidence that PM exposure over a number of years gives rise to the development of chronic bronchitis in the U.S., and a recent study by McDonnell et al. [1999 #1153] provides evidence that ozone exposure is linked to the development of asthma in adults. These results are consistent with research that has found chronic exposure to pollutants leads to declining pulmonary functioning [Abbey, 1998 #249; AckermannLiebrich, 1997 #117; Detels, 1991 #345].[16]

Chronic Bronchitis [Abbey, 1995 #452, California]

Abbey et al. [1995 #452] examined the relationship between estimated $PM_{2.5}$ (annual mean from 1966 to 1977), PM_{10} (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; PM_{10} 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 C-R function is based on the results of the single pollutant model presented in Table 2.

Single Pollutant Model

The estimated coefficient (0.0137) is presented for a one $\mu g/m^3$ change in $PM_{2.5}$ [Abbey, 1995 #452, 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 $\mu g/m^3$ change in $PM_{2.5}$ [Abbey, 1995 #452, Table 2].

Functional Form: Logistic
Coefficient: 0.0137
Standard Error: 0.00680
Incidence Rate: annual bronchitis incidence rate per person [Abbey, 1993 #245, Table 3] = 0.00378

[16] There are a limited number of studies that have estimated the impact of air pollution on chronic bronchitis. An important hindrance is the lack of health data and the associated air pollution levels over a number of years.

Population: population of ages 27 and older[17] without chronic bronchitis = 95.57%[18] of population 27+

Chronic Bronchitis [Schwartz, 1993 #240]

Schwartz [1993 #240] examined survey data collected from 3,874 adults ranging in age from 30 to 74, and living in 53 urban areas in the U.S. The survey was conducted between 1974 and 1975, as part of the National Health and Nutrition Examination Survey, and is representative of the non-institutionalized U.S. population. Schwartz [1993 #240, Table 3] reported chronic bronchitis prevalence rates in the study population by age, race, and gender. Non-white males under 52 years old had the lowest rate (1.7%) and white males 52 years and older had the highest rate (9.3%). The study examined the relationship between the prevalence of reported chronic bronchitis, asthma, shortness of breath (dyspnea) and respiratory illness[19], and the annual levels of TSP, collected in the year prior to the survey (TSP was the only pollutant examined in this study). TSP was significantly related to the prevalence of chronic bronchitis, and marginally significant for respiratory illness. No effect was found for asthma or dyspnea. The C-R function for PM_{10} is estimated from the results of the single pollutant model reported for TSP.

Single Pollutant Model

The estimated coefficient is based on the odds ratio (1.07) associated with 10 $\mu g/m^3$ change in TSP [Schwartz, 1993 #240, p. 9]. Assuming that PM_{10} is 55 percent of TSP[20] and that particulates greater than ten micrometers are harmless, the coefficient is calculated by dividing the TSP coefficient by 0.55. The standard error for the coefficient is calculated from the 95% confidence interval for the odds ratio (1.02 to 1.12) [Schwartz, 1993 #240, p. 9].

Schwartz [1993 #240] examined the *prevalence* of chronic bronchitis, not its *incidence*. To use Schwartz's study and still estimate the change in incidence, there are at least two possible approaches. The first is to simply assume that it is appropriate to use the baseline *incidence* of chronic bronchitis in a C-R function with the estimated coefficient from Schwartz's study, to directly estimate the change in incidence. The second is to estimate the percentage change in the prevalence rate for chronic bronchitis using the estimated coefficient from Schwartz's study in a C-R function, and then to assume that this percentage change applies to a baseline incidence rate obtained from another source. (That is, if the prevalence declines by 25 percent with a drop in

[17] Using the same data set, Abbey et al. [1995 #247, p. 140] reported that the respondents in 1977 ranged in age from 27 to 95.

[18] The American Lung Association [2002 #2357, Table 4] reports a chronic bronchitis prevalence rate for ages 18 and over of 4.43% [American Lung Association, 2002 #2357, Table 4].

[19] Respiratory illness defined as a significant condition, coded by an examining physician as ICD-8 code 460-519.

[20] The conversion of TSP to PM_{10} is from ESEERCO [1994 #323, p. V-5], who cited studies by EPA [1986 #236] and the California Air Resources Board [1982 #329].

PM, then baseline incidence drops by 25 percent with the same drop in PM.) This analysis is using the latter approach, and estimates a percentage change in prevalence which is then applied to a baseline incidence rate. The scaling factor used in the C-R function is the ratio of chronic bronchitis incidence rate (estimated from Abbey et al. [1993 #245]) to chronic bronchitis prevalence rate (estimated from American Lung Association [2002 #2357, Table 4]).

Functional Form: Logistic
Coefficient: 0.0123
Standard Error: 0.00434
Incidence Rate: annual chronic bronchitis prevalence rate per person [American Lung Association, 2002 #2357, Table 4] = 0.0443
Population: population of ages 30 and older without chronic bronchitis = 95.57%[21] of population 30+
Adjustment Factor: ratio of chronic bronchitis incidence to chronic bronchitis prevalence = 0.00378/0.0443 = 0.085 [Abbey, 1993 #245, Table 3; American Lung Association, 2002 #2357, Table 4]

[21] The American Lung Association [2002 #2357, Table 4] reports a chronic bronchitis prevalence rate for ages 18 and over of 4.43% [American Lung Association, 2002 #2357, Table 4].

Exhibit D-4 Concentration-Response (C-R) Functions for Particulate Matter and Hospital Admissions

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Beta	Std Error	Functional Form
Asthma	$PM_{2.5}$	Sheppard et al.	1999	Seattle, WA	<65	All	All	CO	24-hr avg	0.002505	0.001045	Log-linear
Chronic Lung Disease	$PM_{2.5}$	Lippmann et al.	2000	Detroit, MI	65+	All	All	O_3	24-hr avg	0.001089	0.002420	Log-linear
Chronic Lung Disease	$PM_{2.5}$	Moolgavkar	2000	Los Angeles, CA	65+	All	All	CO	24-hr avg	0.0008	0.001000	Log-linear
Chronic Lung Disease	$PM_{2.5}$	Moolgavkar	2000	Los Angeles, CA	18-64	All	All	CO	24-hr avg	0.0020	0.000909	Log-linear
Chronic Lung Disease (less Asthma)	PM_{10}	Samet et al.	2000	14 cities	65+	All	All	None	24-hr avg	0.002839	0.001351	Log-linear
Pneumonia	$PM_{2.5}$	Lippmann et al.	2000	Detroit, MI	65+	All	All	O_3	24-hr avg	0.004480	0.001918	Log-linear
Pneumonia	PM_{10}	Samet et al.	2000	14 cities	65+	All	All	None	24-hr avg	0.002049	0.000570	Log-linear
All Cardiovascular	$PM_{2.5}$	Moolgavkar	2000	Los Angeles, CA	65+	All	All	CO	24-hr avg	0.0005	0.000556	Log-linear
All Cardiovascular	$PM_{2.5}$	Moolgavkar	2000	Los Angeles, CA	18-64	All	All	CO	24-hr avg	0.0009	0.000500	Log-linear
All Cardiovascular	PM_{10}	Samet et al.	2000	14 cities	65+	All	All	None	24-hr avg	0.001183	0.000111	Log-linear
Dysrhythmia	$PM_{2.5}$	Lippmann et al.	2000	Detroit, MI	65+	All	All	O_3	24-hr avg	0.002138	0.002525	Log-linear
Heart Failure	$PM_{2.5}$	Lippmann et al.	2000	Detroit, MI	65+	All	All	O_3	24-hr avg	0.004668	0.001650	Log-linear
Ischemic Heart Disease	$PM_{2.5}$	Lippmann et al.	2000	Detroit, MI	65+	All	All	O_3	24-hr avg	0.001116	0.001339	Log-linear

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Hospitalizations

Hospital Admissions for Asthma [Sheppard, 1999 #792, Seattle]

Sheppard et al. [1999 #792] studied the relation between air pollution in Seattle and nonelderly (<65) hospital admissions for asthma from 1987 to 1994. They used air quality data for PM_{10}, $PM_{2.5}$, coarse $PM10_{10\text{-}2.5}$, SO_2, ozone, and CO in a Poisson regression model with control for time trends, seasonal variations, and temperature-related weather effects.[22] They found asthma hospital admissions associated with PM_{10}, $PM_{2.5}$, $PM_{10\text{-}2.5}$, 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 $PM_{2.5}$ and CO. Results for other co-pollutant models were not reported. The $PM_{2.5}$ C-R function is based on the multipollutant model.

Multipollutant Model ($PM_{2.5}$ and CO)

The coefficient and standard error for the co-pollutant model with CO are calculated from a relative risk of 1.03 (95% CI 1.01-1.06) for an 11.8 $\mu g/m^3$ increase[23] in $PM_{2.5}$ [Sheppard, 1999 #792, p. 28].

Functional Form: Log-linear
Coefficient: 0.002505
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 [Lippmann, 2000 #2328, Detroit]

Lippmann et al. [2000 #2328] 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}, $PM_{2.5}$, and $PM_{10\text{-}2.5}$ 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-

[22] $PM_{2.5}$ levels were estimated from light scattering data.

[23] The reported IQR change in the abstract and text is smaller than reported in Table 3. We assume the change reported in the abstract and text to be correct because greater number of significant figures are reported.

486), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ 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 $PM_{2.5}$ C-R function is based on results of the co-pollutant model with ozone.

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.040 (95% CI 0.877-1.234) for a 36 μg/m^3 increase in $PM_{2.5}$ [Lippmann, 2000 #2328, Table 14, p. 26].

Functional Form: Log-linear
Coefficient: 0.001089
Standard Error: 0.002420
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 Chronic Lung Disease [Moolgavkar, 2000 #2152]

Moolgavkar [2000 #2152] 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, SO_2, NO_2, CO, and PM_{10} 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 PM_{10}. 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.

The $PM_{2.5}$ C-R functions are based on the co-pollutant models ($PM_{2.5}$ and CO) reported for the 20-64 and 65+ age groups. 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, we selected the lag models with the greatest effect estimates for use in the C-R functions.

Ages 65 and older

Multipollutant Model ($PM_{2.5}$ and CO)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 0.8[24] and t-statistic of 0.8 for a 10 μg/m^3 increase in $PM_{2.5}$ in the two-day lag model [Moolgavkar, 2000 #2152, Table 3, p. 80].

Functional Form: Log-linear
Coefficient: 0.0008
Standard Error: 0.001000
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

Ages 18 to 64[25]

Multipollutant Model ($PM_{2.5}$ and CO)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 2.0[26] and t-statistic of 2.2 for a 10 μg/m^3 increase in $PM_{2.5}$ in the two-day lag model [Moolgavkar, 2000 #2152, Table 4, p. 81].

Functional Form: Log-linear
Coefficient: 0.0020
Standard Error: 0.000909
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease admissions per person 18-64 (ICD codes 490-492, 494-496)[27]
Population: population of ages 18 to 64

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[24] In a log-linear model, the percent change is equal to (RR - 1) * 100. In this study, Moolgavkar defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 0.8 would result in a relative risk of 1.008 and coefficient of 0.000797. The "estimated" percent change, as reported by Moolgavkar, of 0.8 results in a relative risk of 1.008032 and coefficient of 0.0008.

[25] Although Moolgavkar [2000 #2152] 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.

[26] In a log-linear model, the percent change is equal to (RR - 1) * 100. In this study, Moolgavkar defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 2.0 would result in a relative risk of 1.020 and coefficient of 0.001980. The "estimated" percent change, as reported by Moolgavkar, of 2.0 results in a relative risk of 1.020201 and coefficient of 0.002.

[27] Moolgavkar [2000 #2152] reports results for ICD codes 490-496. In order to avoid double counting non-elderly asthma hospitalizations (ICD code 493) with Sheppard et al. [1999 #792] in a total benefits estimation, we have excluded ICD code 493 from the baseline incidence rate used in this function.

Hospital Admissions for Chronic Lung Disease (less Asthma) [Samet, 2000 #1810, 14 Cities]

Samet et al. [2000 #1810] examined the relationship between air pollution and hospital admissions for individuals of ages 65 and over in 14 cities across the country.[28] Cities were selected on the basis of available air pollution data for at least four years between 1985 and 1994 during which at least 50% of days had observations between the city-specific start and end of measurements. Hospital admissions were obtained from the Health Care Financing Administration (HCFA) for the years 1992 and 1993. Poisson regression was used in the analysis with unconstrained distributed lag models to examine the possibility that air pollution affects hospital admissions on not only the same day but on later days as well. The use of unconstrained distributed lags has the advantages of (1) not inappropriately biasing down risk estimates due to tight constraints (e.g. one day lag) and (2) not leaving the often arbitrary choice of lag period to the investigator's discretion. The C-R functions are based on the pooled estimate across all 14 cities, using the unconstrained distributed lag model and fixed or random effects estimates, depending on the results of a test for heterogeneity.

For this analysis, the unadjusted, base models for the effect of PM_{10} on hospital admissions were used. The authors performed a second-stage regression to estimate the impact of SO_2 and O_3 on the PM_{10} - hospitalization effect. For ozone, the PM_{10} effect in each city was regressed on the correlation between ozone and particulate matter (the slope of a PM_{10} vs. O_3 regression) in that city. The fitted line for this regression will have a slope of zero if there is no relationship, meaning that the effect of PM_{10} is not dependent on the correlation between PM_{10} and O_3. The adjusted point estimate was obtained by determining the PM_{10} effect when the correlation between the pollutants is zero (i.e. the y-intercept of the fitted line). The effect of O_3 adjustment on the PM_{10} - hospitalization relationship appeared to be minimal except for the case of COPD. In this case, adjustment increased the point estimate of the independent particulate matter effect. The variance of this estimate, however, was quite large and the confidence intervals of the adjusted and unadjusted estimates overlapped substantially. For these reasons, there appeared to be little impact of O_3 adjustment.[29] Furthermore, the statistical power and robustness of this second-stage approach to co-pollutant adjustment are in question because of the small number of observations used in the regression (14 cities) and the potential for one or two observations to dramatically impact the results.[30] Finally, for the case of COPD, adjustment led to an increased PM_{10} independent effect, meaning that if the adjustment is valid, the impact on hospital admissions will be underestimated rather than overestimated.

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[28]The cities under investigation include: Birmingham, Boulder, Canton, Chicago, Colorado Springs, Detroit, Minneapolis/St. Paul, Nashville, New Haven, Pittsburgh, Provo/Orem, Seattle, Spokane, Youngstown.

[29] Joel Schwartz (co-author), personal communication.

[30] Commentary from the Health Review Committee (Samet et al., 2000, p.77) states that "[w]hile the approach used in the morbidity analysis is novel...the question arises as to the adequacy of statistical power for performing these analyses."

Single Pollutant Model

The estimated PM_{10} coefficient is based on a 2.88 percent increase (RR = 1.0288) in admissions due to a PM_{10} change of 10.0 μg/m^3 [Samet, 2000 #1810, Part II - Table 14][31]. The standard error is estimated from the reported lower (0.19 percent) and upper bounds (5.64 percent) of the percent increase [Samet, 2000 #1810, Part II - Table 14].

Functional Form: Log-linear
Coefficient: 0.002839
Standard Error: 0.001351
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease per person 65+ (ICD codes 490-492, 494-496)
Population: population of ages 65 and older

Hospital Admissions for Pneumonia [Lippmann, 2000 #2328, Detroit]

Lippmann et al. [2000 #2328] 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}, $PM_{2.5}$, and $PM_{10-2.5}$ 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), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ 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 $PM_{2.5}$ C-R function is based on the results of the co-pollutant model with ozone.

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.175 (95% CI 1.026-1.345) for a 36 μg/m^3 increase in $PM_{2.5}$ [Lippmann, 2000 #2328, Table 14, p. 26].

Functional Form: Log-linear
Coefficient: 0.004480
Standard Error: 0.001918

[31] The random effects estimate of the unconstrained distributed lag model was chosen for COPD admissions since the chi-square test of heterogeneity was significant (see Samet et al., 2000, Part II - Table 15).

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

Hospital Admissions for Pneumonia [Samet, 2000 #1810, 14 Cities]

Samet et al. [2000 #1810] examined the relationship between air pollution and hospital admissions for individuals of ages 65 and over in 14 cities across the country.[32] Cities were selected on the basis of available air pollution data for at least four years between 1985 and 1994 during which at least 50% of days had observations between the city-specific start and end of measurements. Hospital admissions were obtained from the Health Care Financing Administration (HCFA) for the years 1992 and 1993. Poisson regression was used in the analysis with unconstrained distributed lag models to examine the possibility that air pollution affects hospital admissions on not only the same day but on later days as well. The use of unconstrained distributed lags has the advantages of (1) not inappropriately biasing down risk estimates due to tight constraints (e.g. one day lag) and (2) not leaving the often arbitrary choice of lag period to the investigator's discretion. The C-R functions are based on the pooled estimate across all 14 cities, using the unconstrained distributed lag model and fixed or random effects estimates, depending on the results of a test for heterogeneity.

For this analysis, the unadjusted, base models for the effect of PM_{10} on hospital admissions were used. The authors performed a second-stage regression to estimate the impact of SO_2 and O_3 on the PM_{10} - hospitalization effect. For ozone, the PM_{10} effect in each city was regressed on the correlation between ozone and particulate matter (the slope of a PM_{10} vs. O_3 regression) in that city. The fitted line for this regression will have a slope of zero if there is no relationship, meaning that the effect of PM_{10} is not dependent on the correlation between PM_{10} and O_3. The adjusted point estimate was obtained by determining the PM_{10} effect when the correlation between the pollutants is zero (i.e. the y-intercept of the fitted line). The effect of O_3 adjustment on the PM_{10} - hospitalization relationship appeared to be minimal except for the case of COPD. In this case, adjustment increased the point estimate of the independent particulate matter effect. The variance of this estimate, however, was quite large and the confidence intervals of the adjusted and unadjusted estimates overlapped substantially. For these reasons, there appeared to be little impact of O_3 adjustment.[33] Furthermore, the statistical power and robustness of this second-stage approach to co-pollutant adjustment are in question because of the small number of observations used in the regression (14 cities) and the potential for one or two observations to dramatically impact the results.[34] Finally, for the case of COPD, adjustment led to an increased PM_{10} independent effect, meaning that if the adjustment is valid, the impact on hospital admissions will be underestimated rather than overestimated.

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[32]The cities under investigation include: Birmingham, Boulder, Canton, Chicago, Colorado Springs, Detroit, Minneapolis/St. Paul, Nashville, New Haven, Pittsburgh, Provo/Orem, Seattle, Spokane, Youngstown.

[33] Joel Schwartz (co-author), personal communication.

[34] Commentary from the Health Review Committee (Samet et al., 2000, p.77) states that "[w]hile the approach used in the morbidity analysis is novel...the question arises as to the adequacy of statistical power for performing these analyses."

Single Pollutant Model

The estimated PM_{10} coefficient is based on a 2.07 percent increase (RR = 1.0207) in admissions due to a PM_{10} change of 10.0 μg/m^3 [Samet, 2000 #1810, Part II - Table 14][35]. The standard error is estimated from the reported lower (0.94 percent) and upper bounds (3.22 percent) of the percent increase [Samet, 2000 #1810, Part II - Table 14].

Functional Form: Log-linear
Coefficient: 0.002049
Standard Error: 0.000570
Incidence Rate: region-specific daily hospital admission rate for pneumonia per person 65+ (ICD codes 480-487)
Population: population of ages 65 and older

Hospital Admissions for All Cardiovascular [Moolgavkar, 2000 #2029, Los Angeles]

Moolgavkar [2000 #2029] 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. $PM_{2.5}$ 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 $PM_{2.5}$. The strongest overall effects were observed for SO_2 and CO. In a single pollutant model, $PM_{2.5}$ was statistically significant for lag 0 and lag 1. In co-pollutant models with CO, the $PM_{2.5}$ effect dropped out and CO remained significant. For ages 20-64, SO_2 and CO exhibited the strongest effect and any $PM_{2.5}$ effect dropped out in co-pollutant models with CO. The $PM_{2.5}$ C-R functions are based on co-pollutant ($PM_{2.5}$ and CO) models.

Ages 65 and older

Multipollutant Model ($PM_{2.5}$ and CO)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 0.5[36] and t-statistic of 0.9 for a 10 μg/m^3 increase in $PM_{2.5}$ in the one day lag

[35] The random effects estimate of the unconstrained distributed lag model was chosen for pneumonia admissions since the chi-square test of heterogeneity was significant (see Samet et al., 2000, Part II - Table 15).

[36] In a log-linear model, the percent change is equal to (RR - 1) * 100. In a similar hospitalization study by Moolgavkar [2000 #2152], he defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 0.5 would result in a relative risk of 1.005 and coefficient of 0.000499. Assuming that the 0.5 is the "estimated" percent change described previously would result in a relative risk of 1.005013 and coefficient of 0.0005. We assume that the "estimated" percent changes reported in this study reflect the definition from [Moolgavkar, 2000 #2152].

model [Moolgavkar, 2000 #2029, Table 3, p. 1202].

Functional Form: Log-linear
Coefficient: 0.0005
Standard Error: 0.000556
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions per person 65+ (ICD codes 390-409, 411-459)[37]
Population: population of ages 65 and older

Ages 18 to 64[38]

Multipollutant Model ($PM_{2.5}$ and CO)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 0.9[39] and t-statistic of 1.8 for a 10 $\mu g/m^3$ increase in $PM_{2.5}$ in the zero lag model [Moolgavkar, 2000 #2029, Table 4, p. 1203].

Functional Form: Log-linear
Coefficient: 0.0009
Standard Error: 0.000500
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions per person ages 18 to 64 (ICD codes 390-409, 411-459)[40]
Population: population of ages 18 to 64

[37] Moolgavkar [2000 #2029] reports results for ICD codes 390-429. In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. [2001 #2157]. The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

[38] Although Moolgavkar [2000 #2029] 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.

[39] In a log-linear model, the percent change is equal to (RR - 1) * 100. In a similar hospitalization study by Moolgavkar [2000 #2152], he defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 0.9 would result in a relative risk of 1.009 and coefficient of 0.000896. Assuming that the 0.9 is the "estimated" percent change described previously would result in a relative risk of 1.009041 and coefficient of 0.0009. We assume that the "estimated" percent changes reported in this study reflect the definition from [Moolgavkar, 2000 #2152].

[40] Moolgavkar [2000 #2029] reports results that include ICD code 410 (heart attack). In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. [2001 #2157]. The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

Hospital Admissions for All Cardiovascular [Samet, 2000 #1810, 14 Cities]

Samet et al. [2000 #1810] examined the relationship between air pollution and hospital admissions for individuals of ages 65 and over in 14 cities across the country.[41] Cities were selected on the basis of available air pollution data for at least four years between 1985 and 1994 during which at least 50% of days had observations between the city-specific start and end of measurements. Hospital admissions were obtained from the Health Care Financing Administration (HCFA) for the years 1992 and 1993. Poisson regression was used in the analysis with unconstrained distributed lag models to examine the possibility that air pollution affects hospital admissions on not only the same day but on later days as well. The use of unconstrained distributed lags has the advantages of (1) not inappropriately biasing down risk estimates due to tight constraints (e.g. one day lag) and (2) not leaving the often arbitrary choice of lag period to the investigator's discretion. The C-R functions are based on the pooled estimate across all 14 cities, using the unconstrained distributed lag model and fixed or random effects estimates, depending on the results of a test for heterogeneity.

For this analysis, the unadjusted, base models for the effect of PM_{10} on hospital admissions were used. The authors performed a second-stage regression to estimate the impact of SO_2 and O_3 on the PM_{10} - hospitalization effect. For ozone, the PM_{10} effect in each city was regressed on the correlation between ozone and particulate matter (the slope of a PM_{10} vs. O_3 regression) in that city. The fitted line for this regression will have a slope of zero if there is no relationship, meaning that the effect of PM_{10} is not dependent on the correlation between PM_{10} and O_3. The adjusted point estimate was obtained by determining the PM_{10} effect when the correlation between the pollutants is zero (i.e. the y-intercept of the fitted line). The effect of O_3 adjustment on the PM_{10} - hospitalization relationship appeared to be minimal except for the case of COPD. In this case, adjustment increased the point estimate of the independent particulate matter effect. The variance of this estimate, however, was quite large and the confidence intervals of the adjusted and unadjusted estimates overlapped substantially. For these reasons, there appeared to be little impact of O_3 adjustment.[42] Furthermore, the statistical power and robustness of this second-stage approach to co-pollutant adjustment are in question because of the small number of observations used in the regression (14 cities) and the potential for one or two observations to dramatically impact the results.[43] Finally, for the case of COPD, adjustment led to an increased PM_{10} independent effect, meaning that if the adjustment is valid, the impact on hospital admissions will be underestimated rather than overestimated.

[41]The cities under investigation include: Birmingham, Boulder, Canton, Chicago, Colorado Springs, Detroit, Minneapolis/St. Paul, Nashville, New Haven, Pittsburgh, Provo/Orem, Seattle, Spokane, Youngstown.

[42] Joel Schwartz (co-author), personal communication.

[43] Commentary from the Health Review Committee (Samet et al., 2000, p.77) states that "[w]hile the approach used in the morbidity analysis is novel...the question arises as to the adequacy of statistical power for performing these analyses."

Single Pollutant Model

The estimated PM_{10} coefficient is based on a 1.19 percent increase (RR = 1.0119) in admissions due to a PM_{10} change of 10.0 μg/m^3 [Samet, 2000 #1810, Part II - Table 14][44]. The standard error is estimated from the reported lower (0.97 percent) and upper bounds (1.41 percent) of the percent increase [Samet, 2000 #1810, Part II - Table 14].

Functional Form: Log-linear
Coefficient: 0.001183
Standard Error: 0.000111
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular disease per person 65+ (ICD codes 390-459)
Population: population of ages 65 and older

Hospital Admissions for Dysrhythmia [Lippmann, 2000 #2328, Detroit]

Lippmann et al. [2000 #2328] 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}, $PM_{2.5}$, and $PM_{10-2.5}$ 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), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ 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 $PM_{2.5}$ C-R function is based on the co-pollutant model with ozone.

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.080 (95% CI 0.904-1.291) for a 36 μg/m^3 increase in $PM_{2.5}$ [Lippmann, 2000 #2328, Table 14, p. 27].

Functional Form: Log-linear
Coefficient: 0.002138

[44] The fixed effects estimate of the unconstrained distributed lag model was chosen for CVD admissions since the chi-square test of heterogeneity was non-significant (see Samet et al., 2000, Part II - Table 15).

Standard Error: 0.002525
Incidence Rate: region-specific daily hospital admission rate for dysrhythmia admissions per person 65+ (ICD code 427)
Population: population of ages 65 and older

Hospital Admissions for Heart Failure [Lippmann, 2000 #2328, Detroit]

Lippmann et al. [2000 #2328] 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}, $PM_{2.5}$, and $PM_{10-2.5}$ 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), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ 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 $PM_{2.5}$ C-R function is based on the co-pollutant model with ozone.

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.183 (95% CI 1.053-1.329) for a 36 $\mu g/m^3$ increase in $PM_{2.5}$ [Lippmann, 2000 #2328, Table 14, p. 27].

Functional Form: Log-linear
Coefficient: 0.004668
Standard Error: 0.001650
Incidence Rate: region-specific daily hospital admission rate for heart failure admissions per person 65+ (ICD code 428)
Population: population of ages 65 and older

Hospital Admissions for Ischemic Heart Disease [Lippmann, 2000 #2328, Detroit]

Lippmann et al. [2000 #2328] 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}, $PM_{2.5}$, and $PM_{10-2.5}$ 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), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ 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 $PM_{2.5}$ C-R function is based on the co-pollutant model with ozone.

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.041 (95% CI 0.947-1.144) for a 36 $\mu g/m^3$ increase in $PM_{2.5}$ [Lippmann, 2000 #2328, Table 14, p. 27].

Functional Form: Log-linear
Coefficient: 0.001116
Standard Error: 0.001339
Incidence Rate: region-specific daily hospital admission rate for ischemic heart disease admissions per person 65+ (ICD codes 411-414)[45]
Population: population of ages 65 and older

[45] Lippmann et al. [2000 #2328] reports results for ICD codes 410-414. In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. [2001 #2157]. The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

Exhibit D-5 Concentration-Response (C-R) Functions for Particulate Matter and Emergency Room Visits

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Beta	Std Error	Functional Form
Asthma	$PM_{2.5}$	Norris et al.	1999	Seattle, WA	<18	All	All	NO_2, SO_2	24-hr avg	0.016527	0.004139	Log-linear
Asthma	PM_{10}	Schwartz et al.	1993	Seattle, WA	<65	All	All	None	24-hr avg	0.00367	0.00126	Log-linear

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Emergency Room Visits

Emergency Room Visits for Asthma [Norris, 1999 #1263]

Norris et al. [1999 #1263] 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 $PM_{2.5}$), 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. The $PM_{2.5}$ C-R function is on the multipollutant model reported.

Multipollutant Model ($PM_{2.5}$, NO_2, and SO_2)

In a model with NO_2 and SO_2, the $PM_{2.5}$ coefficient and standard error are calculated from a relative risk of 1.17 (95% CI 1.08-1.26) for a 9.5 $\mu g/m^3$ increase in $PM_{2.5}$ [Norris, 1999 #1263, 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

Emergency Room Visits for Asthma [Schwartz, 1993 #860, Seattle]

Schwartz et al. [1993 #680] examined the relationship between air quality and emergency room visits for asthma (ICD codes 493,493.01,493.10,493.90,493.91) in persons under 65 and 65 and over, living in Seattle from September 1989 to September 1990. Using single-pollutant models they found daily levels of PM_{10} linked to ER visits in individuals ages under 65, and they found no effect in individuals ages 65 and over. They did not find a significant effect for SO_2 and ozone in either age group. The results of the single pollutant model for PM_{10} are used in this analysis.

Single Pollutant Model

The PM_{10} coefficient and standard error are reported by Schwartz et al. [1993 #860, p. 829] for a unit $\mu g/m^3$ increase in four-day average PM_{10} levels.

Functional Form: Log-linear
Coefficient: 0.00367

Standard Error: 0.00126
Incidence Rate: region-specific daily emergency room rate for asthma admissions per person <65 (ICD code 493)
Population: population of ages under 65

Exhibit D-6 Concentration-Response (C-R) Functions for Particulate Matter and Acute Effects

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Beta	Std Error	Functional Form
Acute Bronchitis	$PM_{2.5}$	Dockery et al.	1996	24 communities	8-12	All	All	None	Annual Avg	0.027212	0.017096	Logistic
Acute Myocardial Infarction, Nonfatal	$PM_{2.5}$	Peters et al.	2001	Boston, MA	18+	All	All	None	24-hr avg	0.024121	0.009285	Logistic
Any of 19 Respiratory Symptoms	PM_{10}	Krupnick	1990	Los Angeles, CA	18-64	All	All	O_3	24-hr avg	0.000461	0.000239	Linear
Lower Respiratory Symptoms	$PM_{2.5}$	Schwartz and Neas	2000	6 cities	7-14	All	All	$PM_{10-2.5}$	24-hr avg	0.016976	0.006680	Logistic
Minor Restricted Activity Days	$PM_{2.5}$	Ostro and Rothschild	1989	nationwide	18-64	All	All	O_3	24-hr avg	0.00741	0.00070	Log-linear
Work Loss Days	$PM_{2.5}$	Ostro	1987	nationwide	18-64	All	All	None	24-hr avg	0.0046	0.00036	Log-linear

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Acute Effects

Acute Bronchitis [Dockery, 1996 #25]

Dockery et al. [1996 #25] 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 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.[46] They also found nitrates were linked to asthma, and sulfates linked to chronic phlegm. It is important to note that thestudy 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. Earlier work, by Dockery et al. [1989 #327], based on six U.S. cities, found acute bronchitis and chronic cough significantly related to PM_{15}. Because it is based on a larger sample, the Dockery et al. [1996 #25] study is the better study to develop a C-R function linking $PM_{2.5}$ with bronchitis.

Bronchitis was counted in the study only if there were "reports of symptoms in the past 12 months" [Dockery, 1996 #25, 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. For this analysis, we assumed that the C-R function based on Dockery et al. is measuring acute bronchitis. The C-R function is based on results of the single pollutant model reported in Table 1.

Single Pollutant Model

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 $\mu g/m^3$) versus the least polluted city ($PM_{2.1}$ = 5.8 $\mu g/m^3$) [Dockery, 1996 #25, Tables 1 and 4]. The original study used $PM_{2.1}$, however, we use 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, 2002 #2354, Table 11]
Population: population of ages 8-12

[46] 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.

Acute Myocardial Infarction (Heart Attacks), Nonfatal [Peters, 2001 #2157]

Peters et al. [2001 #2157] 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 PM_{10}, $PM_{10-2.5}$, $PM_{2.5}$,"black carbon", O_3, CO, NO_2, and SO_2 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 PM_{10} as well. None of the other particle measures or gaseous pollutants were significantly associated with acute myocardial infarction for the two hour or twenty-four hour period before onset.

The patient population for this study was selected from health centers across the United States. 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, we apply an age range of 18 and over in the C-R 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 #2374, Table 10].

Single Pollutant Model

The coefficient and standard error are calculated from an odds ratio of 1.62 (95% CI 1.13-2.34) for a 20 $\mu g/m^3$ increase in twenty-four hour average $PM_{2.5}$ [Peters, 2001 #2157, 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) [47]
Population: population of ages 18 and older

[47]This estimate assumes that all heart attacks that are not instantly fatal will result in a hospitalization. In addition, Rosamond et al. [1999 #2373] 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). We applied a factor of 0.93 to the number of hospitalizations to estimate the number of nonfatal heart attacks per year.

Any of 19 Respiratory Symptoms [Krupnick, 1990 #35]

Krupnick et al. [1990 #35] estimated the impact of air pollution on the incidence of any of 19 respiratory symptoms or conditions in 570 adults and 756 children living in three communities in Los Angeles, California from September 1978 to March 1979. Krupnick et al. [1990 #35] listed 13 specific "symptoms or conditions": head cold, chest cold, sinus trouble, croup, cough with phlegm, sore throat, asthma, hay fever, doctor-diagnosed ear infection, flu, pneumonia, bronchitis, and bronchiolitis. The other six symptoms or conditions are not specified.

In their analysis, they included COH, ozone, NO_2, and SO_2, and they used a logistic regression model that takes into account whether a respondent was well or not the previous day. A key difference between this and the usual logistic model, is that the model they used includes a lagged value of the dependent variable. In single-pollutant models, daily O_3, COH, and SO_2 were significantly related to respiratory symptoms in adults. Controlling for other pollutants, they found that ozone was still significant. The results were more variable for COH and SO_2, perhaps due to collinearity. NO_2 had no significant effect. No effect was seen in children for any pollutant. The results from the two-pollutant model with COH and ozone are used to develop a C-R function.

Multipollutant Model (PM_{10} and ozone)

The C-R function used to estimate the change in ARD2 associated with a change in daily average PM_{10} concentration is based on Krupnick et al. [1990 #35, p. 12]:[48]

$$\Delta ARD2 \cong \beta^{*}_{PM_{10}} \cdot \Delta PM_{10} \cdot pop\,,$$

Functional Form: Linear
Coefficient: first derivative of the stationary probability = 0.000461
Standard Error: 0.000239
Population: population of ages 18-64 years[49]

The logistic regression model used by Krupnick et al. [1990 #35] takes into account whether a respondent was well or not the previous day. Following Krupnick et al. (p. 12), the probability that one is sick is on a given day is:

[48]Krupnick and Kopp [1988 #318, p. 2-24] and ESEERCO [1994 #323, p. V-32] used the same C-R functional form as that used here.

[49]Krupnick et al. [1990 #35, Table 1] reported the age distribution in their complete data, but they did not report the ages of individuals that were considered "adult." This analysis assumes that individuals 18 and older were considered adult. Only a small percentage (0.6%) of the study population is above the age of 60, so the C-R function was limited to the adult population. up through the age of 65.

$$probability(ARD2) = \frac{p_0}{1 - p_1 + p_0}$$

$$probability(ARD2 | sickness\ or\ not_{t-1}) = p_i = \frac{1}{1 - e^{\beta_0 + \beta_1 \cdot ARD2_{t-1} + X \cdot \beta}}, for\ i = 0,1.$$

where:

X	=	the matrix of explanatory variables
p_0	=	the probability of sickness on day t, given wellness on day t-1, and
p_1	=	the probability of sickness on day t, given sickness on day t-1.

In other words, the transition probabilities are estimated using a logistic function; the key difference between this and the usual logistic model, is that the model includes a lagged value of the dependent variable.

To calculate the impact of COH (or other pollutants) on the probability of ARD2, it is possible, in principle, to estimate ARD2 before the change in COH and after the change:

$$\Delta ARD2 = ARD2_{after} - ARD2_{before}.$$

However the full suite of coefficient estimates are not available.[50] Rather than use the full suite of coefficient values, the impact of COH on the probability of probability of ARD2 may be approximated by the derivative of ARD2 with respect to COH:

$$\frac{\partial probability(ARD2)}{\partial COH} = \frac{p_0 \cdot (1 - p_1) \cdot \beta_{COH} \cdot [p_1 + (1 - p_0)]}{(1 - p_1 + p_0)^2} = \beta^*_{COH},$$

where β_{COH} is the reported logistic regression coefficient for COH. Since COH data are not available for the benefits analysis, an estimated PM_{10} logistic regression coefficient is used based on the following assumed relationship between PM_{10}, COH, and TSP:

$$COH = 0.116 \cdot TSP$$

$$PM_{10} = 0.55 \cdot TSP$$

[50]The model without NO_2 [Krupnick, 1990 #35, Table V equation 3] was used in this analysis, but the full suite of coefficient estimates for this model were not reported. Krupnick et al. [1990 #35, Table IV] reported all of the estimated coefficients for a model of children and for a model of adults when four pollutants were included (ozone, COH, SO_2, and NO_2). However, because of high collinearity between NO_2 and COH, NO_2 was dropped from some of the reported analyses (Krupnick et al., p. 10), and the resulting coefficient estimates changed substantially [see \Krupnick, 1990 #35, Table IV]. Both the ozone and COH coefficients dropped by about a factor of two or more.

$$\Rightarrow COH = 0.2109 \cdot PM_{10}$$

$$\Rightarrow \beta_{PM_{10}} = 0.2109 \cdot \beta_{COH} = 0.2109 \cdot 0.0088 = 0.001856.$$

This analysis uses $\beta_{COH} = 0.0088$ [Krupnick, 1990 #35, Table V equation 3]. The conversion from COH to TSP is based on study-specific information provided to ESEERCO [1994 #323, p. V-32]. The conversion of TSP to PM_{10} is from also from ESEERCO [1994 #323, p. V-5], which cited studies by EPA [1986 #236] and the California Air Resources Board [1982 #329].

The change in the incidence of ARD2 associated with a given change in COH is then estimated by:

$$\frac{\partial ARD2}{\partial PM_{10}} \cong \frac{\Delta ARD2}{\Delta PM_{10}}$$

$$\Rightarrow \frac{\Delta ARD2}{\Delta PM_{10}} \cong \beta^*_{PM_{10}}$$

$$\Rightarrow \Delta ARD2 \cong \beta^*_{PM_{10}} \cdot \Delta PM_{10}\ .$$

This analysis uses transition probabilities obtained from Krupnick et al. as reported by ESEERCO [1994 #323, p. V-32], for the adult population: $p_1 = 0.7775$ and $p_0 = 0.0468$. This implies:

$$\beta^*_{PM_{10}} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.001856 \cdot \left[0.7775_1 + (1 - 0.0468)\right]}{(1 - 0.7775 + 0.0468)^2} = 0.000461.$$

The *standard error* for the coefficient is derived using the reported standard error of the logistic regression coefficient in Krupnick et al. [1990 #35, Table V]:

$$\Rightarrow \beta_{PM_{10},\,high} = 0.2109 \cdot \beta_{COH,\,high} = 0.2109 \cdot \left(0.0088 + (1.96 \cdot 0.0046)\right) = 0.003757$$

$$\Rightarrow \beta^*_{PM_{10},\,high} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.003757 \cdot \left[0.7775 + (1 - 0.0468)\right]}{(1 - 0.7775 + 0.0468)^2} = 0.000934$$

$$\sigma_{\beta,\,high} = \frac{\beta_{PM_{10},\,high} - \beta_{PM_{10}}}{1.96} = \frac{(0.000934 - 0.000461)}{1.96} = 0.000236$$

$$\beta_{PM_{10},low} = 0.2109 \cdot \beta_{COH,low} = 0.2109 \cdot (0.0088 - (1.96 \cdot 0.0046)) = -4.555 \cdot 10^{-5}$$

$$\Rightarrow \beta^*_{PM_{10},low} = \frac{0.0468 \cdot (1 - 0.7775) \cdot (-4.555 \cdot 10^{-5}) \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = -1.132 \cdot 10^{-5}$$

$$\Rightarrow \sigma_{\beta,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{(0.000461 + 1.132 \cdot 10^{-5})}{1.96} = 0.000241$$

$$\sigma_{\beta} = \frac{\sigma_{\beta,high} + \sigma_{\beta,low}}{2} = 0.000239 .$$

Lower Respiratory Symptoms [Schwartz, 2000 #1657]

Schwartz et al. [2000 #1657] replicated a previous analysis [Schwartz, 1994 #96] linking PM levels to lower respiratory symptoms in children in six cities in the U.S. The original 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 previous study focused on PM_{10}, acid aerosols, and gaseous pollutants, although single-pollutant $PM_{2.5}$ results were reported. Schwartz et al. [2000 #1657] focused more on the associations between $PM_{2.5}$ and $PM_{10-2.5}$ and lower respiratory symptoms. In single and co-pollutant models, $PM_{2.5}$ was significantly associated with lower respiratory symptoms, while $PM_{10-2.5}$ was not. $PM_{10-2.5}$ exhibited a stronger association with cough than did $PM_{2.5}$. The $PM_{2.5}$ C-R function for lower respiratory symptoms is based on the results of the reported co-pollutant model ($PM_{2.5}$ and $PM_{10-2.5}$).

Multipollutant Model ($PM_{2.5}$ and $PM_{10-2.5}$)

In a model with $PM_{10-2.5}$, the $PM_{2.5}$ coefficient and standard error are calculated from the reported odds ratio (1.29) and 95% confidence interval (1.06-1.57) associated with a 15 $\mu g/m^3$ change in $PM_{2.5}$ [Schwartz , 2000 #1657, Table 2].

Functional Form: Logistic
Coefficient: 0.016976
Standard Error: 0.006680
Incidence Rate: daily lower respiratory symptom incidence rate per person = 0.0012 [Schwartz, 1994 #96, Table 2]
Population: population of ages 7 to 14

Minor Restricted Activity Days: Ostro and Rothschild [1989 #60]

Ostro and Rothschild [1989 #60] 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.[51] 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. The C-R function for PM is based on this co-pollutant model.

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. The elderly appear more likely to die due to PM exposure than other age groups [e.g., \Schwartz, 1994 #149, p. 30;] and a number of studies have found that hospital admissions for the elderly are related to PM exposures [e.g., \Schwartz, 1994 #147; Schwartz, 1994 #144].

Multipollutant Model ($PM_{2.5}$ and ozone)

Using the results of the two-pollutant model, we developed separate coefficients 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 #60, Table 4] using the inverse of the variance as the weight:

$$\beta = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = 0.00741 .$$

[51] The study population is based on the Health Interview Survey (HIS), conducted by the National Center for Health Statistics. In publications from this ongoing survey, non-elderly adult populations are generally reported as ages 18-64. From the study, it is not clear if the age range stops at 65 or includes 65 year olds. We apply the C-R function to individuals ages 18-64 for consistency with other studies estimating impacts to non-elderly adult populations.

The standard error of the coefficient is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$\sigma_\beta^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981}\frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981}\frac{1}{\sigma_{\beta_i}^2}}\right) = \left(\frac{\sum_{i=1976}^{1981}\frac{\beta_i}{\sigma_{\beta_i}^2}}{\gamma}\right) = \sum_{i=1976}^{1981}\text{var}\left(\frac{\beta_i}{\sigma_{\beta_i}^2\cdot\gamma}\right).$$

This reduces down to:

$$\sigma_\beta^2 = \frac{1}{\gamma} \Rightarrow \sigma_\beta = \sqrt{\frac{1}{\gamma}} = 0.00070.$$

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 #60, p. 243]
Population: adult population ages 18 to 64

Work Loss Days [Ostro, 1987 #456]

Ostro [1987 #456] 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.[52] The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average $PM_{2.5}$ levels[53] were significantly linked to work-loss days, RADs, and RRADs, however there was some year-to-year variability in the results. 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 #456, Table III] using the inverse of the variance as the weight.

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. The elderly appear more likely to die due

[52] The study population is based on the Health Interview Survey (HIS), conducted by the National Center for Health Statistics. In publications from this ongoing survey, non-elderly adult populations are generally reported as ages 18-64. From the study, it is not clear if the age range stops at 65 or includes 65 year olds. We apply the C-R function to individuals ages 18-64 for consistency with other studies estimating impacts to non-elderly adult populations.

[53] 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.

to PM exposure than other age groups [e.g., \Schwartz, 1994 #149, p. 30;] and a number of studies have found that hospital admissions for the elderly are related to PM exposures [e.g., \Schwartz, 1994 #147; Schwartz, 1994 #144]. On the other hand, the number of workers over the age of 65 is relatively small; it was approximately 3% of the total workforce in 2001 [U.S. Bureau of the Census, 2002 #2387, Table 561].

Single Pollutant Model

The coefficient used in the C-R function is a weighted average of the coefficients in Ostro [1987 #456, Table III] using the inverse of the variance as the weight:

$$\beta = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = 0.0046 .$$

The standard error of the coefficient is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$\sigma_\beta^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\gamma} \right) = \sum_{i=1976}^{1981} \text{var}\left(\frac{\beta_i}{\sigma_{\beta_i}^2 \cdot \gamma} \right).$$

This eventually reduces down to:

$$\sigma_\beta^2 = \frac{1}{\gamma} \Rightarrow \sigma_\beta = \sqrt{\frac{1}{\gamma}} = 0.00036 .$$

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 [Adams, 1999 #2355, Table 41; U.S. Bureau of the Census, 1997 #447, No. 22]
Population: adult population ages 18 to 64

Exhibit D-7 Concentration-Response (C-R) Functions for Particulate Matter and Asthma-Related Effects

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Beta	Std Error	Functional Form	Notes
Acute Bronchitis	$PM_{2.5}$	McConnell et al.	1999	Southern California	9-15	All	All	None	Annual Avg	0.022431	0.015957	Logistic	
Asthma Exacerbation, Asthma Attacks	PM_{10}	Whittemore and Korn	1980	Los Angeles, CA	All	All	All	O_3	24-hr avg	0.001436	0.000558	Logistic	
Asthma Exacerbation, Cough	$PM_{2.5}$	Ostro et al.	2001	Los Angeles, CA	8-13	Black	All	None	24-hr avg	0.003177	0.001156	Logistic	New onset of symptoms
Asthma Exacerbation, Cough	PM_{10}	Vedal et al.	1998	Vancouver, CAN	6-13	All	All	None	24-hr avg	0.007696	0.003786	Logistic	
Asthma Exacerbation, Moderate or Worse	$PM_{2.5}$	Ostro et al.	1991	Denver, CO	All	All	All	None	24-hr avg	0.0006	0.0003	Linear (log of pollutant)	
Asthma Exacerbation, One or More Symptoms	PM_{10}	Yu et al.	2000	Seattle, WA	5-13	All	All	CO, SO_2	24-hr avg	0.004879	0.005095	Logistic	
Asthma Exacerbation, Shortness of Breath	$PM_{2.5}$	Ostro et al.	2001	Los Angeles, CA	8-13	Black	All	None	24-hr avg	0.003177	0.001550	Logistic	New onset of symptoms
Asthma Exacerbation, Wheeze	$PM_{2.5}$	Ostro et al.	2001	Los Angeles, CA	8-13	Black	All	None	24-hr avg	0.002565	0.001030	Logistic	New onset of symptoms
Chronic Phlegm	$PM_{2.5}$	McConnell et al.	1999	Southern California	9-15	All	All	None	Annual Avg	0.063701	0.025580	Logistic	
Upper Respiratory Symptoms	PM_{10}	Pope et al.	1991	Utah Valley	9-11	All	All	None	24-hr avg	0.0036	0.0015	Logistic	

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Asthma-Related Effects

Acute Bronchitis [McConnell, 1999 #1900]

McConnell et al. [1999 #1900] examined the relationship between air pollution and bronchitic symptoms among asthmatic 4th, 7th, and 10th grade children in southern California.[54] The authors collected information on the prevalence of bronchitis, chronic cough, and chronic phlegm among children with and without a history of asthma and/or wheeze. They used annual measurements of ozone, PM_{10}, $PM_{2.5}$, NO_2, and acids in a logistic regression model with adjustments for personal covariates. Neither bronchitis, cough, or phlegm were associated with any of the pollutants among children with no history of wheeze or asthma or a history of wheeze without diagnosed asthma. Among asthmatics, PM_{10} was significantly associated with bronchitis and phlegm; $PM_{2.5}$ was significantly associated with phlegm and marginally associated with bronchitis; NO_2 and acids were both significantly associated with phlegm; and ozone was not significantly associated with any of the endpoints.

Bronchitis was defined in the study by the question: "How many times in the past 12 months did your child have bronchitis?" [McConnell, 1999 #1900, p. 757]. It is unclear, however, if the cases of bronchitis are acute and temporary, or if the bronchitis is a chronic condition. McConnell et al. found a relationship between PM and chronic phlegm but none with chronic cough, each of which may be indicators of chronic bronchitis. For this analysis, we assumed that the C-R function based on McConnell et al. is measuring acute bronchitis. The $PM_{2.5}$ C-R function for bronchitis among asthmatics is based on the results of the single pollutant model reported in Table 3.

Single Pollutant Model

The estimated logistic coefficient and standard error are based on the odds ratio (1.4) and 95% confidence interval (0.9-2.3) associated with an increase in yearly mean 2-week average $PM_{2.5}$ of 15 $\mu g/m^3$. [McConnell, 1999 #1900, Table 3]

Functional Form: Logistic
Coefficient: 0.022431
Standard Error: 0.015957
Incidence Rate: annual incidence rate of one or more episodes of bronchitis per asthmatic = 0.326 [McConnell, 1999 #1900, Table 2]
Population: population of asthmatics ages 9 to 15 = 5.67%[55] of population ages 9 to 15

[54] Assuming that a child enters kindergarten at age 5, 4th grade corresponds to age 9 and 10th grade corresponds to age 15. We therefore applied the results of this study to children ages 9 to 15.

[55] The American Lung Association [2002 #2358, Table 7] estimates asthma prevalence for children ages 5 to 17 at 5.67% (based on data from the 1999 National Health Interview Survey).

Asthma Attacks [Whittemore and Korn, 1980 #634]

Whittemore and Korn [1980 #634] examined the relationship between air pollution and asthma attacks in a survey of 443 children and adults, living in six communities in southern California during three 34-week periods in 1972-1975. The analysis focused on TSP and oxidants (O_x). Respirable PM, NO_2, SO_2 were highly correlated with TSP and excluded from the analysis. In a two pollutant model, daily levels of both TSP and oxidants were significantly related to reported asthma attacks. The results from this model were used, and the oxidant result was adjusted so it may be used with ozone data.

Multipollutant Model (PM_{10} and ozone)

The PM_{10} C-R function is based on the results of a co-pollutant model of TSP and ozone [Whittemore, 1980 #634, Table 5]. Assuming that PM_{10} is 55 percent of TSP[56] and that particulates greater than ten micrometers are harmless, the coefficient is calculated by dividing the TSP coefficient (0.00079) by 0.55. The standard error is calculated from the two-tailed p-value (<0.01) reported by Whittemore and Korn [1980 #634, Table 5], which implies a t-value of at least 2.576 (assuming a large number of degrees of freedom).

Functional Form: Logistic
Coefficient: 0.001436
Standard Error: 0.000558
Incidence Rate: daily incidence of asthma attacks = 0.0550[57]
Population: population of asthmatics of all ages = 3.86% of the population of all ages [American Lung Association, 2002 #2358, Table 7]

Asthma Exacerbation, Cough [Ostro, 2001 #2317]

Ostro et al. [2001 #2317] 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 PM_{10}, $PM_{2.5}$, NO_2, and O_3 in a logistic regression model with control for age, income, time trends, and temperature-related weather effects.[58] 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

[56]The conversion of TSP to PM_{10} is from ESEERCO [1994 #323, p. V-5], who cited studies by EPA [1986 #236] and the California Air Resources Board [1982 #329].

[57] Based on an analysis of the 1999 National Health Interview Survey, the daily incidence of wheezing attacks for adult asthmatics is estimated to be 0.0550. In the same survey, wheezing attacks for children were examined, however, the number of wheezing attacks per year were censored at 12 (compared to censoring at 95 for adults). Due to the potential for underestimation of the number of children's wheezing attacks, we used the adult rate for all individuals.

[58] The authors note that there were 26 days in which $PM_{2.5}$ concentrations were reported higher than PM_{10} 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.

followed by a symptom-free day. The authors found cough prevalence associated with PM_{10} and $PM_{2.5}$ and cough incidence associated with $PM_{2.5}$ PM_{10}, and NO_2. Ozone was not significantly associated with cough among asthmatics. The $PM_{2.5}$ C-R function is based on the results of the single pollutant model looking at the onset of new symptoms.

Single Pollutant Model

The coefficient and standard error are based on an odds ratio of 1.10 (95% CI 1.03-1.18) for a 30 $\mu g/m^3$ increase in 12-hour average $PM_{2.5}$ concentration.

The C-R function based on this model will estimate the number of new onset episodes of cough avoided. In order to convert this estimate to the total number of episodes avoided, the results are adjusted by an estimate of the duration of symptom episodes. The average duration can be estimated from Ostro et al. [2001 #2317] using the ratio of the probability of a symptom episode to the probability of a new onset episode. For cough, this ratio is 2.2 (14.5% divided by 6.7%) [Ostro, 2001 #2317, p.202].

In addition, not all children are at-risk for a new onset of cough, as defined by the study. On average, 14.5% of African-American asthmatics have cough on a given day [Ostro, 2001 #2317, p.202]. Only those who are symptom-free on the previous day are at-risk for a new onset episode (1-0.145 = 85.5%). As a result, a factor of 85.5% is used in the function to estimate the population of African-American 8 to 13 year old children at-risk for a new cough episode.

Functional Form: Logistic
Coefficient: 0.003177
Standard Error: 0.001156
Incidence Rate: daily new onset cough (incidence) rate per person [Ostro, 2001 #2317, p.202] = 0.067
Population: asthmatic African-American population ages 8 to 13 at-risk for a new episode of cough = 6.21% of African-American population ages 8 to 13 multiplied (85.5% at-risk[59] times 7.26% asthmatic[60])
Adjustment Factor: average number of consecutive days with a cough episode (days) = 2.2

Asthma Exacerbation, Cough [Vedal, 1998 #416]

Vedal et al. [1998 #416] 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-

[59] On average, 17.3% of African-American asthmatics have cough episodes on a given day [Ostro, 2001 #2317, p.202]. Only those who are symptom-free on the previous day (1-0.145 = 85.5%) are at-risk for a new onset episode.

[60] The American Lung Association [2002 #2358, Table 9] estimates asthma prevalence for African-American children ages 5 to 17 at 7.26% (based on data from the 1999 National Health Interview Survey).

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 PM_{10} levels and daily increases in various respiratory symptoms among these groups. In the entire sample of children, PM_{10} was significantly associated with cough, phlegm, nose symptoms, and throat soreness. Among children with diagnosed asthma, the authors report a significant association between PM_{10} 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 strategy, the results from the full sample of children are not generalizeable to the entire population. The C-R function presented below is based on results among asthmatics only.

Single Pollutant Model

The PM_{10} coefficient and standard error are based on an increase in odds of 8% (95% CI 0-16%) reported in the abstract for a 10 $\mu g/m^3$ increase in daily average PM_{10}.

Functional Form: Logistic
Coefficient: 0.007696
Standard Error: 0.003786
Incidence Rate: daily cough rate per person [Vedal, 1998 #416, Table 1, p. 1038] = 0.086
Population: asthmatic population ages 6 to 13 = 5.67%[61] of population ages 6 to 13

Asthma Exacerbation, Moderate or Worse [Ostro, 1991 #64]

Ostro et al. [1991 #64] examined the effect of air pollution on asthmatics, ages 18 to 70, living in Denver, Colorado from December 1987 to February 1988. The respondents in this study were asked to record daily a subjective rating of their overall asthma status each day (0=none, 1=mild, 2=moderate, 3=severe, 4=incapacitating). Ostro et al. then examined the relationship between moderate (or worse) asthma and H^+, sulfate, SO_2, $PM_{2.5}$, estimated $PM_{2.5}$, PM_{10}, nitrate, and nitric acid. Daily levels of H^+ were linked to cough, asthma, and shortness of breath. $PM_{2.5}$ was linked to asthma. Sulfate was linked to shortness of breath. No effects seen for other pollutants. The C-R function is based on a single-pollutant linear regression model where the log of the pollutant is used.

Single Pollutant Model

Two $PM_{2.5}$ coefficients are presented, both equal 0.0006, however only one is significant. The coefficient based on data that does not include estimates of missing $PM_{2.5}$ values is not

[61] The American Lung Association [2002 #2358, Table 7] estimates asthma prevalence for children 5-17 at 5.67% (based on data from the 1999 National Health Interview Survey).

significant (std error = 0.0053); the coefficient that includes estimates of missing $PM_{2.5}$ values (estimated using a function of sulfate and nitrate) is significant at $p < 0.5$ (std error = 0.0003). The latter coefficient is used here.

The C-R function to estimate the change in the number of days with moderate (or worse)

$$\Delta Days\ Moderate / Worse\ Asthma = -\beta \cdot \ln\left(\frac{PM_{2.5,\,after}}{PM_{2.5,\,before}}\right) \cdot pop\,,$$

asthma

Functional Form: Linear (using log of the pollutant)
Coefficient: 0.0006
Standard Error: 0.0003
Population: population of asthmatics of all ages[62] = 3.86% of the population of all ages [American Lung Association, 2002 #2358, Table 7]

Asthma Exacerbation, One or More Symptoms [Yu, 2000 #2112]

Yu et al. [2000 #2112] examined the association between air pollution and asthmatic symptoms among mild to moderate asthmatic children ages 5-13 in Seattle. They collected air quality data for CO, SO_2, PM_{10}, and $PM_{1.0}$ and asked study subjects to record symptoms daily. They used logistic regression models with generalized estimating equations in two different approaches. A "marginal approach" was used to estimate the impact of air pollution on asthma symptoms and a "transition approach" was used to estimate the association conditioned on the previous day's outcome. The primary endpoint, odds of at least one asthma symptom, was significantly associated with CO, PM_{10}, and $PM_{1.0}$ in single pollutant models. In multipollutant models, CO remained significant while PM effects declined slightly. The magnitude of the effects were similar between the "marginal" and "transition" approaches. The C-R function is based on the results of the "transition approach," where the previous day's symptoms is an explanatory variable.

Multipollutant Model (PM_{10}, CO, SO_2)

The C-R function is based on the results of the "transition approach," where the previous day's symptoms is an explanatory variable. The multipollutant PM_{10} coefficient and standard error are based on the odds ratio (1.05) and 95% confidence interval (0.95-1.16) for a 10 $\mu g/m^3$ increase in one-day lagged daily average PM_{10} [Yu, 2000 #2112, Table 4, p. 1212].

[62] The C-R function is applied to asthmatics of all ages, although the study population consists of asthmatics between the ages of 18 and 70. It seems reasonable to assume that individuals over the age of 70 are at least as susceptible as individuals in the study population. It also seems reasonable to assume that individuals under the age of 18 are also susceptible. For example, controlling for oxidant levels, Whittemore and Korn [1980 #634] found TSP significantly related to asthma attacks in a study population comprised primarily (59 percent) of individuals less than 16 years of age.

Functional Form: Logistic
Coefficient: 0.004879
Standard Error: 0.005095
Incidence Rate: daily rate of at least one asthma episode per person [Yu, 2000 #2112, Table 2, p. 1212] = 0.60
Population: asthmatic population ages 5 to 13 = 5.67%[63] of population ages 5 to 13

Asthma Exacerbation, Shortness of Breath [Ostro, 2001 #2317]

Ostro et al. [2001 #2317] studied the relationship 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 PM_{10}, $PM_{2.5}$, NO_2, and ozone in a logistic regression model with control for age, income, time trends, and temperature-related weather effects. Asthma symptom endpoints were defined in two ways: "probability of a day with symptoms" and "new onset of a symptom episode". New onset of a symptom episode was defined as a day with symptoms followed by a symptom-free day. The authors found that both the prevalent and incident episodes of shortness of breath were associated with $PM_{2.5}$ and PM_{10}. Neither ozone nor NO_2 were significantly associated with shortness of breath among asthmatics. The $PM_{2.5}$ C-R function is based on the results of a single pollutant model looking at the onset of new symptoms.

Single Pollutant Model

The coefficient and standard error are based on an odds ratio of 1.10 (95% CI 1.00-1.20) for a 30 $\mu g/m^3$ increase in 12-hour average $PM_{2.5}$ concentration [Ostro, 2001 #2317, Table 5, p.204].

The C-R function based on this model will estimate the number of new onset episodes of shortness of breath avoided. In order to convert this estimate to the total number of episodes avoided, the results are adjusted by an estimate of the duration of symptom episodes. The average duration can be estimated from Ostro et al. [2001 #2317] using the ratio of the probability of a symptom episode to the probability of a new onset episode. For shortness of breath, this ratio is 2.0 (7.4% divided by 3.7%) [Ostro, 2001 #2317, p.202].

In addition, not all children are at-risk for a new onset of shortness of breath, as defined by the study. On average, 7.4% of African-American asthmatics have shortness of breath episodes on a given day [Ostro, 2001 #2317, p.202]. Only those who are symptom-free on the previous day are at-risk for a new onset episode (1-0.074 = 92.6%). As a result, a factor of 92.6% is used in the function to estimate the population of African-American 8 to 13 year old children at-risk for a new shortness of breath episode.

[63] The American Lung Association [2002 #2358, Table 7] estimates asthma prevalence for children 5 to 17 at 5.67% (based on data from the 1999 National Health Interview Survey).

Functional Form: Logistic
Coefficient: 0.003177
Standard Error: 0.001550
Incidence Rate: daily new onset shortness of breath (incidence) rate per person [Ostro, 2001 #2317, p.202] = 0.037
Population: asthmatic African-American population ages 8 to 13 at-risk for a new episode of shortness of breath = 6.72% of African-American population ages 8 to 13 multiplied (92.6% at-risk[64] times 7.26% asthmatic[65])
Adjustment Factor: average number of consecutive days with a shortness of breath episode (days) = 2.0

Asthma Exacerbation, Wheeze [Ostro, 2001 #2317]

Ostro et al. [2001 #2317] 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 PM_{10}, $PM_{2.5}$, NO_2, and O_3 in a logistic regression model with control for age, income, time trends, and temperature-related weather effects. 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 both the prevalence and incidence of wheeze associated with $PM_{2.5}$ PM_{10}, and NO_2. Ozone was not significantly associated with wheeze among asthmatics. The $PM_{2.5}$ C-R function is based on the results of a single pollutant model looking at the onset of new symptoms.

Single Pollutant Model

The coefficient and standard error are based on an odds ratio of 1.08 (95% CI 1.01-1.14) for a 30 $\mu g/m^3$ increase in 12-hour average $PM_{2.5}$ concentration [Ostro, 2001 #2317, Table 5, p.204].

The C-R function based on this model will estimate the number of new onset episodes of wheeze avoided. In order to convert this estimate to the total number of episodes avoided, the results are adjusted by an estimate of the duration of symptom episodes. The average duration can be estimated from Ostro et al. [2001 #2317] using the ratio of the probability of a symptom episode to the probability of a new onset episode. For wheeze, this ratio is 2.3 (17.3% divided by 7.6%) [Ostro, 2001 #2317, p.202].

[64] On average, 7.4% of African-American asthmatics have shortness of breath episodes on a given day [Ostro, 2001 #2317, p.202]. Only those who are symptom-free on the previous day (1-0.074 = 92.6%) are at-risk for a new onset episode.

[65] The American Lung Association [2002 #2358, Table 9] estimates asthma prevalence for African-American children ages 5 to 17 at 7.26% (based on data from the 1999 National Health Interview Survey).

In addition, not all children are at-risk for a new onset of wheeze, as defined by the study. On average, 17.3% of African-American asthmatics have wheeze on a given day [Ostro, 2001 #2317, p.202]. Only those who are symptom-free on the previous day are at-risk for a new onset episode (1-0.173 = 82.7%). As a result, a factor of 82.7% is used in the function to estimate the population of African-American 8 to 13 year old children at-risk for a new wheeze episode.

Functional Form: Logistic
Coefficient: 0.002565
Standard Error: 0.001030
Incidence Rate: daily new onset wheeze (incidence) rate per person [Ostro, 2001 #2317, p.202] = 0.076
Population: asthmatic African-American population ages 8 to 13 at-risk for a new episode of wheeze = 6.00% of African-American population ages 8 to 13 multiplied (82.7% at-risk[66] times 7.26% asthmatic[67])
Adjustment Factor: average number of consecutive days with a wheeze episode (days) = 2.3

Chronic Phlegm [McConnell, 1999 #1900]

McConnell et al. [1999 #1900] examined the relationship between air pollution and bronchitic symptoms among asthmatic 4th, 7th, and 10th grade children in southern California.[68] The authors collected information on the prevalence of bronchitis, chronic cough, and chronic phlegm among children with and without a history of asthma and/or wheeze. They used annual measurements of ozone, PM_{10}, $PM_{2.5}$, NO_2, and acids in a logistic regression model with adjustments for personal covariates. Neither bronchitis, cough, or phlegm were associated with any of the pollutants among children with no history of wheeze or asthma or a history of wheeze without diagnosed asthma. Among asthmatics, PM_{10} was significantly associated with bronchitis and phlegm; $PM_{2.5}$ was significantly associated with phlegm and marginally associated with bronchitis; NO_2 and acids were both significantly associated with phlegm; and ozone was not significantly associated with any of the endpoints.

Phlegm was defined in the study by the question: "Other than with colds, does this child usually seem congested in the chest or bring up phlegm?" [McConnell, 1999 #1900, p. 757]. The authors refer to this definition as "chronic phlegm" and we also assume that the term "usually" refers to chronic, rather than acute, phlegm. The PM C-R functions for chronic phlegm among asthmatics are based on the results of the single pollutant model reported in Table 3.

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[66] On average, 17.3% of African-American asthmatics have wheeze episodes on a given day [Ostro, 2001 #2317, p.202]. Only those who are symptom-free on the previous day (1-0.173 = 82.7%) are at-risk for a new onset episode.

[67] The American Lung Association [2002 #2358, Table 9] estimates asthma prevalence for African-American children ages 5 to 17 at 7.26% (based on data from the 1999 National Health Interview Survey).

[68] Assuming that a child enters kindergarten at age 5, 4th grade corresponds to age 9 and 10th grade corresponds to age 15. We therefore applied the results of this study to children ages 9 to 15.

Single Pollutant Model

The estimated logistic coefficient and standard error are based on the odds ratio (2.6) and 95% confidence interval (1.2-5.4) associated with an increase in yearly mean 2-week average $PM_{2.5}$ of 15 $\mu g/m^3$. [McConnell, 1999 #1900, Table 3]

Functional Form: Logistic
Coefficient: 0.063701
Standard Error: 0.025580
Incidence Rate: annual incidence rate of phlegm per asthmatic = 0.257 [McConnell, 1999 #1900, Table 2]
Population: population of asthmatics ages 9 to 15 = 5.67%[69] of population ages 9 to 15

Upper Respiratory Symptoms [Pope, 1991 #77]

Using logistic regression, Pope et al. [1991 #77] estimated the impact of PM_{10} 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 PM_{10} 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, NO_2, and SO_2 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, 1991 #77, 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, 1991 #77, Table 5] show PM_{10} significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM_{10} effect. The results from the school-based sample are used here.

Single Pollutant Model

The coefficient and standard error for a one $\mu g/m^3$ change in PM_{10} is reported in Table 5.

Functional Form: Logistic

[69] The American Lung Association [2002 #2358, Table 7] estimates asthma prevalence for children ages 5 to 17 at 5.67% (based on data from the 1999 National Health Interview Survey).

Coefficient: 0.0036
Standard Error: 0.0015
Incidence Rate: daily upper respiratory symptom incidence rate per person = 0.3419 [Pope, 1991 #77, Table 2]
Population: asthmatic population ages 9 to 11 = 5.67%[70] of population ages 9 to 11

Ozone Concentration-response Functions

Short-term Mortality

Exhibit D-8 summarizes the C-R functions used to estimate the relationship between ozone and short-term mortality. Detailed summaries of each of the studies used to generate the functions are described below, along with the parameters used in each of the functions.

[70] The American Lung Association [2002 #2358, Table 7] estimates asthma prevalence for children ages 5 to 17 at 5.67% (based on data from the 1999 National Health Interview Survey).

Exhibit D-8 Concentration-Response (C-R) Functions for Ozone and Short-Term Mortality

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
Non-Accidental	Ito and Thurston	1996	Chicago, IL	All	All	All	PM_{10}	1-hr max	Log-linear	0.000634	0.000251
Non-Accidental	Kinney et al.	1995	Los Angeles, CA	All	All	All	PM_{10}	1-hr max	Log-linear	0	0.000214
Non-Accidental	Moolgavkar et al.	1995	Philadelphia, PA	All	All	All	SO_2, TSP	24-hr avg	Log-linear	0.000611	0.000216
Non-Accidental	Samet et al.	1997	Philadelphia, PA	All	All	All	CO, NO_2, SO_2, TSP	24-hr avg	Log-linear	0.000936	0.000312

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Short-Term Mortality, Non-Accidental [Ito, 1996 #187, Chicago]

Ito and Thurston [1996 #187] examined the relationship between daily non-accidental mortality and air pollution levels in Cook County, Illinois from 1985 to 1990. They examined daily levels of ozone, PM_{10}, SO_2, and CO, and found a significant relationship for ozone and PM_{10} with both pollutants in the model; no significant effects were found for SO_2 and CO. In single pollutant models the effects were slightly larger. The C-R function for ozone is based on the results of the co-pollutant model.

Multipollutant Model (ozone and PM_{10})

In a co-pollutant model with PM_{10}, the coefficient (0.000634) and standard error (0.000251) were obtained directly from the author because the published paper reported incorrect information.

Functional Form: Log-linear
Coefficient: 0.000634
Standard Error: 0.000251
Incidence Rate: county-level daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

Short-Term Mortality, Non-Accidental [Kinney, 1995 #191, Los Angeles]

Kinney et al. [1995 #191] examined the relationship between daily non-accidental mortality and air pollution levels in Los Angeles, California from 1985 to 1990. They examined ozone, PM_{10}, and CO, and found a significant relationship for each pollutant in single pollutant models. The effect for ozone dropped to zero with the inclusion of PM_{10} in the model, while the effect for CO and PM_{10} appeared co-pollutant ozone models. The C-R function for ozone is based on the results of the co-pollutant model.

Multipollutant Model (ozone and PM_{10})

In a model with PM_{10}, the coefficient and standard error are based on the relative risk (1.00) and 95% confidence interval (0.94-1.06) reported for a 143 ppb increase in daily one-hour maximum ozone concentration [Kinney, 1995 #191, Table 2, p. 64].

Functional Form: Log-linear
Coefficient: 0
Standard Error: 0.000214
Incidence: county-level daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

Short-Term Mortality, Non-Accidental [Moolgavkar, 1995 #49, Philadelphia]

Moolgavkar et al. [1995 #49] examined the relationship between daily non-accidental mortality and air pollution levels in Philadelphia, Pennsylvania from 1973 to 1988. They examined ozone, TSP, and SO_2 in a three-pollutant model, and found a significant relationship for ozone and SO_2; TSP was not significant. In season-specific models, ozone was significantly associated with mortality only in the summer months. The C-R function for ozone is based on the full-year three-pollutant model reported in Table 5 [Moolgavkar et al., 1995 #49, p. 482].

Multipollutant Model (ozone, SO_2, TSP)

The coefficient and standard error are based on the relative risk (1.063) and 95% confidence interval (1.018-1.108) associated with a 100 ppb increase in daily average ozone [Moolgavkar et al., 1995 #49, p. 482, Table 5].

Functional Form: Log-linear
Coefficient: 0.000611
Standard Error: 0.000216
Incidence Rate: county-level daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

Short-Term Mortality, Non-Accidental [Samet, 1997 #685, Philadelphia]

Samet et al. [1997 #685] examined the relationship between daily non-accidental mortality and air pollution levels in Philadelphia, Pennsylvania from 1974 to 1988. They examined ozone, TSP, SO_2, NO_2, and CO in a Poisson regression model. In single pollutant models, ozone, SO_2, TSP, and CO were significantly associated with mortality. In a five-pollutant model, they found a positive statistically significant relationship for each pollutant except NO_2. The C-R function for ozone is based on the five-pollutant model (ozone, CO, NO_2, SO_2, and TSP) reported in Table 9 [Samet, 1997 #685, p. 20].

Multipollutant Model (ozone, CO, NO_2, SO_2, and TSP)

In a model with CO, NO_2, SO_2, and TSP, the ozone coefficient and standard error are based on the percent increase (1.91) and t-statistic (3) associated with a 20.219 ppb increase in two-day average ozone [Samet, 1997 #685, p. 20, Table 9].

Functional Form: Log-linear
Coefficient: 0.000936
Standard Error: 0.000312
Incidence Rate: county-level daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

Exhibit D-9 Concentration-Response (C-R) Functions for Ozone and Chronic Illness

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
Chronic Asthma	McDonnell et al.	1999	SF, SD, South Coast Air Basin	27+	All	Male	None	annual avg 8-hr avg	Logistic	0.0277	0.0135

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Chronic Illness

Exhibit D-9 summarizes the C-R function ([McDonnell, 1999 #1153]) used to estimate the relationship between ozone and chronic asthma. A more detailed summary of McDonnell et al. [1999 #1153], and the parameters used in the function, is described below.

Chronic Asthma [McDonnell, 1999 #1153]

McDonnell et al. [1999 #1153] used the same cohort of Seventh-Day Adventists as Abbey et al. [1991 #242;, 1993 #245], and examined the association between air pollution and the onset of asthma in adults between 1977 and 1992. Males who did not report doctor-diagnosed asthma in 1977, but reported it in 1987 or 1992, had significantly higher ozone exposures, controlling for other covariates; no significant effect was found between ozone exposure and asthma in females. No significant effect was reported for females or males due to exposure to PM, NO_2, SO_2, or SO_4. The C-R function for ozone is based on the single pollutant model for males reported in Table 5 [McDonnell, 1999 #1153, 1999, p. 117].

Single Pollutant Model

The coefficient and standard error for males is reported in Table 5 for a unit increase in annual average eight-hour ozone concentrations.[71]

Functional Form: Logistic
Coefficient: 0.0277
Standard Error: 0.0135
Incidence Rate: annual asthma incidence rate per person = 0.00219 [McDonnell, 1999 #1153, 1999, Table 4]
Population: non-asthmatic males age 27 and over = 97.9%[72] of males 27+

[71] The eight-hour ozone concentration is defined as 9:00 A.M. to 4:59 P.M. The study used the 1973-1992 mean 8-hour average ambient ozone concentration [McDonnell, 1999 #1153, p. 113].

[72] The prevalence of asthma among males 27 and older (2.10 percent) was estimated from the 2000 National Health Interview Survey (NHIS) public use data, available at ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHIS/2000.

Exhibit D-10 Concentration-Response (C-R) Functions for Ozone and Hospital Admissions

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
All Respiratory	Burnett et al.	2001	Toronto, CAN	<2	All	All	$PM_{2.5}$	1-hr max	Log-linear	0.006309	0.001834
All Respiratory	Schwartz	1995	New Haven, CT	65+	All	All	PM_{10}	24-hr avg	Log-linear	0.002652	0.001398
All Respiratory	Schwartz	1995	Tacoma, WA	65+	All	All	PM_{10}	24-hr avg	Log-linear	0.007147	0.002565
Chronic Lung Disease	Moolgavkar et al.	1997	Minneapolis, MN	65+	All	All	CO, PM_{10}	24-hr avg	Log-linear	0.002743	0.001699
Chronic Lung Disease (less Asthma)	Schwartz	1994	Detroit, MI	65+	All	All	PM_{10}	24-hr avg	Log-linear	0.00549	0.00205
Pneumonia	Moolgavkar et al.	1997	Minneapolis, MN	65+	All	All	NO_2, PM_{10}, SO_2	24-hr avg	Log-linear	0.003696	0.001030
Pneumonia	Schwartz	1994	Detroit, MI	65+	All	All	PM_{10}	24-hr avg	Log-linear	0.00521	0.0013
Pneumonia	Schwartz	1994	Minneapolis, MN	65+	All	All	PM_{10}	24-hr avg	Log-linear	0.003977	0.001865

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Hospital Admissions

Exhibit D-10 summarizes the C-R functions used to estimate the relationship between ozone and hospital admissions. Detailed summaries of each of the studies used to generate the functions are described below, along with the parameters used in each of the functions.

Hospital Admissions for All Respiratory [Burnett, 2001 #2202, Toronto]

Burnett et al. [2001 #2202] studied the association between air pollution and acute respiratory hospital admissions (ICD codes 493, 466, 464.4, 480-486) in Toronto from 1980-1994, among children less than 2 years of age. They collected hourly concentrations of the gaseous pollutants, CO, NO_2, SO_2, and ozone. Daily measures of particulate matter were estimated for the May to August period of 1992-1994 using TSP, sulfates, and coefficient of haze data. The authors report a positive association between ozone in the May through August months and respiratory hospital admissions, for several single days after elevated ozone levels.

The strongest association was found using a five-day moving average of ozone. No association was found in the September through April months. In co-pollutant models with a particulate matter or another gaseous pollutant, the ozone effect was only slightly diminished. The effects for PM and gaseous pollutants were generally significant in single pollutant models but diminished in co-pollutant models with ozone, with the exception of CO. The C-R function for ozone is based on a co-pollutant model with $PM_{2.5}$, using the five-day moving average of one-hour max ozone.

Multipollutant Model (ozone and $PM_{2.5}$)

In a model with $PM_{2.5}$, the coefficient and standard error are based on the percent increase (33.0) and t-statistic (3.44) associated with a 45.2 ppb increase in the five-day moving average of one-hour max ozone [Burnett, 2001 #2202, Table 3].

Functional Form: Log-linear
Coefficient: 0.006309
Standard Error: 0.001834
Incidence Rate: region-specific daily hospital admission rate for all respiratory admissions per person less than 2 years of age (ICD codes 464, 466, 480-487, 493)
Population: population less than 2 years of age

Hospital Admissions for All Respiratory [Schwartz, 1995 #153, New Haven]

Schwartz [1995 #153] examined the relationship between air pollution and respiratory hospital admissions (ICD codes 460-519) for individuals 65 and older in New Haven, Connecticut, from January 1988 to December 1990. In single-pollutant models, PM_{10} and SO_2 were significant, while ozone was marginally significant. In a co-pollutant model with ozone and PM_{10}, both pollutants were significant. PM_{10} remained significant in a model with SO_2,

while ozone was marginally significant when adjusted for SO_2. SO_2 was significant in a co-pollutant model with PM_{10} but not with ozone. The ozone C-R function is based on results from the co-pollutant model with PM_{10}.

Multipollutant Model (ozone and PM_{10})

In a model with PM_{10}, the coefficient and standard error are estimated from the relative risk (1.07) and 95% confidence interval (1.00-1.15) for a 50 μg/m^3 increase in average daily ozone levels [Schwartz,1995 #153, Table 3, p. 534].[73]

Functional Form: Log-linear
Coefficient: 0.002652
Standard Error: 0.001398
Incidence Rate: region-specific daily hospital admission rate for respiratory admissions per person 65+ (ICD codes 460-519)
Population: population of ages 65 and older

Hospital Admissions for All Respiratory [Schwartz, 1995 #153, Tacoma]

Schwartz [1995 #153] examined the relationship between air pollution and hospital admissions for individuals 65 and older in Tacoma, Washington, from January 1988 to December 1990. In single-pollutant models, PM_{10}, ozone, and SO_2 were all significant. Ozone remained significant in separate co-pollutant models with PM_{10} and SO_2. PM_{10} remained significant in a co-pollutant model with SO_2, but not in a co-pollutant model with ozone. SO_2 was not significant in either of the co-pollutant models. The ozone C-R function is based on results from the co-pollutant model with PM_{10}.

Multipollutant Model (ozone and PM_{10})

In a model with PM_{10}, the coefficient and standard error are estimated from the relative risk (1.20) and 95% confidence interval (1.06-1.37) for a 50 μg/m^3 increase in average daily ozone levels [Schwartz, 1995 #153, Table 6, p. 535].[74]

Functional Form: Log-linear
Coefficient: 0.007147

[73] To calculate the coefficient, a conversion of 1.96 μg/m^3 per ppb is used, based on a density of ozone of 1.96 grams per liter (at 25 degrees Celsius). Since there are 1000 liters in a cubic meter and a million μg in a gram, this density means that there are 1.96 billion μg of ozone in a cubic meter of ozone. If a cubic meter has just one ppb of ozone, then this means that this particular cubic meter has 1.96 μg of ozone (i.e., one ppb = 1.96 μg/m^3).

[74]To calculate the coefficient, a conversion of 1.96 μg/m^3 per ppb is used, based on a density of ozone of 1.96 grams per liter (at 25 degrees Celsius). Since there are 1000 liters in a cubic meter and a million μg in a gram, this density means that there are 1.96 billion μg of ozone in a cubic meter of ozone. If a cubic meter has just one ppb of ozone, then this means that this particular cubic meter has 1.96 μg of ozone (i.e., one ppb = 1.96 μg/m^3).

Standard Error: 0.002565
Incidence Rate: region-specific daily hospital admission rate for respiratory admissions per person 65+ (ICD codes 460-519)
Population: population of ages 65 and older

Hospital Admissions for Chronic Lung Disease [Moolgavkar, 1997 #53, Minneapolis]

Moolgavkar et al. [1997 #53] examined the relationship between air pollution and hospital admissions (ICD codes 490-496) for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1991. In a Poisson regression, they found no significant effect for any of the pollutants (PM_{10}, ozone, or CO). The effect for ozone was marginally significant. The model with a 100 df smoother was reported to be optimal (p. 368). The C-R function is based on the results from a three-pollutant model (ozone, CO, PM_{10}) using the 100 df smoother.

Multipollutant Model (ozone, CO, PM_{10})

In a model with CO and PM_{10}, the estimated coefficient and standard error are based on the percent increase (4.2) and 95% confidence interval of the percent increase (-1.0-9.4) associated with a change in daily average ozone levels of 15 ppb [Moolgavkar, 1997 #53, Table 4 and p. 366].

Functional Form: Log-linear
Coefficient: 0.002743
Standard Error: 0.001699
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease per person 65+ (ICD codes 490-496)
Population: population of ages 65 and older

Hospital Admissions for Chronic Lung Disease (less Asthma) [Schwartz, 1994 #144, Detroit]

Schwartz [1994 #144] examined the relationship between air pollution and hospital admissions (ICD codes 491-492, 494-496) for individuals 65 and older in Detroit, Michigan, from January 1986 to December 1989. In a two-pollutant Poisson regression model, Schwartz found both PM_{10} and ozone significantly linked to pneumonia and COPD. The authors state that effect estimates were relatively unchanged compared to the unreported single pollutant models. No significant associations were found between either pollutant and asthma admissions. The C-R function for chronic lung disease incidence is based on the results of the "basic" co-pollutant model (ozone and PM_{10}) presented in Table 4 (p. 651).[75]

[75] Schwartz [1994 #144] also reports results using generalized additive models to fit time and temperature variables, however no standard error or confidence intervals were reported.

Multipollutant Model (ozone and PM_{10})

The coefficient and standard error for the "basic" model are reported in Table 4 [Schwartz , 1994 #144, p.651] for a one ppb change in daily average ozone.

Functional Form: Log-linear
Coefficient: 0.00549
Standard Error: 0.00205
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease per person 65+ (ICD codes 490-492, 494-496)
Population: population of ages 65 and older

Hospital Admissions for Pneumonia [Moolgavkar, 1997 #53, Minneapolis]

Moolgavkar et al. [1997 #53] examined the relationship between air pollution and pneumonia hospital admissions (ICD 480-487) for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1991. In a four pollutant Poisson model examining pneumonia admissions in Minneapolis, ozone was significant, while NO_2, SO_2, and PM_{10} were not significant. The model with a 130 df smoother was reported to be optimal (p. 368). The ozone C-R function is based on the results from the four-pollutant model with a 130 df smoother.

Multipollutant Model (ozone, NO_2, PM_{10}, SO_2)

In a model with NO_2, PM_{10},and SO_2, the estimated coefficient and standard error are based on the percent increase (5.7) and 95% confidence interval of the percent increase (2.5-8.9) associated with an increase in daily average ozone levels of 15 ppb [Moolgavkar, 1997 #53, Table 4 and p. 366].

Functional Form: Log-linear
Coefficient: 0.003696
Standard Error: 0.00103
Incidence Rate: region-specific daily hospital admission rate for pneumonia per person 65+ (ICD codes 480-487)
Population: population of ages 65 and older

Hospital Admissions for Pneumonia [Schwartz, 1994 #144, Detroit]

Schwartz [1994 #144] examined the relationship between air pollution and hospital admissions for individuals 65 and older in Detroit, Michigan, from January 1986 to December 1989. In a two-pollutant Poisson regression model, Schwartz found both PM_{10} and ozone significantly linked to pneumonia and COPD. The authors state that effect estimates were

relatively unchanged compared to the unreported single pollutant models. No significant associations were found between either pollutant and asthma admissions. The PM_{10} C-R function for pneumonia incidence is based on results of the "basic" co-pollutant model (ozone and PM_{10}).[76]

Multipollutant Model (ozone and PM_{10})

The ozone C-R function for pneumonia incidence is based on the coefficient and standard error for the "basic" co-pollutant model presented in Table 4 [Schwartz, 1994 #144, p. 651].

Functional Form: Log-linear
Coefficient: 0.00521
Standard Error: 0.0013
Incidence Rate: region-specific daily hospital admission rate for pneumonia per person 65+ (ICD codes 480-487)
Population: population of ages 65 and older

Hospital Admissions for Pneumonia [Schwartz, 1994 #143, Minneapolis]

Schwartz [1994 #143] examined the relationship between air pollution and hospital admissions for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1989. In single-pollutant Poisson regression models, both ozone and PM_{10} were significantly associated with pneumonia admissions. In a two-pollutant model, Schwartz found PM_{10} significantly related to pneumonia; ozone was weakly linked to pneumonia. The results were not sensitive to the methods used to control for seasonal patterns and weather. The ozone C-R function is based on the results of the two-pollutant model (PM_{10} and ozone) with spline smoothing for temporal patterns and weather.

Multipollutant Model (ozone and PM_{10})

In a model with PM_{10} and spline functions to adjust for time and weather, the coefficient and standard error are based on the relative risk (1.22) and 95% confidence interval (1.02, 1.47) for a 50 ppb increase in daily average ozone levels [Schwartz, 1994 #143, Table 4].

Functional Form: Log-linear
Coefficient: 0.003977
Standard Error: 0.001865
Incidence Rate: region-specific daily hospital admission rate for pneumonia per person 65+ (ICD codes 480-487)
Population: population of ages 65 and older

[76] Schwartz [1994 #144] also reports results using generalized additive models to fit time and temperature variables, however no standard error or confidence intervals were reported.

Exhibit D-11 Concentration-Response (C-R) Functions for Ozone and Emergency Room Visits

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
Asthma	Cody et al.	1992	New Jersey (Northern)	All	All	All	SO_2	5-hr avg	Linear	0.0203	0.00717
Asthma	Stieb et al.	1996	New Brunswick, CAN	All	All	All	None	1-hr max	Quadratic	0.00004	0.00002
Asthma	Weisel et al.	1995	New Jersey (Northern and Central)	All	All	All	None	5-hr avg	Linear	0.0443	0.00723

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Emergency Room Visits

Exhibit D-11 summarizes the C-R functions used to estimate the relationship between ozone and emergency room visits. Detailed summaries of each of the studies used to generate the functions are described below, along with the parameters used in each of the functions.

Emergency Room Visits for Asthma [Cody, 1992 #914, Northern NJ]

Cody et al. [1992 #914] examined the relationship between ER visits and air pollution for persons of all ages in central and northern New Jersey, from May to August in 1988-1989. In a two pollutant multiple linear regression model, ozone was linked to asthma visits, and no effect was seen for SO_2. They modeled PM_{10} in separate analysis because of limited (every sixth day) sampling. No significant effect was seen for PM_{10}. The C-R function for ozone is based on results of a co-pollutant model with SO_2 [Cody, 1992 #914, Table 6, p. 191].

Multipollutant Model (ozone and SO_2)

The ozone coefficient and standard error are reported per 1 ppm increment of five-hour ozone levels, which are converted to a 1 ppb increment by dividing by 1,000 [Cody, 1992 #914, Table 6, p. 191].

Functional Form: Linear
Coefficient: 0.0203
Standard Error: 0.00717

Baseline Population: baseline population of Northern New Jersey[77] = 4,436,976
Population: population of all ages

Emergency Room Visits for Asthma [Stieb, 1996 #218, New Brunswick]

Stieb et al. [1996 #218] examined the relationship between ER visits and air pollution for persons of all ages in St. John, New Brunswick, Canada, from May through September in 1984-1992. Ozone was significantly linked to ER visits, especially when ozone levels exceeded 75 ppb. The authors reported results from a linear model, quadratic model, and linear-quadratic model using daily average and 1-hour maximum ozone. In the linear model, ozone was borderline significant. In the quadratic and linear-quadratic models, ozone was highly significant. This is consistent with the author's conclusion that "only ozone appeared to have a nonlinear relationship with visit rates" (p. 1356) and that "quadratic, linear-quadratic, and indicator models consistently fit the data better than the linear model ..." (p. 1358). The linear term in the linear-quadratic model is negative, implying that at low ozone levels, increases in ozone are associated with decreases in risk. Since this does not seem biologically plausible, the ozone C-R function described here is based on the results of the quadratic regression model presented in Table 2 [Stieb et al., 1996 #218, p. 1356], for a change in one-hour maximum ozone levels.

Single Pollutant Model

The coefficient and standard error of the quadratic model are reported in Table 2 [Stieb et al., 1996 #218, p. 1356] for a 1 ppb increase in 1-hour daily maximum ozone levels. The C-R function to estimate avoided emergency visits derived from a quadratic regression model is shown below:

$$\Delta\, Asthma\ ERVisits = \frac{\beta}{BasePop} \cdot [(O_{3,baseline})^2 - (O_{3,control})^2] \cdot pop,$$

Functional Form: Quadratic
Coefficient: 0.00004
Standard Error: 0.00002
Baseline Population: baseline population of St. John, New Brunswick [Stieb, 1996 #218, p. 1354] = 125,000
Population: population of all ages

[77] The population estimate is based on the 1990 population for the eight counties containing hospitals or in the central core of the study. Cody et al. [1992 #914, Figure 1] presented a map of the study area; the counties are: Bergen, Essex, Hudson, Middlesex, Morris, Passaic, Somerset, and Union.

Emergency Room Visits for Asthma [Weisel, 1995 #688, Northern NJ]

Weisel et al. [1995 #688] examined the relationship between ER visits and air pollution for persons of all ages in central and northern New Jersey, from May to August in 1986-1990. A significant relationship was reported for ozone. The C-R function is based on the results of the single pollutant models reported by Weisel et al. [1995 #688, Table 2].

Single Pollutant Model

The coefficient (β) used in the C-R function is a weighted average of the coefficients in Weisel et al. [1995 #688, Table 2] using the inverse of the variance as the weight:

$$\beta = \left(\frac{\sum_{i=1986}^{1990} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1986}^{1990} \frac{1}{\sigma_{\beta_i}^2}} \right) = 0.0443 .$$

The standard error of the coefficient (σ_β) is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$\sigma_\beta^2 = \text{var}\left(\frac{\sum_{i=1986}^{1990} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1986}^{1990} \frac{1}{\sigma_{\beta_i}^2}} \right) = \left(\frac{\sum_{i=1986}^{1990} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\gamma} \right) = \sum_{i=1986}^{1990} \text{var}\left(\frac{\beta_i}{\sigma_{\beta_i}^2 \cdot \gamma} \right).$$

This eventually reduces down to:

$$\sigma_\beta^2 = \frac{1}{\gamma} \Rightarrow \sigma_\beta = \sqrt{\frac{1}{\gamma}} = 0.00723 .$$

Functional Form: Linear
Coefficient: 0.0443
Standard Error: 0.00723
Baseline Population: baseline population of Northern New Jersey[78] = 4,436,976
Population: population of all ages

[78] The population estimate is based on the 1990 population for the eight counties containing hospitals or in the central core of the study. Cody et al. [1992 #914, Figure 1] presented a map of the study area; the counties are: Bergen, Essex, Hudson, Middlesex, Morris, Passaic, Somerset, and Union.

Exhibit D-12 Concentration-Response (C-R) Functions for Ozone and Acute Effects

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
Any of 19 Respiratory Symptoms	Krupnick	1990	Los Angeles, CA	18-64	All	All	COH	1-hr max	Linear	0.000137	0.000070
Minor Restricted Activity Days	Ostro and Rothschild	1989	nationwide	18-64	All	All	$PM_{2.5}$	24-hr avg	Log-linear	0.0022	0.000658
School Loss Days, All Cause	Chen et al.	2000	Washoe Co, NV	6-11	All	All	CO, PM_{10}	1-hr max	Linear	0.013247	0.004985
School Loss Days, All Cause	Gilliland et al.	2001	Southern California	9-10	All	All	None	8-hr avg	Log-linear	0.00755	0.004527
Worker Productivity	Crocker and Horst	1981	nationwide	18-64	All	All	None	24-hr avg	Linear	0.14	–

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Acute Morbidity

Exhibit D-12 summarizes the C-R functions used to estimate the relationship between ozone and acute morbidity. Detailed summaries of each of the studies used to generate the functions are described below, along with the parameters used in each of the functions.

Any of 19 Respiratory Symptoms: Krupnick [1990 #35]

Krupnick et al. [1990 #35] estimated the impact of air pollution on the incidence of any of 19 respiratory symptoms or conditions in 570 adults and 756 children living in three communities in Los Angeles, California from September 1978 to March 1979. Krupnick et al. [1990 #35] listed 13 specific "symptoms or conditions": head cold, chest cold, sinus trouble, croup, cough with phlegm, sore throat, asthma, hay fever, doctor-diagnosed ear infection, flu, pneumonia, bronchitis, and bronchiolitis. The other six symptoms or conditions are not specified.

In their analysis, they included coefficient of haze (COH, a measure of particulate matter concentrations), ozone, NO_2, and SO_2, and they used a logistic regression model that takes into account whether a respondent was well or not the previous day. A key difference between this and the usual logistic model, is that the model they used includes a lagged value of the dependent variable. In single-pollutant models, daily ozone, COH, and SO_2 were significantly related to respiratory symptoms in adults. Controlling for other pollutants, they found that ozone was still significant. The results were more variable for COH and SO_2, perhaps due to collinearity. NO_2 had no significant effect. No effect was seen in children for any pollutant. The results from the two-pollutant model with COH and ozone are used to develop a C-R function.

Multipollutant Model (ozone and coefficient of haze)

The C-R function used to estimate the change in ARD2 associated with a change in daily one-hour maximum ozone[79] is based on Krupnick et al. [1990 #35, p. 12]:[80]

$$\Delta ARD2 \cong \beta^* \cdot \Delta O_3 \cdot pop \,,$$

Functional Form: Linear
Coefficient: first derivative of the stationary probability = 0.000137
Standard Error: 0.0000697

[79]Krupnick et al. [1990 #35] used parts per hundred million (pphm) to measure ozone; the coefficient used here is based on ppb.

[80]Krupnick and Kopp [1988 #318, p. 2-24] and ESEERCO [1994 #323, p. V-32] used the same C-R functional form as that used here.

Population: population of ages 18-64 years[81]

The logistic regression model used by Krupnick et al. [1990 #35] takes into account whether a respondent was well or not the previous day. Following Krupnick et al. (p. 12), the probability that one is sick is on a given day is:

$$probability(ARD2) = \frac{p_0}{1 - p_1 + p_0}$$

$$p_i = probability(ARD2 | sickness\ or\ not_{t-1}) = \frac{1}{1 - e^{\beta_0 + \beta_1 \cdot ARD2_{t-1} + X \cdot \beta}}, for\ i = 0,1.$$

where:

X	=	the matrix of explanatory variables
p_0	=	the probability of sickness on day t, given wellness on day t-1, and
p_1	=	the probability of sickness on day t, given sickness on day t-1.

In other words, the transition probabilities are estimated using a logistic function; the key difference between this and the usual logistic model, is that the model includes a lagged value of the dependent variable.

To calculate the impact of ozone (or other pollutants) on the probability of ARD2, it is possible, in principle, to estimate ARD2 before the change in ozone and after the change:

$$\Delta ARD2 = ARD2_{after} - ARD2_{before}.$$

However the full suite of coefficient estimates are not available.[82] Rather than use the full suite of coefficient values, the impact of ozone on the probability of ARD2 may be approximated by the derivative of ARD2 with respect to ozone:[83]

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[81]The coefficient estimates are based on the sample of "adults," and assumes that individuals 18 and older were considered adult. According to Krupnick et al. [1990 #35, Table 1], about 0.6 percent of the study sample was over the age of 60. This is a relatively small fraction, so it is further assumed that the results do not apply to individuals 65 years of age and older.

[82]The model without NO_2 [Krupnick, 1990 #35, Table V equation 3] was used in this analysis, but the full suite of coefficient estimates for this model were not reported. Krupnick et al. (Table IV) reported all of the estimated coefficients for a model of children and for a model of adults when four pollutants were included (ozone, COH, SO_2, and NO_2). However, because of high collinearity between NO_2 and COH, NO_2 was dropped from some of the reported analyses (Krupnick et al., p. 10), and the resulting coefficient estimates changed substantially (see Krupnick et al., Table V). Both the ozone and COH coefficients dropped by about a factor of two or more.

[83]The derivative result is reported by Krupnick et al. [1990 #35, p. 12].

$$\frac{\partial probability(ARD2)}{\partial O_3} = \frac{p_0 \cdot (1 - p_1) \cdot \beta \cdot [p_1 + (1 - p_0)]}{(1 - p_1 + p_0)^2} = \beta^* ,$$

where β is the reported logistic regression coefficient for ozone. The change in the incidence of ARD2 associated with a given change in ozone is then estimated by:

$$\frac{\partial ARD2}{\partial O_3} \cong \frac{\Delta ARD2}{\Delta O_3}$$

$$\Rightarrow \frac{\Delta ARD2}{\Delta O_3} \cong \beta^*$$

$$\Rightarrow \Delta ARD2 \cong \beta^* \cdot \Delta O_3 .$$

This analysis uses transition probabilities obtained from Krupnick et al. as reported by ESEERCO [1994 #323, p. V-32] for the adult population: $p_1 = 0.7775$ and $p_0 = 0.0468$. This implies:

$$\beta^* = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.00055 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 0.000137 .$$

The *standard error* for the coefficient is derived using the reported standard error of the logistic regression coefficient in Krupnick et al. [1990 #35, Table V]:

$$\beta_{high} = 0.00055 + (1.96 \cdot 0.00027) = 0.00108$$

$$\Rightarrow \beta^*_{high} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.00108 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 0.000268$$

$$\sigma_{\beta, high} = \frac{\beta_{high} - \beta}{1.96} = \frac{(0.000268 - 0.000137)}{1.96} = 0.0000668$$

$$\beta_{low} = 0.00055 - (1.96 \cdot 0.00027) = 0.0000208$$

$$\Rightarrow \beta^*_{low} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.0000208 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 5.17 \cdot 10^{-6}$$

$$\Rightarrow \sigma_{\beta,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{\left(0.000137 + 5.17 \cdot 10^{-6}\right)}{1.96} = 0.0000725$$

$$\sigma_{\beta} = \frac{\sigma_{\beta,high} + \sigma_{\beta,low}}{2} = 0.0000697 .$$

Minor Restricted Activity Days: Ostro and Rothschild [1989 #60]

Ostro and Rothschild [1989 #60] 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.[84] The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for $PM_{2.5}$, two-week average ozone had a 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. The C-R function for ozone is based on the co-pollutant model with $PM_{2.5}$.

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 ozone as individuals under 65. A number of studies have found that hospital admissions for the elderly are related to ozone exposures [e.g., \Schwartz,1995 #153; Schwartz , 1994 #144].

Multipollutant Model (ozone and $PM_{2.5}$)

The coefficient and standard error used in the C-R function are based on a weighted average of the coefficients in Ostro and Rothschild [1989 #60, Table 4]. The derivation of these estimates is described below.

Functional Form: Log-linear
Coefficient: 0.00220
Standard Error: 0.000658
Incidence Rate: daily incidence rate for minor restricted activity days (MRAD) = 0.02137 [Ostro and Rothschild , 1989 #60, p. 243]
Population: adult population ages 18 to 64

[84] The study population is based on the Health Interview Survey (HIS), conducted by the National Center for Health Statistics. In publications from this ongoing survey, non-elderly adult populations are generally reported as ages 18-64. From the study, it is not clear if the age range stops at 65 or includes 65 year olds. We apply the C-R function to individuals ages 18-64 for consistency with other studies estimating impacts to non-elderly adult populations.

The coefficient used in the C-R function is a weighted average of the coefficients in Ostro and Rothschild [1989 #60, Table 4] using the inverse of the variance as the weight:[85]

$$\beta = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = 0.00220 .$$

The standard error of the coefficient is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$\sigma_\beta^2 = VAR\left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = VAR\left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\gamma} \right) = \sum_{i=1976}^{1981} VAR\left(\frac{\beta_i}{\sigma_{\beta_i}^2 \cdot \gamma} \right).$$

This reduces down to:

$$\sigma_\beta^2 = \frac{1}{\gamma} \Rightarrow \sigma_\beta = \sqrt{\frac{1}{\gamma}} = 0.000658 .$$

School Loss Days, All Cause [Chen, 2000 #2101]

Chen et al. [2000 #2101] studied the association between air pollution and elementary school absenteeism (grades 1-6)[86] in Washoe County, Nevada. Daily absence data were available for all elementary schools in the Washoe Country School District. The authors regressed daily total absence rate on the three air pollutants, meteorological variables, and indicators for day of the week, month, and holidays. They reported statistically significant associations between both ozone and CO and daily total absence rate for grades one through six. PM_{10} was negatively associated with absence rate, after adjustment for ozone, CO, and meteorological and temporal variables. The C-R function for ozone is based on the results from a multiple linear regression model with CO, ozone, and PM_{10}.

[85] The calculation of the MRAD coefficient and its standard error is exactly analogous to the calculation done for the work-loss days coefficient based on Ostro [1987 #456].

[86] Assuming that most children start kindergarten at age 5, the corresponding ages for grades 1 through 6 would be 6 through 11.

Multipollutant Model (ozone, CO, and PM_{10})

The coefficient and standard error are presented in Table 3 [Chen, 2000 #2101, p. 1008] for a unit ppm increase in the two-week average of daily one-hour maximum ozone concentration. This is converted to unit ppb increase by dividing by 1,000.

The reported coefficient represents an *absolute* increase in absenteeism rate for a unit increase in ozone. If we apply this study to other locations, we assume that the same absolute increase will occur for a unit increase in ozone, regardless of the baseline rate. If the study location has a particularly high baseline rate, we may be overestimating decreases in absenteeism nationally, and vice-versa. As an example, consider if the baseline absenteeism rate were 10% in the study and 5% nationally. An absolute increase in absence rate of 2% associated with a given increase in ozone reflects a relative increase in absence rate of 20% for the study population. However, in the national estimate, we would assume the same absolute increase of 2%, but this would reflect a relative increase in the absenteeism rate of 40%.

An alternative approach is to estimate apply the *relative* increase in absenteeism rate in the C-R function by adjusting the results by the ratio of the national absenteeism rate to the study-specific rate. As a result, the percent increase in absenteeism rate associated with an increase in ozone is extrapolated nationally rather than the absolute increase in absenteeism rate. The incidence derivation section above describes the data used to estimate national and study-specific absence rates.

In addition to this scaling factor, there are two other scaling factors which are applied to the function. A scaling factor of 0.01 is used to convert the beta from a percentage (x 100) per unit increase of ozone to a proportion per unit increase of ozone. As a result it can be applied directly to the national population of school children ages 6 through 11 to estimate the number of absences avoided.

The final scaling factor adjusts for the number of school days in the ozone season. In the modeling program, the function is applied to every day in the ozone season (May 1 - September 30), however, in reality, school absences will be avoided only on school days. We assume that children are in school during weekdays for all of May, two weeks in June, one week in August, and all of September. This corresponds to approximately 2.75 months out of the 5 month season, resulting in an estimate of 39.3% of days (2.75/5*5/7). The C-R function parameters are shown below.

Functional Form: Linear
Coefficient: 0.013247
Standard Error: 0.004985
Population: population of children ages 6-11

Scaling Factor 1: Ratio of national school absence rate to study-specific school absence rate[87] = 1.081
Scaling Factor 2: Convert beta in percentage terms to a proportion = 0.01
Scaling Factor 3: Proportion of days that are school days in the ozone season[88] = 0.393

School Loss Days, All Cause [Gilliland, 2001 #2151]

Gilliland et al. [2001 #2151] examined the association between air pollution and school absenteeism among 4th grade school children (ages 9-10) in 12 southern Californian communities. The study was conducted from January through June 1996. The authors used school records to collect daily absence data and parental telephone interviews to identify causes. They defined illness-related absences as respiratory or non-respiratory. A respiratory illness was defined as an illness that included at least one of the following: runny nose/sneezing, sore throat, cough, earache, wheezing, or asthma attack. The authors used 15 and 30 day distributed lag models to quantify the association between ozone, PM_{10}, and NO_2 and incident school absences. Ozone levels were positively associated with all school absence measures and significantly associated with all illness-related school absences (non-respiratory illness, respiratory illness, URI and LRI). Neither PM_{10} nor NO_2 was significantly associated with illness-related school absences, but PM_{10} was associated with non-illness related absences. The C-R function for ozone is based on the results of the single pollutant model.

Gilliland et al. [2001 #2151] defines an incident absence as an absence that followed attendance on the previous day and the incidence rate as the number of incident absences on a given day over the population at risk for an absence on a given day (i.e. those children who were not absent on the previous day). Since school absences due to air pollution may last longer than one day, an estimate of the average duration of school absences could be used to calculated the total avoided school loss days from an estimate of avoided new absences. A simple ratio of the total absence rate divided by the new absence rate would provide an estimate of the average duration of school absences, which could be applied to the estimate of avoided new absences as follows:

$$Duration = \frac{totalAbsences}{newAbsences}$$

$$\Delta TotalAbsences = -\left[incidence \cdot \left(e^{-\beta \cdot \Delta O_3} - 1\right)\right] \cdot duration \cdot pop$$

[87] National school absence rate of 5.5% obtained from the U.S. Department of Education [1996 #2377, Table 42-1]. Study-specific school absence rate of 5.09% obtained from Chen et al. [2000 #2101, Table 1].

[88] Ozone is modeled for the 5 months from May 1 through September 30. We assume that children are in school during weekdays for all of May, 2 weeks in June, 1 week in August, and all of September. This corresponds to approximately 2.75 months out of the 5 month season, resulting in an estimate of 39.3% of days (2.75/5*5/7).

Since the function is log-linear, the baseline incidence rate (in this case, the rate of new absences) is multiplied by duration, which reduces to the total school absence rate. Therefore, the same result would be obtained by using a single estimate of the total school absence rate in the C-R function. Using this approach, we assume that the same relationship observed between pollutant and new school absences in the study would be observed for total absences on a given day. As a result, the total school absence rate is used in the function below. The derivation of this rate is described in the section on baseline incidence rate estimation.

Single Pollutant Model

For all absences, the coefficient and standard error are based on a percent increase of 16.3 percent (95% CI -2.6 percent, 38.9 percent) associated with a 20 ppb increase in 8-hour average ozone concentration [2001 #2151, Table 6, p. 52].

A scaling factor is used to adjust for the number of school days in the ozone season. In the modeling program, the function is applied to every day in the ozone season (May 1 - September 30), however, in reality, school absences will be avoided only on school days. We assume that children are in school during weekdays for all of May, two weeks in June, one week in August, and all of September. This corresponds to approximately 2.75 months out of the 5 month season, resulting in an estimate of 39.3% of days (2.75/5*5/7).

In addition, not all children are at-risk for a new school absence, as defined by the study. On average, 5.5% of school children are absent from school on a given day [U.S. Department of Education, 1996 #2377, Table 42-1]. Only those who are in school on the previous day are at risk for a new absence (1-0.055 = 94.5%). As a result, a factor of 94.5% is used in the function to estimate the population of school children at-risk for a new absence.

Functional Form: Log-linear
Coefficient: 0.007550
Standard Error: 0.004527
Incidence Rate: daily school absence rate = 0.055 [U.S. Department of Education, 1996 #2377, Table 42-1]
Population: population of children ages 9-10 not absent from school on a given day[89] = 94.5% of children ages 9-10
Scaling Factor: Proportion of days that are school days in the ozone season[90] = 0.393

Worker Productivity: Crocker and Horst [1981 #636]

[89] The proportion of children not absent from school on a given day (5.5%) is based on 1996 data from the U.S. Department of Education [1996 #2377, Table 42-1].

[90] Ozone is modeled for the 5 months from May 1 through September 30. We assume that children are in school during weekdays for all of May, 2 weeks in June, 1 week in August, and all of September. This corresponds to approximately 2.75 months out of the 5 month season, resulting in an estimate of 39.3% of days (2.75/5*5/7).

To monetize benefits associated with increased worker productivity resulting from improved ozone air quality, we used information reported in Crocker and Horst [1981 #636] and summarized in EPA [1994 #637]. Crocker and Horst examined the impacts of ozone exposure on the productivity of outdoor citrus workers. The study measured productivity impacts as the change in income associated with a change in ozone exposure, given as the elasticity of income with respect to ozone concentration (-0.1427).[91] The reported elasticity translates a ten percent reduction in ozone to a 1.4 percent increase in income. Given the national median daily income for outdoor workers engaged in strenuous activity reported by the U.S. Census Bureau [2002 #2387], $68 per day (2000$),[92] a ten percent reduction in ozone yields about $0.97 in increased daily wages. We adjust the national median daily income estimate to reflect regional variations in income using a factor based on the ratio of county median household income to national median household income. No information was available for quantifying the uncertainty associated with the central valuation estimate. Therefore, no uncertainty analysis was conducted for this endpoint.

Single Pollutant Model

The C-R function for estimating changes in worker productivity is shown below:

$$\Delta productivity = \beta \cdot \frac{Q_1 - Q_0}{Q_1} \cdot dailyincome \cdot pop \cdot,$$

Functional Form: Linear
Coefficient: 0.1427
Daily Income: median daily income for outdoor workers[93]
Population: population of adults 18 to 64 employed as farm workers

[91] The relationship estimated by Crocker and Horst between wages and ozone is a log-log relationship. Therefore the elasticity of wages with respect to ozone is a constant, equal to the coefficient of the log of ozone in the model.

[92] The national median daily income for workers engaged in "farming, forestry, and fishing" from the U.S. Census Bureau [2002 #2387, Table 621, p. 403] is used as a surrogate for outdoor workers engaged in strenuous activity.

[93] The national median daily income for workers engaged in "farming, forestry, and fishing" was obtained from the U.S. Census Bureau [2002 #2387, Table 621, p. 403] and is used as a surrogate for outdoor workers engaged in strenuous activity. This national median daily income ($68) is then scaled by the ratio of national median income to county median income to estimate county median daily income for outdoor workers.

Exhibit D-13 Concentration-Response (C-R) Functions for Ozone and Asthma-Related Effects

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
Asthma Exacerbation, Asthma Attacks	Whittemore and Korn	1980	Los Angeles, CA	All	All	All	TSP	1-hr max	Logistic	0.001843	0.000715

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Asthma-Related Effects

Exhibit D-13 summarizes the C-R functions used to estimate the relationship between ozone and asthma-related effects. Detailed summaries of each of the studies used to generate the functions are described below, along with the parameters used in each of the functions.

Asthma Attacks [Whittemore and Korn, 1980 #634]

Whittemore and Korn [1980 #634] examined the relationship between air pollution and asthma attacks in a survey of 443 children and adults, living in six communities in southern California during three 34-week periods in 1972-1975. The analysis focused on TSP and oxidants (O_x). Respirable PM, NO_2, SO_2 were highly correlated with TSP and excluded from the analysis. In a two pollutant model, daily levels of both TSP and oxidants were significantly related to reported asthma attacks. The results from this model were used, and the oxidant result was adjusted so it may be used with ozone data.

Multipollutant Model (ozone and PM_{10})

The daily one-hour ozone coefficient is based on an oxidant coefficient (1.66) estimated from data expressed in ppm. The coefficient is converted to ppb by dividing by 1,000 and to ozone by multiplying by 1.11.[94] The standard error is calculated from the two-tailed p-value (<0.01) reported by Whittemore and Korn [1980 #634, Table 5], which implies a t-value of at least 2.576 (assuming a large number of degrees of freedom).

Functional Form: Logistic
Coefficient: 0.001843
Standard Error: 0.000715
Incidence Rate: daily incidence of asthma attacks = 0.0550[95]
Population: population of asthmatics of all ages = 3.86% of the population of all ages [American Lung Association, 2002 #2358, Table 7]

[94] The study used oxidant measurements in ppm [Whittemore, 1980 #634, p. 688]; these have been converted to ozone measurements in ppb, assuming ozone comprises 90% of oxidants (i.e., 1.11*ozone=oxidant). It is assumed that the harm of oxidants is caused by ozone. The view expressed in the Ozone Staff Paper [U.S. EPA, 1996 #455, p.164] is consistent with assuming that ozone is the oxidant of concern at normal ambient concentrations: "Further, among the photochemical oxidants, the acute-exposure chamber, field, and epidemiological human health data base raises concern only for ozone at levels of photochemical oxidants commonly reported in ambient air. Thus, the staff recommends that ozone remain as the pollutant indicator for protection of public health from exposure to all photochemical oxidants found in the ambient air."

[95] Based on an analysis of the 1999 National Health Interview Survey, the daily incidence of wheezing attacks for adult asthmatics is estimated to be 0.0550. In the same survey, wheezing attacks for children were examined, however, the number of wheezing attacks per year were censored at 12 (compared to censoring at 95 for adults). Due to the potential for underestimation of the number of children's wheezing attacks, we used the adult rate for all individuals.