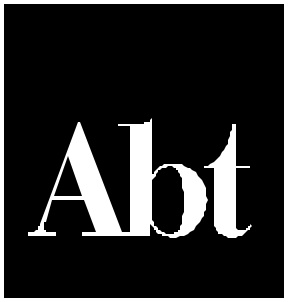

Abt Associates Inc.

Particulate-Related Health Impacts of Emissions in 2001 From 41 Major US Power Plants

November 2002

Prepared for
Environmental Integrity Project
Rockefeller Family Fund
Washington, DC

Project Manager
Eric Schaeffer

Prepared by
Abt Associates Inc.
4800 Montgomery Lane
Bethesda, MD 20814-5341

Particulate-Related Health Impacts of Emissions in 2001 From 41 Major US Power Plants

Executive Summary

Extensive studies worldwide, including important studies by the Harvard School of Public Health and the American Cancer Society, link exposure to fine particle air pollution to heart disease, lung cancer, respiratory ailments, and premature death. Most fine particles are formed when emissions of sulfur dioxide (SO_2) and nitrogen oxides (NO_x) react with ammonia to form particles less than 2.5 microns in diameter, which interfere with the ability of the lungs to absorb oxygen. Power plants are responsible for about two thirds of the SO_2 and one quarter of the NO_x emitted in 2001. While some progress has been made in gradually reducing these pollutants since the passage of the 1990 Amendments to the Clean Air Act, it has been unevenly distributed.

Title IV of the 1990 Amendments to the Clean Air Act (the "acid rain" provisions) and other programs have helped reduce SO_2 emissions from power plants by 32% from 1990 levels through a "cap and trade" program. Because companies can avoid pollution controls under Title IV by purchasing emission "credits" from plants that have cleaned up, SO_2 emissions have actually increased in some states, and declined by significantly less than the 32% national average in other states. Table A presents the state-wide totals for the 1990 and 2001 SO_2 emissions from power plants, and identifies whether each state increased or decreased its emissions over that period. Texas, North Carolina, and Virginia had the largest increases in total tons of SO_2 emitted. Other states, like Pennsylvania, reduced their emissions substantially less than the national average.

Progress in decreasing SO_2 at individual power plants also was uneven over this period. Of the nearly 600 sulfur-emitting plants operating in both 1990 and 2001, 42% (252 plants) increased their sulfur emissions by 2001, and another 10% (62) decreased by less than half the national average. Of particular concern are the major plants that increased their sulfur emissions or declined by less than 15%. Table B provides data for 41 large power plants that meet two criteria:

- Emitted at least 40,000 tons of SO_2 emissions in 2001, and
- Had SO_2 emissions either increase or decline by less than half the national average between 1990 and 2001

For example, SO_2 emissions from three large power plants in western Pennsylvania – Hatfield's Ferry, Keystone, and Homer City – jumped from 408,000 tons in 1990 to 483,000 tons in 2001. In 2001, the 41 plants evaluated by this study accounted for one quarter of the SO_2 emissions from the 1,110 power plants covered by Title IV. Of the 41 plants, 31 increased their emissions, and 10 decreased their emissions by less than 15%.

Sulfur and nitrogen emissions from power plants significantly increase the amount of fine particles in the air. These emissions are associated with a wide range of adverse health effects, including premature mortality, hospital admissions, and work loss days. The Environmental Integrity Project (EIP) asked Abt Associates to estimate the health effects associated with the emissions from 41 of the power plants covered by Title IV.

Abt Associates used EPA's Source Receptor (S-R) Matrix to forecast the amount of fine particles formed from power plant emissions in each county within the continental U.S. The S-R Matrix is not as sophisticated as other models EPA uses to estimate fine particles. However, the S-R Matrix is not nearly as resource-intensive as these more complex models, and is readily able to estimate the impact of individual power plants. EPA has used the S-R Matrix for numerous policy analyses, including EPA's Regulatory Impact Analyses of the 1997 revisions to the Particulate Matter (PM) National Ambient Air Quality Standards (NAAQS), the 1998 State Implementation Plan (SIP) NO_x Call, the 1999 Regional Haze Rule, and the 1990 Tier II automobile exhaust standards. Although more complex PM models exist and are being improved constantly, the S-R Matrix gives a reasonably good first approximation of the likely PM impacts of the power plant emissions.

Abt Associates estimates that between 4,800 and 5,600 premature deaths in 2001 are associated with the emissions from these 41 plants. The alternative estimates are based on two well known recent studies of the relationships between air pollution and premature mortality; the analysis by the Health Effect Institute (Krewski et al., 2000), and a newer analysis that examined the same people over a longer period of time (Pope et al., 2002). In addition to the premature mortality, Abt Associates also estimated over 3,000 hospital admissions or emergency room visits, 930,000 work loss days, 111,000 asthma attacks, and other health effects are associated with the emissions from the 41 plants. Table C shows some of the health effect estimates linked to the SO_2 and NOx emissions from each of the 41 power plants. Additional health results are provided in the report in Exhibits 3.7 through 3.9.

Power plant pollution spreads over a wide area, with most of the fine particle pollution occurring within a 500-mile radius of the plant. Accordingly, the premature mortality and other diseases that result from the specific power plants identified in Table C include estimated impacts both inside and outside the particular state.

Table A State SO_2 and NOx Emission Data. 1990 and 2001

State	SO_2 1990 (Tons)	2001 (Tons)	Change	Ratio '01 to '90		NOx 2001 (Tons)
Oregon	4,936	17,837	12,901	361.4%		11,150
Nebraska	50,378	70,251	19,873	139.4%		47,909
Kansas	87,676	120,307	32,631	137.2%		84,705
Virginia	158,626	217,435	58,809	137.1%		80,529
Montana	17,922	24,403	6,481	136.2%	↑	39,435
North Carolina	336,451	450,486	114,035	133.9%		144,336
North Dakota	123,464	154,934	31,470	125.5%		79,114
South Carolina	167,414	202,501	35,087	121.0%		81,692
Texas	462,345	541,263	78,918	117.1%		310,871
Mississippi	119,071	138,358	19,287	116.2%		57,811
Washington	58,434	66,912	8,478	114.5%		18,333
Louisiana	98,703	112,804	14,101	114.3%		81,289
Arkansas	69,160	78,705	9,545	113.8%		47,558
Minnesota	81,166	91,768	10,602	113.1%		81,083
Colorado	83,186	90,388	7,202	108.7%		71,728
Wyoming	80,877	84,471	3,594	104.4%	Increased Emissions	84,321
Oklahoma	101,852	101,447	-405	99.6%		84,320
Nevada	55,780	54,703	-1,077	98.1%		44,262
New Mexico	63,839	62,198	-1,641	97.4%		83,340
Michigan	369,845	347,070	-22,775	93.8%		140,951
Maryland	282,453	254,482	-27,971	90.1%		72,935
Utah	32,051	28,321	-3,730	88.4%		71,517
Florida	645,131	569,153	-75,978	88.2%		290,843
Alabama	528,627	466,113	-62,514	88.2%		167,497
Pennsylvania	1,213,385	944,877	-268,508	77.9%		220,048
Iowa	173,033	133,562	-39,471	77.2%		78,478
Delaware	46,918	35,411	-11,507	75.5%		13,938
New Hampshire	67,863	48,124	-19,739	70.9%		6,836
Wisconsin	282,243	189,374	-92,869	67.1%		101,169
New Jersey	74,979	50,270	-24,709	67.0%	↓ Decreased Emissions	35,320
Connecticut	52,408	34,117	-18,291	65.1%		13,060
Arizona	119,898	73,329	-46,569	61.2%		93,097
New York	414,789	250,715	-164,074	60.4%		93,181
Maine	11,330	6,817	-4,513	60.2%		2,085
Kentucky	905,084	535,445	-369,639	59.2%		231,822
Georgia	874,630	489,626	-385,004	56.0%		162,379
Indiana	1,499,176	795,506	-703,670	53.1%		306,531
West Virginia	968,611	498,056	-470,555	51.4%		204,304
Ohio	2,211,628	1,125,475	-1,086,153	50.9%		332,903
South Dakota	28,906	13,619	-15,287	47.1%		16,539
Tennessee	796,528	356,608	-439,920	44.8%		154,962
Massachusetts	232,012	102,934	-129,078	44.4%		33,519
Illinois	893,793	368,218	-525,575	41.2%		199,860
Missouri	775,726	231,562	-544,164	29.9%		144,742
Washington, DC	2,523	754	-1,769	29.9%		429
California	7,365	1,886	-5,479	25.6%		18,837
Rhode Island	1,090	9	-1,081	0.8%		752
Idaho	0	4	4			198
Vermont	0	5	5			229
Total	15,733,305	10,632,613	-5,100,692	67.6%		4,742,747

Table B SO_2 and NOx Emission Data for 41 Power Plants

		SO2 Emissions (tons)				NOx Emissions (Tons)
		1990	2001	Ratio, 2001/1990		2001
Emissions for Individual Major Power Plants						
Jeffrey EC	KS	16,528	60,924	368.6%		14,722
Brandon Shores	MD	22,077	46,766	211.8%		13,746
ED Edwards	IL	24,082	50,126	208.1%		9,612
Leland Olds	ND	27,364	51,456	188.0%		14,780
Greene County	AL	23,390	43,115	184.3%		11,536
Scherer	GA	41,417	75,423	182.1%		31,274
Harding St. Station	IN	32,735	43,053	131.5%		6,724
Big Brown	TX	55,278	70,594	127.7%		12,520
Homer City	PA	109,449	137,573	125.7%		24,991
Barry	AL	53,378	65,902	123.5%		23,026
Monticello	TX	71,056	87,263	122.8%		19,106
Chesterfield	VA	53,920	65,995	122.4%		16,418
Big Cajun	LA	41,930	50,217	119.8%		21,158
Cheswick	PA	41,279	49,002	118.7%		6,749
Keystone	PA	134,848	159,725	118.4%		19,239
FJ Gannon	FL	47,803	55,036	115.1%		25,332
Centralia	WA	58,434	66,906	114.5%		18,244
Hatfields Ferry	PA	163,432	185,496	113.5%		27,402
Roxboro	NC	83,332	94,206	113.0%		25,448
Marshall	NC	68,628	77,291	112.6%		20,124
Widows Creek	AL	38,291	42,788	111.7%		26,242
Crystal River	FL	86,824	94,851	109.2%		34,712
Big Sandy	KY	51,157	55,846	109.2%		19,899
Belews Creek	NC	76,251	83,077	109.0%		34,203
Johnsonville	TN	86,688	94,199	108.7%		20,394
Rockport	IN	53,755	57,365	106.7%		34,997
Pleasants	WV	42,066	44,815	106.5%		14,562
JH Campbell	MI	41,002	43,513	106.1%		30,887
Martin Lake	TX	62,392	66,134	106.0%		22,080
JH Miller	AL	46,323	47,615	102.8%		29,944
R Gallagher	IN	46,458	47,511	102.3%		6,663
Kingston	TN	92,821	90,291	97.3%		26,166
WA Parish	TX	54,528	52,534	96.3%		14,079
Fort Martin	WV	83,534	79,661	95.4%		12,334
Mohave	NV	44,372	41,299	93.1%		19,430
John E Amos	WV	84,943	78,851	92.8%		36,368
Bull Run	TN	47,146	43,049	91.3%		17,319
EW Brown	KY	55,104	49,106	89.1%		7,800
Warrick	IN	59,697	52,777	88.4%		17,228
Morgantown	MD	85,290	75,335	88.3%		16,760
Conesville	OH	113,635	96,738	85.1%		22,092
Total		2,522,637	2,873,423	114.9%		826,308

↑ Increased Emissions

↓ Decreased Emissions

Table C Health Effects from SO_2 and NOx Emissions from 41 Power Plants

	Mortality		Chronic Disease	Hospital Admission	ER Visits	Daily Illness	
	Pope et al. '02	Krewski et al. '00	Chronic Bronchitis	3 Different Types, Combined	Asthma (ages < 65)	Work Loss Day	Asthma Attack
Health Effects for Individual Major Power Plants							
Barry AL	60	69	40	21	15	11,066	1,340
Belews Creek NC	149	171	107	55	40	29,503	3,458
Big Brown TX	71	82	52	27	21	14,604	1,761
Big Cajun 2 LA	43	50	29	15	11	8,317	979
Big Sandy KY	102	118	70	37	26	19,528	2,260
Brandon Shores MD	86	99	62	33	23	16,893	2,001
Bull_Run TN	73	85	51	27	19	14,144	1,643
Centralia WA	29	34	26	13	10	6,930	855
Chesterfield VA	132	152	97	50	36	26,827	3,136
Cheswick PA	99	114	67	37	25	18,524	2,149
Conesville OH	201	232	138	74	51	37,868	4,424
Crystal River FL	107	123	69	39	24	17,500	2,282
ED Edwards IL	103	118	70	37	27	19,661	2,325
EW Brown KY	90	103	60	32	23	17,127	1,951
FJ Gannon FL	95	110	62	34	21	15,416	2,019
Fort Martin WV	155	179	106	57	39	29,558	3,395
Greene County AL	52	60	34	18	13	9,659	1,140
Harding St Station IN	101	116	70	36	27	19,331	2,292
Hatfields Ferry PA	360	415	246	132	90	68,601	7,856
Homer City PA	269	310	187	100	68	51,337	5,962
Jeffrey EC KS	41	47	28	15	11	7,868	931
JH Miller AL	68	79	48	25	18	12,997	1,581
JH Campbell MI	91	105	63	33	24	17,415	2,054
John E Amos WV	146	168	103	54	38	28,089	3,295
Johnsonville TN	155	179	110	57	42	29,315	3,613
Keystone PA	302	348	208	112	76	57,634	6,663
Kingston TN	149	171	127	63	48	28,727	4,128
Leland Olds ND	101	116	69	37	26	19,405	2,301
Marshall NC	139	161	118	59	44	27,734	3,852
Martin Lake TX	68	79	49	26	19	13,042	1,634
Mohave NV	41	47	35	18	14	9,264	1,214
Monticello TX	84	96	58	31	23	16,033	1,959
Morgantown MD	130	149	91	48	34	25,269	2,938
Pleasants WV	85	98	59	32	22	16,194	1,897
R Gallagher IN	101	116	68	36	26	19,166	2,222
Rockport IN	112	129	75	39	28	21,083	2,450
Roxboro NC	153	176	115	59	43	30,462	3,737
Scherer GA	107	123	73	38	28	20,924	2,414
WA Parish TX	56	65	42	21	18	12,783	1,474
Warrick IN	155	179	103	54	39	29,242	3,374
Widows Creek AL	79	91	56	29	21	15,123	1,818
Total	4,740	5,457	3,341	1,758	1,250	910,165	108,774

Additional health results are provided in the report in Exhibits 3.7 through 3.9.

Particulate-Related Health Impacts of Emissions in 2001 From 41 Major US Power Plants

Introduction

Power plants are large emitters of sulfur dioxide (SO_2) and nitrogen oxides (NO_x), particularly in the Midwest, where power plants dominate emissions. Perhaps the most hazardous contribution of these gaseous emissions is through the formation of secondary fine particulate matter. Over the past decade, numerous studies have linked particulate matter (PM) to a wide range of adverse human health effects, ranging from premature death, hospital admissions and asthma attacks to chronic bronchitis. A substantial portions of the ambient levels of secondarily formed fine PM is formed from SO_2 and NO_x emissions from fossil fuel powered electricity generation, especially coal-fired power plants. The other major emission source that leads to the formation of ambient fine PM are mobile sources (cars and trucks), with other emission sources accounting for a smaller share of the total fine PM levels.

Since passage of the Clean Air Act Amendments of 1990, the amount of SO_2 emitted by power plants covered by Title IV (the "Acid Rain Bill") of the Clean Air Act has gone down by 32 percent (5.1 million tons). Although total US SO_2 emissions have gone nearly one third, not all individual power plants decreased their emissions by that much. Of the 608 power plants operating in both 1990 and 2001, 285 facilities increased their emissions, and 54 decreased their SO_2 emissions by less than 15 percent.

In a previous analysis for the Environmental Integrity Project, Abt Associates estimated the quantity of health effects attributable to fine PM formed from emissions from the major generating facilities of eight electricity generating companies.[1] That analysis examined the health effects attributable to expected future levels of emissions from 83 power plants in the central and eastern United States. This previous study examined the emissions estimated to be released in 2007, after full implementation of all currently mandated major federal regulatory programs affecting electricity generation emissions. Another study Abt Associates prepared for the Clean Air Task Force[2] also looked at 2007, examining the health impacts associated with all major power plants throughout the US.

Instead of analyzing the health impacts in a future year (2007), this current report estimates these adverse health effects of the 2001 SO_2 and NO_x emissions from 41 major power plants. The 41 power plants meet two criteria:

- Emitted at least 40,000 Tons of SO_2 in 2001, and

---

[1] *Particulate-Related Health Impacts of Eight Electric Utility Systems,* Abt Associates Inc., April, 2002.

[2] *The Particulate-Related Health Benefits of Reducing Power Plant Emissions,* Abt Associates Inc., October 2000

! Decreased their SO_2 emissions by 15 % or less since 1990

These 41 plants emitted over 2.9 million tons of SO_2 in 2001, accounting for 28 % of all US SO_2 emissions from power plants. The plants also emitted 840,000 tons of NO_x , or 18 % of total NO_x power plant emissions.

Using EPA data on actual SO_2 and NO_x emissions in 2001 from 41 major electricity generating plants, in this study Abt Associates uses the Source-Receptor (S-R) Matrix (the same simple reduced-form air quality model previously used to estimate impacts in the 83 facility study) to estimate the amount of ambient PM in each county in the US resulting from the emissions from each of the 41 major electricity generating plants. As in the previous analyses, well-established relationships between PM levels and adverse health effects were used to estimate the numbers of excess cases of health effects, ranging from asthma attacks to premature mortality, that are attributable to the increased PM levels from the power plants' emissions.

Section 1 describes the 2001 SO_2 and NO_x emission inventory for the 41 power plants, and the methods used to estimate the ambient PM levels associated with those emissions. Section 2 includes a brief review of the studies that we used to estimate each health effect. In Section 3, we present the results of our analysis. In addition, this study has five appendices. Appendix A lists the nine electric utility systems and the individual plants comprising each system. Appendices B, C and D present a detailed examination of how we derived our pollution emission estimates and translated emissions into forecasts of ambient PM levels. Appendix E presents a derivation of the PM concentration-response (C-R) functions used in the analysis.

1. Particulate Matter Modeling and Emissions

We used a version of the Phase II Source Receptor (S-R) Matrix to forecast PM formation in each county within the continental US. The S-R Matrix is a reduced-form model that consists of fixed coefficients that reflect the relationship between annual average PM concentration values at a single receptor in the center of each county, and the contribution by PM species to this concentration from each emission source in all counties in the 48 contiguous states.

The S-R Matrix is not as sophisticated as other models such as the Regulatory Modeling System for Aerosols and Acid Deposition (REMSAD), California Puff Model (CAL-PUFF) and Community Multi-Scale Air Quality (CMAQ). However, the S-R Matrix is not nearly as resource-intensive as these more complex models, and is readily able to estimate the impact of individual power plants, as well as groups of power plants. EPA has used the S-R Matrix for numerous policy analyses, including EPA's Regulatory Impact Analyses of the 1997 revisions to the PM NAAQS, the 1998 SIP NO_x Call, the 1999 Regional Haze Rule, and the 1990 Tier II automobile exhaust standards. Although more complex PM models exist and are being improved constantly, it is not unreasonable to say that the S-R Matrix gives a reasonably good first approximation of the likely PM impacts of the emissions from the power plants in this study.

For this analysis we estimated the portion of PM formed from the power plants' emissions using the major power plant component of the S-R Matrix, which Pechan developed with the Climatological Regional Dispersion Model (CRDM) (Pechan, 1997 and 2000). For inputs, the S-R Matrix uses emission sources throughout the 48 states and border areas and produces county-level annual average concentrations of various PM-related species, including primary PM less than 10 microns, or PM_{10}, and PM less than 2.5 microns, or $PM_{2.5}$, nitrate, sulfate and ammonia.

The CRDM estimates county-level annual-average PM and PM precursor concentrations from county-level source areas across the United States, by explicitly quantifying the relationships describing the dispersion, chemistry, and deposition processes. It includes wet and dry deposition of gases and PM as well as linear chemical oxidation of SO_2 to sulfate and NO_x to nitrate. As in SLIM3 and ISC2LT, the model is based upon a sector-average approach for transport and dispersion (Turner, 1979) that is recommended for calculating long-term average concentrations.

Using results from CRDM, Pechan developed four matrixes of S-R transfer coefficients that link emissions from every county and major elevated point source in the US and border areas to air quality within every US county. Each coefficient in a matrix represents the incremental ambient air quality impact of a given species or emission at a given receptor from a particular area or point source.

Rather than develop a full 2001 emissions inventory for all source categories, this analysis used one portion of the S-R Matrix: the source-receptor coefficients for 468 major power plants operating in 2001. These 468 plants account for the vast majority of the 2001 SO_2 and NO_x emissions from power plants. Of the 1,110 facilities included in EPA's online database[3] of facilities affected by Title IV, the 468 power plants included in the 2001 S-R Matrix account for 97.2% of the total SO_2 emissions in 2001, and 94.8% of the total NO_x emissions, from all Title IV facilities.

The S-R Matrix results of the contribution of ammonium sulfate and nitrate in every county coming from each modeled power plant developed for the previous Abt Associates analyses based on 2007 emissions projections were adjusted for use in this 2001 current analysis by scaling the power plant contributions to reflect the differences in the emissions in each of the two years. The total 2001 emissions for the 468 modeled power plants are relatively close to the 2007 emissions: the actual SO_2 emissions in 2001 were 1.53% higher than the SO_2 emissions estimated for 2007 from the same 468 plants, while the NO_x emissions were 9.87% higher. Given the linear nature of the S-R Matrix transfer coefficients, it is possible to scale the S-R Matrix estimated concentrations by the 2001/2007 ratio of the emissions of each emission species.

To give an example of how the scaling was conducted, in 2001 the Barry Steam Plant in Mobile County, Alabama, emitted 65,902 tons of SO_2. The 2007 analysis estimated the Barry

---

[3]Data Source: EPA's Clean Air Markets Division - Data and Maps "Create Queries - Queries with Emissions Data" at http://cfpub.epa.gov/gdm/ (data obtained Sept. 30, 2002)

Plant SO_2 emissions at 62,536 tons. Hence the 2001/2007 ratio of SO_2 emissions is 1.0538. Therefore, to adjust the S-R Matrix results from 2007 to 2001, the estimated ambient concentration of ammonium sulfate in each county estimated to originate from the Barry Plant's 2007 SO_2 emissions is multiplied by 1.0538 to estimate the amount of sulfate from the Barry Plant in 2001. This Barry Plant SO_2 adjustment factor is applied to every county that is impacted by sulfur from the Barry Plant. Similarly, we adjusted the amount of nitrate in each county from the Barry Plant by the 2001/2007 ratio of NO_x emissions from the Barry Plant (ratio = 1.367). We made a similar set of adjustments for each of the 468 power plants modeled in the S-R Matrix.

The 2001 emissions data for Title IV-affected power plants is available online through EPA's Clean Air Markets Division website. Using information from the Clean Air Markets website, we aggregated the total annual emissions of SO_2 and NO_x from each operating unit within each facility to the facility level. Appendix A presents the state level emission totals for all power plants, and Appendix B presents the emission totals for each of the 41 modeled facilities.

2. Adverse Health Effects

This section describes the individual effects and the methods used to quantify changes in the expected number of incidences of various health effects. Exhibit 2-1 presents the PM-related health endpoints included in this analysis. Appendix C presents a detailed description of each of the concentration-response (C-R) functions used in this analysis.

Exhibit 2-1 PM-Related Health Endpoints

Health Effect	Population	PM Measure	Study
Mortality			
Associated with long-term exposure	Ages 30+	$PM_{2.5}$	Pope et al. (2002) Krewski et al. (2000)
Chronic Illness			
Chronic Bronchitis	Ages 30+	PM_{10}	Schwartz (1993)
Hospital Admissions			
COPD (ICD-9 codes 4490-492, 494-496)	Age 65+	PM_{10}	Samet et al. (2000)[a]
Pneumonia (ICD-9 codes 480-487)	Age 65+	PM_{10}	Samet et al. (2000)[a]
Cardiovascular (ICD-9 codes 390-429)	Age 65+	PM_{10}	Samet et al. (2000)[a]
Asthma (ICD code 493)	< 65	$PM_{2.5}$	Sheppard et al. (1999)
Asthma-related ER visits	< 65	PM_{10}	Schwartz et al. (1993)
Respiratory Symptoms/Illnesses Not Requiring Hospitalization			
Acute bronchitis	Ages 8-12	$PM_{2.5}$	Dockery et al. (1989)
Lower respiratory symptoms (LRS)	Ages 7-14	$PM_{2.5}$	Schwartz et al. (1994)
Upper respiratory symptoms (URS)	Asthmatics, ages 9-11	PM_{10}	Pope et al. (1991)
Minor restricted activity day (MRAD) (adjusted for asthma attacks)	Ages 18-65	$PM_{2.5}$ (estimated)	Ostro and Rothschild (1989) [b]
Work loss days (WLDs)	Ages 18-65	$PM_{2.5}$	Ostro (1987)
Asthma Attacks	Asthmatics, all ages	PM_{10}	Whittemore and Korn (1980)

[a] The pooled estimate, based on distributed lag models in each of 14 cities, is used because the estimated coefficients based on pooling are substantially more stable than the individual city-specific estimates.

[b] We subtract asthma attacks from our MRAD estimate to avoid the possibility of double-counting effects.

Premature Mortality

Effects of changes in PM concentrations on mortality are estimated as a count of the expected number of premature deaths attributable to a given reduction in PM concentrations. There are two types of exposure to elevated levels of air pollution that may result in premature mortality: acute and chronic. Acute (short-term) exposure (e.g., exposure on a given day) to peak pollutant concentrations may result in excess mortality on the same day or within a few days of the elevated exposure. Chronic (long-term) exposure (e.g., exposure over a period of a year or more) to higher levels of pollution may result in mortality in excess of what it would be if pollution levels were lower. The excess mortality that occurs will not necessarily be associated with any particular episode of elevated air pollution levels. The estimates of premature mortality presented in this report are based on long-term studies.

Short-Term Versus Long-Term Studies

There are two types of epidemiological studies that examine the relationship between mortality and exposure. Long-term studies (e.g., Pope et al., 2002) estimate the association between long-term exposure to air pollution and the survival of members of a large study population over an extended period of time. Such studies examine the health endpoint of concern in relation to the general long-term level of the pollutant of concern, for example, relating annual mortality to some measure of annual pollutant level. Daily peak concentrations would impact the results only insofar as they affect the measure of long-term pollutant concentration.

By contrast, short-term studies relate daily levels of the pollutant to daily mortality. By their basic design, daily studies can detect acute effects but cannot detect the effects of long-term exposures. A chronic exposure study design (a prospective cohort study, such as the Pope and Krewski studies) is best able to identify the long-term exposure effects, and may detect some of the short-term exposure effects as well. Because a long-term exposure study may detect some of the same short-term exposure effects detected by short-term studies, including both types of study in a benefit analysis would likely result in some degree of double counting of benefits. While the long-term study design is preferred, these types of studies are expensive to conduct and consequently there are relatively few well-designed long-term studies. Because long-term studies are considered to be the better method, they are used to estimate the premature mortality associated with the 41 power plants in this report.

Issues Concerning Degree of Prematurity of Mortality

It is possible that the short-term studies are detecting an association between PM and mortality that is primarily occurring among terminally ill people. Critics of the use of short-term studies for policy analysis purposes correctly point out that an added risk factor that results in terminally ill people dying a few days or weeks earlier than they otherwise would have (referred to as “short-term harvesting”) is potentially included in the measured PM mortality “signal” detected in such a study. While some of the detected excess deaths may have resulted in a substantial reduction in lifespan, others may have resulted in a relatively small decrease in lifespan. Studies by Spix et al. (1993) and Pope et al. (1992) yield conflicting evidence, suggesting that harvesting may represent anywhere from zero to 50 percent of the

deaths estimated in short-term studies, while Zeger et al. (1999) and Schwartz (2000; 2001) report that short-term harvesting does not appear to represent a large fraction of mortality.[4]

Estimating PM-Related Premature Mortality

We estimate $PM_{2.5}$ -related mortality using C-R functions estimated from two long-term studies: by Pope et al. (2002) and Krewski et al. (2000). The Pope et al. (2002) study is a continuation of the study by Pope et al. (1995), which estimated the association between long-term (chronic) exposure to $PM_{2.5}$ and the survival of members of a large study population. The Pope et al. (2002) study produces modestly (i.e., 13%) smaller estimates of attributable premature mortality than a study by Krewski et al. (2000). The Krewski et al. (2000) study is a reanalysis of the original Pope et al. (1995) data. The Krewski reanalysis was conducted for Health Effects Institute (HEI), a research organization jointly sponsored by industry and the EPA. The HEI-sponsored reanalysis provided an opportunity for in-depth peer review, replication of the original results, and additional research exploring alternative methods.

The Pope et al. (2002) study reported separate results for not only all-cause deaths, but also separate results for lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-519), and "all other" deaths.[5] Like the earlier studies, Pope et al. (2002) found that mean $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. In addition, Pope et al. (2002) identified a significant relationship between $PM_{2.5}$ and lung cancer mortality, a result not found in the earlier studies. We present separate estimates of the incidence of lung cancer and cardiopulmonary mortality based on Pope et al. (2002), in addition to the all-cause estimates from both the Pope and Krewski studies.

Pope et al. (2002) used three alternative measures of $PM_{2.5}$ exposure – metropolitan area-wide annual mean PM levels from the beginning of the 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 PM data, conducted 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). All are significantly linked to premature mortality, with the exposure estimate based on data from 1979-1983 showing the smallest impact. Following previous work performed for EPA, we use the all-cause mortality coefficient to estimate the impact of $PM_{2.5}$ on premature mortality. In deciding between the three exposure measures, we chose the more conservative estimate based on data from 1979-1983.

---

[4]Zeger et al. (1999, p. 171) reported that: "The TSP-mortality association in Philadelphia is inconsistent with the harvesting-only hypothesis, and the harvesting-resistant estimates of the TSP relative risk are actually larger – not smaller – than the ordinary estimates."

[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.

Chronic Bronchitis

Onset of bronchitis has been associated with exposure to air pollutants. Three studies have linked the onset of chronic bronchitis in adults to PM. These results are consistent with research that has found that chronic exposure to pollutants leads to declining pulmonary functioning (Detels et al., 1991; Ackermann-Liebrich et al., 1997; Abbey et al., 1998).

We estimate the changes in the number of new cases of PM-related chronic bronchitis using the study by Schwartz (1993) listed in Exhibit 2-2. The Schwartz study is somewhat older than other available studies (e.g., a series of studies by Abbey et al.), and uses a cross-sectional design. However, the Schwartz study is based on a national sample, unlike the Abbey et al. study which is restricted to a sample of California residents.

Exhibit 2-2 Chronic Bronchitis Studies

Location	Study	Pollutants Used in Final Model	Population Age
United States	Schwartz (1993)	PM_{10}	30+

Schwartz (1993) examined survey data collected from 3,874 adults ranging in age from 30 to 74, and living in 53 urban areas in the US. The survey was conducted between 1971 and 1975, as part of the National Health and Nutrition Examination Survey, and is representative of the non-institutionalized US population. Schwartz (1993, 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 and annual levels of total suspended particulates (TSP) collected in the year prior to the survey.

Hospital Admissions

We estimate the impact of PM on both respiratory and cardiovascular hospital admissions, as well as emergency room visits for asthma. Exhibit 2-3 lists the respiratory and cardiovascular hospital admissions studies that we use to develop C-R functions.

Exhibit 2-3 Hospital Admissions Studies

Location	Study	Health Effects Estimated (ICD code)	Pollutants Used in Final Model	Age of Study Population
Fourteen U.S. Cities [a]	Samet et al. (2000)	pneumonia (480-487); COPD (490-492, 494-6)	PM_{10}	>64
Seattle, WA	Sheppard et al. (1999)	asthma (493)	$PM_{2.5}$	<65
Fourteen U.S. Cities [a]	Samet et al. (2000)	cardiovascular illness (390 - 429)	PM_{10}	>64

[a] Birmingham, Alabama; Boulder, Colorado; Canton, Ohio; Chicago, Illinois; Colorado Springs, Colorado; Detroit, Michigan; Minneapolis/St. Paul, Minnesota; Nashville, Tennessee; New Haven, Connecticut; Pittsburgh, Pennsylvania; Provo/Orem, Utah; Seattle, Washington; Spokane, Washington; and Youngstown, Ohio.

PM-Related Respiratory and Cardiovascular Hospital Admissions

Respiratory and cardiovascular hospital admissions are the two broad categories of hospital admissions that have been related to exposure to both PM and ozone. Several epidemiological studies have estimated C-R functions that included both PM and ozone. However, a study by the Health Effects Institute (HEI) (Samet et al., 2000) estimated separate models for PM_{10} and pneumonia, Chronic Obstructive Pulmonary Disease (COPD) and cardiovascular diseases in each of fourteen cities in the United States, as well as pooled estimates across these cities. The fourteen cities included in the HEI hospital admissions study are Birmingham, Alabama; Boulder, Colorado; Canton, Ohio; Chicago, Illinois; Colorado Springs, Colorado; Detroit, Michigan; Minneapolis/St. Paul, Minnesota; Nashville, Tennessee; New Haven, Connecticut; Pittsburgh, Pennsylvania; Provo/Orem, Utah; Seattle, Washington; Spokane, Washington; and Youngstown, Ohio.

The Samet et al. (2000) pooled estimates are preferable to previously estimated models for several reasons. First, they used distributed lag models that they designed to capture not only same-day effects of PM but the effects of PM on a series of days subsequent to exposure. This captures the full impact of PM on hospital admissions. Samet et al. noted that because of serial correlation, the coefficients of the PM lags tend to be unstable (i.e., have large variances) in single-city models; however, the pooled estimates, based on all fourteen cities, are more stable because they are based on much larger sample sizes. A second advantage of the pooled estimates is that they represent the PM effect across a range of cities in the US. Although other studies have estimated C-R functions in various cities in the US, many of these cities (e.g., Minneapolis/St. Paul, Birmingham, Detroit, Spokane, New Haven, and Seattle) are included in Samet et al., which is a more recent analysis of the PM-hospital admissions relationships includes these cities.

Although the Samet et al. (2000) models do not include other pollutants, they do investigate the impact of omitting other pollutants on the estimated PM effects on hospital admissions (see Figures 33 and 34, Samet et al. (2000)). The study authors conclude that the omission of SO_2 and ozone from the

models had virtually no effect on the estimated PM effect in any of the three pooled estimates (for cardiovascular diseases, COPD, and pneumonia). While Figure 34 suggests that this is the case for cardiovascular diseases and pneumonia, the omission of ozone from the model appears to have resulted in a downward-biased estimate of the PM effect on hospital admissions for COPD. This suggests that using the pooled estimate for COPD will tend to understate the PM effect.

We use Samet et al. (2000) to estimate separate C-R functions for pneumonia and COPD hospital admissions for people 65 years and older. In addition, we use another study by Sheppard et al. (1999) to estimate a C-R function for asthma hospital admissions for people under 65.

Asthma-Related Emergency Room (ER) Visits

We use one C-R function to estimate the effects of PM exposure to asthma-related ER visits. In a study of Seattle residents, Schwartz et al. (1993) found PM_{10} to be significantly related to asthma-related ER visits.

Because we are estimating ER visits as well as hospital admissions for asthma, we must avoid counting twice the ER visits for asthma that are subsequently admitted to the hospital. To avoid double-counting, the baseline incidence rate for emergency room visits is adjusted by subtracting the percentage of patients that are admitted into the hospital. Three studies provide some information to do this: Richards et al. (1981, p. 350) reported that 13% of children's ER visits ended up as hospital admissions; Lipfert (1993, p. 230) reported that ER visits (for all causes) are two to five times more frequent than hospital admissions; and Smith et al. (1997, p. 789) reported 445,000 asthma-related hospital admissions in 1987 and 1.2 million asthma ER visits. The study by Smith et al. appears to be the most relevant as it is a national study and looks at all age groups. Assuming that air-pollution related hospital admissions first pass through the ER, the reported incidence rates suggest that 37% (or 445,000/1,200,000) of ER visits are subsequently admitted to the hospital, or that ER visits for asthma occur 2.7 times as frequently as hospital admissions for asthma. The baseline incidence of asthma ER visits is therefore taken to be 2.7 times the baseline incidence of hospital admissions for asthma. To avoid double-counting, however, only 63% of the resulting change in asthma ER visits associated with a given change in pollutant concentrations is counted in the ER visit incidence change.

Acute Illnesses and Symptoms Not Requiring Hospitalization

We consider in this section a number of acute symptoms that do not require hospitalization, such as acute bronchitis, and upper and lower respiratory symptoms. Exhibit 2-4 lists the studies that we use to develop C-R functions.

Exhibit 2-4 Studies of Symptoms/Illnesses Not Requiring Hospitalization

Health Effect	Study	Pollutants	Study Population
Acute bronchitis	Dockery et al. (1996)	$PM_{2.5}$	Ages 8-12
Upper respiratory symptoms (URS)	Pope et al. (1991)	PM_{10}	Asthmatics, ages 9-11
Lower respiratory symptoms (LRS)	Schwartz et al. (1994)	$PM_{2.5}$	Ages 7-14
Minor restricted activity day (MRAD)	Ostro and Rothschild (1989)	$PM_{2.5}$	Ages 18-65
Asthma Attacks	Whittemore and Korn (1980)	PM_{10}	Asthmatics, all ages
Work loss days (WLDs)	Ostro (1987)	$PM_{2.5}$	Ages 18-65

Acute Bronchitis

Dockery et al. (1996) examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a study of 13,369 children ages 8-12 living in 24 communities in the US and Canada. Health data were collected between 1988 and 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 levels of sulfates and particle acidity were significantly related to bronchitis, and $PM_{2.5}$ and PM_{10} were marginally significantly related to bronchitis.

Upper Respiratory Symptoms (URS)

Using logistic regression, Pope et al. (1991) 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. Pope et al. then related the daily occurrences of URS and LRS, as defined above, 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 reportedly low during this period, and were not included in the analysis.

The sample in the Pope et al. is relatively small and is most representative of the asthmatic population, rather than the general population. The school-based subjects (ranging in age from 9 to 11) were chosen based on "a positive response to one or more of three questions: ever wheezed without a cold, wheezed for 3 days or more out of the week for a month or longer, and/or had a doctor say the 'child has asthma.'" (Pope et al., 1991, p. 669) The patient-based subjects (ranging in age from 8 to 72) were receiving treatment for asthma and were referred by local physicians. Regression results for the school-based sample (Pope et al., 1991, Table 5) show that PM_{10} was 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.

Lower Respiratory Symptoms (LRS)

Schwartz et al. (1994) used logistic regression to link lower respiratory symptoms in children with SO_2, NO_2, ozone, PM_{10}, $PM_{2.5}$, sulfate and H^+ (hydrogen ion). Children were selected for the study if they were exposed to indoor sources of air pollution, including gas stoves and parental smoking. The study enrolled 1,844 children into year-long studies that were 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 have been in the eighth grade (ages 13-14), suggesting an age range of 7 to 14 years.

In single pollutant models, SO_2, NO_2, $PM_{2.5}$, and PM_{10} were significantly linked to cough. In two-pollutant models, PM_{10} had the most consistent relationship with cough; ozone was marginally significant, controlling for PM_{10}. In models for upper respiratory symptoms, Schwartz et al. reported a marginally significant association for PM_{10}. In models for lower respiratory symptoms, they reported significant single-pollutant models, using SO_2, ozone, $PM_{2.5}$, PM_{10}, sulfate (SO_4) , and acidity (H^+ ions).

Minor Restricted Activity Days (MRADs)

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ on the incidence of minor restricted activity days (MRAD) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. We developed separate coefficients for each year in the analysis (1976-1981), which were then combined for use in this analysis. For the coefficient in the C-R function, we use an inverse-variance weighted average of the coefficients in Ostro (1987, Table IV).

Asthma Attacks

Whittemore and Korn (1980) 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 from 1972 to1975. The analysis focused on Total Suspended Particles (TSP, roughly equivalent to PM_{15}) and ozone. Respirable PM, NO_2, and SO_2 were highly correlated with TSP and excluded from the analysis. In a two-pollutant model, daily levels of both TSP and ozone were significantly related to reported asthma attacks.

Work-Loss Days (WLD)

Ostro (1987) estimated the impact of $PM_{2.5}$ on the incidence of work-loss days (WLDs), restricted activity days (RADs), and respiratory-related RADs (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976 to 1981. Ostro reported that two-week average $PM_{2.5}$ levels were significantly linked to WLDs, RADs, and RRADs; however, some year-to-year variability occurred in the results. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were then pooled. For the coefficient in the C-R function, we use an inverse-variance weighted average of the coefficients in Ostro (1987, Table IV).

3. Results

This analysis estimates the health effects attributable to the SO_2 and NO_x emissions from 41 major power plants in 2001. The estimated health effects from these 41 power plants include:

- Premature Mortality (2 estimates, and 2 cause-specific estimates)
- Chronic Bronchitis
- Hospital Admissions for COPD, Pneumonia and Asthma
- Emergency Room Visits for Asthma
- Daily Illness for Acute Bronchitis, Upper Respiratory Disease and Lower Respiratory Disease
- Daily Incidence of Work Loss Days, Minor Restricted Activity Days and Asthma Attacks

As described above, these 41 power plants were selected because they emitted at least 40,000 tons of SO_2 emissions in 2001, and had SO_2 emissions either increase or decline by less than half the national average between 1990 and 2001. The 41 major power plants include:

- Barry AL
- Belews Creek NC
- Big Brown TX
- Big Cajun 2 LA
- Big Sandy KY
- Brandon Shores MD
- Bull_Run TN
- Centralia WA
- Chesterfield VA
- Cheswick PA
- Conesville OH
- Crystal River FL
- ED Edwards IL
- EW Brown KY
- FJ Gannon FL
- Fort Martin WV
- Greene County AL
- Harding St Station IN
- Hatfields Ferry PA
- Homer City PA
- Jeffrey EC KS
- JH Miller AL
- JH Campbell MI
- John E Amos WV
- Johnsonville TN
- Keystone PA
- Kingston TN
- Leland Olds ND
- Marshall NC
- Martin Lake TX
- Mohave NV
- Monticello TX
- Morgantown MD
- Pleasants WV
- R Gallagher IN
- Rockport IN
- Roxboro NC
- Scherer GA
- WA Parish TX
- Warrick IN
- Widows Creek AL

Exhibit 3.1 shows the state level SO_2 emissions in 1990 and 2001from all Title IV-affected power plants. The SO_2 emissions decreased in some states during this time, such as Ohio, while they increased in other states like Texas. Exhibit 3.2 provides a different perspective on the 1990/2001 SO_2 emissions data, giving 2001 emissions as a percentage of 1990 emissions. The percentage is greater than 100% for states in which emissions increased (such as Virginia, with 2001 SO_2 emissions 137% of 1990 emissions), while states that decreased have a percentage less than 100%. Total national SO_2 emissions decreased over this period, with 2001 national SO_2 emissions equal to 67.6% of 1990 national SO_2 emissions.

The combination of Exhibits 3.1 and 3.2 offers a more complete picture of the change in SO_2 emissions between 1990 and 2001. For example, while Ohio has the highest amount of SO_2 emissions of any state in both 1990 and 2001, it also had one of the largest percentage decreases over that time period, with 2001 emissions only 51% of Ohio's 1990 emissions. Other states, such as Texas, increased their SO_2 emissions over that period. For instance, Texas' SO_2 emissions increased by 17%, and Texas went from the twelfth largest SO_2 emitting state in 1990 to the fifth largest in 2001. Exhibit 3.3 provides the state emissions data.

Exhibit 3.1. State Level SO_2 Emissions in 1990 and 2001 from all Title IV Affected Facilities

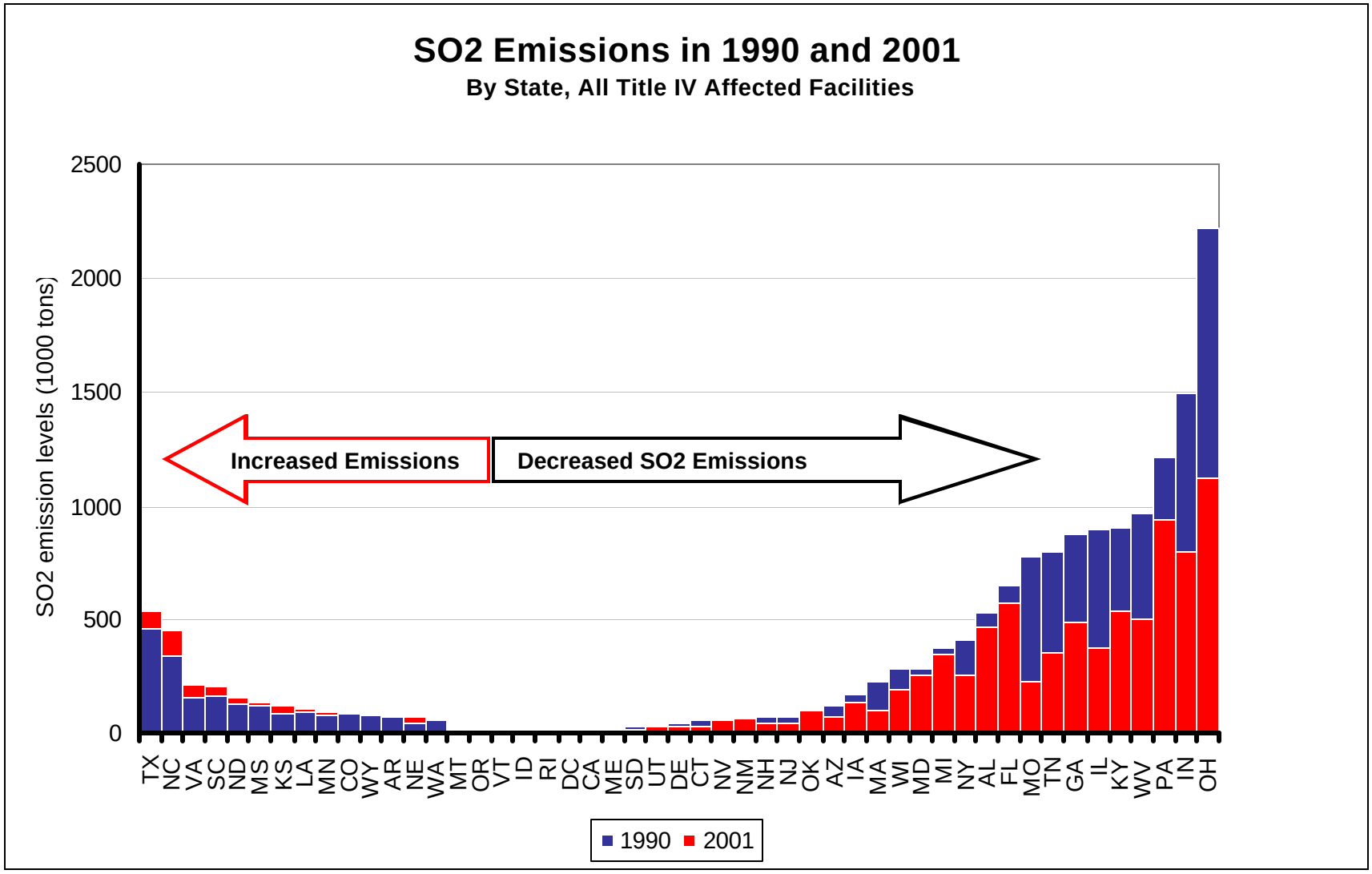

Exhibit 3.2 Relative Change Between 1990 and 2001 In State SO_2 Emissions From All Title IV Facilities

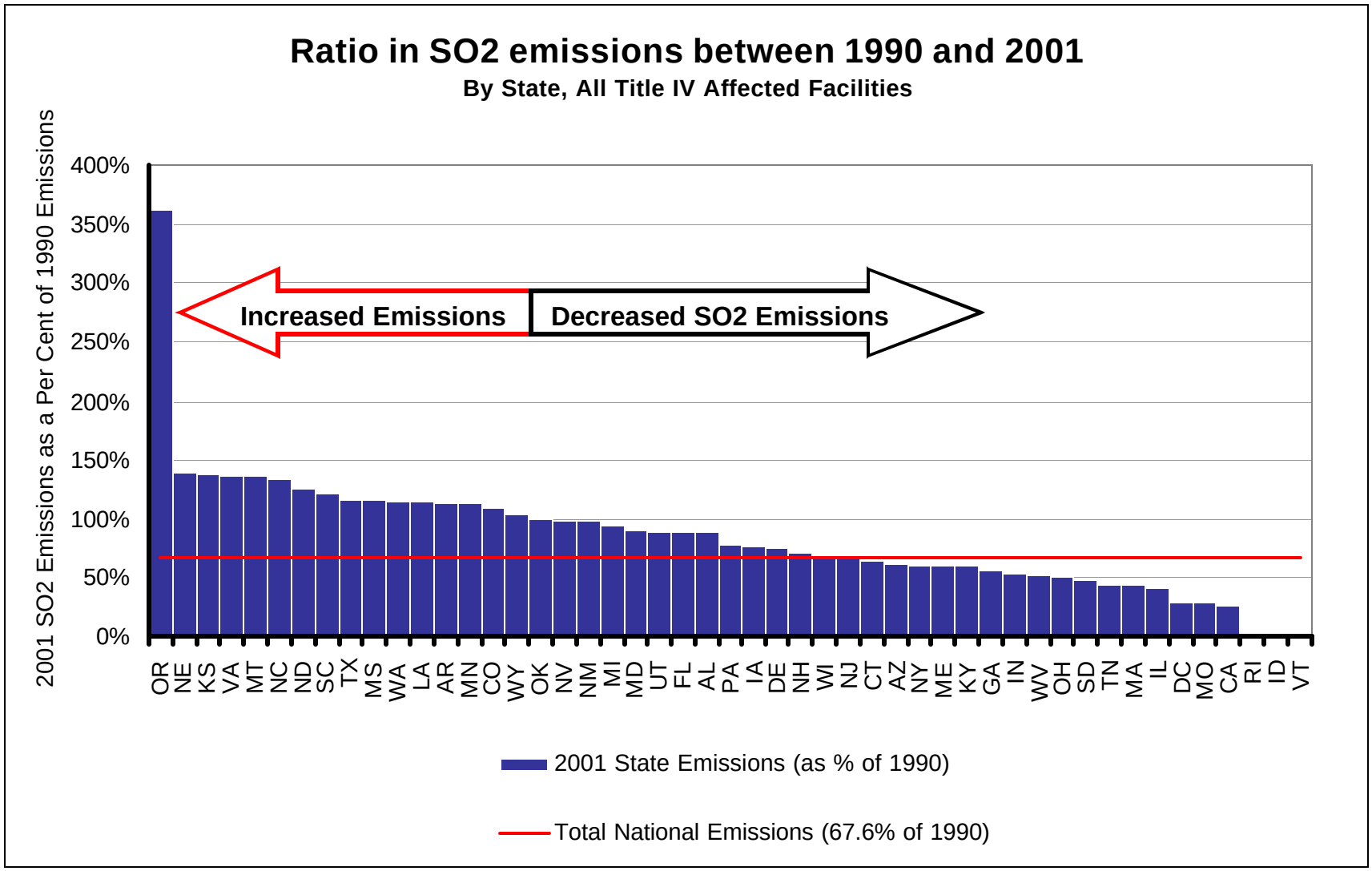

Exhibit 3.3 State SO_2 and NOx Emissions Data 1990 and 2001

State	SO_2 1990 (Tons)	SO_2 2001 (Tons)	Change	Ratio '01 to '90		NOx 2001 (Tons)
Oregon	4,936	17,837	12,901	361.4%		11,150
Nebraska	50,378	70,251	19,873	139.4%		47,909
Kansas	87,676	120,307	32,631	137.2%		84,705
Virginia	158,626	217,435	58,809	137.1%		80,529
Montana	17,922	24,403	6,481	136.2%	↑	39,435
North Carolina	336,451	450,486	114,035	133.9%		144,336
North Dakota	123,464	154,934	31,470	125.5%		79,114
South Carolina	167,414	202,501	35,087	121.0%		81,692
Texas	462,345	541,263	78,918	117.1%		310,871
Mississippi	119,071	138,358	19,287	116.2%		57,811
Washington	58,434	66,912	8,478	114.5%		18,333
Louisiana	98,703	112,804	14,101	114.3%		81,289
Arkansas	69,160	78,705	9,545	113.8%		47,558
Minnesota	81,166	91,768	10,602	113.1%		81,083
Colorado	83,186	90,388	7,202	108.7%		71,728
Wyoming	80,877	84,471	3,594	104.4%	Increased Emissions	84,321
Oklahoma	101,852	101,447	-405	99.6%		84,320
Nevada	55,780	54,703	-1,077	98.1%		44,262
New Mexico	63,839	62,198	-1,641	97.4%		83,340
Michigan	369,845	347,070	-22,775	93.8%		140,951
Maryland	282,453	254,482	-27,971	90.1%		72,935
Utah	32,051	28,321	-3,730	88.4%		71,517
Florida	645,131	569,153	-75,978	88.2%		290,843
Alabama	528,627	466,113	-62,514	88.2%		167,497
Pennsylvania	1,213,385	944,877	-268,508	77.9%		220,048
Iowa	173,033	133,562	-39,471	77.2%		78,478
Delaware	46,918	35,411	-11,507	75.5%		13,938
New Hampshire	67,863	48,124	-19,739	70.9%		6,836
Wisconsin	282,243	189,374	-92,869	67.1%		101,169
New Jersey	74,979	50,270	-24,709	67.0%	↓ Decreased Emissions	35,320
Connecticut	52,408	34,117	-18,291	65.1%		13,060
Arizona	119,898	73,329	-46,569	61.2%		93,097
New York	414,789	250,715	-164,074	60.4%		93,181
Maine	11,330	6,817	-4,513	60.2%		2,085
Kentucky	905,084	535,445	-369,639	59.2%		231,822
Georgia	874,630	489,626	-385,004	56.0%		162,379
Indiana	1,499,176	795,506	-703,670	53.1%		306,531
West Virginia	968,611	498,056	-470,555	51.4%		204,304
Ohio	2,211,628	1,125,475	-1,086,153	50.9%		332,903
South Dakota	28,906	13,619	-15,287	47.1%		16,539
Tennessee	796,528	356,608	-439,920	44.8%		154,962
Massachusetts	232,012	102,934	-129,078	44.4%		33,519
Illinois	893,793	368,218	-525,575	41.2%		199,860
Missouri	775,726	231,562	-544,164	29.9%		144,742
Washington, DC	2,523	754	-1,769	29.9%		429
California	7,365	1,886	-5,479	25.6%		18,837
Rhode Island	1,090	9	-1,081	0.8%		752
Idaho	0	4	4			198
Vermont	0	5	5			229
Total	15,733,305	10,632,613	-5,100,692	67.6%		4,742,747

Emission Data and Results for 41 Modeled Plants

The 2001 SO_2 and NO_x emissions data for all 41 modeled power plants are presented in Exhibits 3.4 through 3.6.

The attributable health effects of emissions from the 41 individual facilities are presented in Exhibit 3.7 (Premature Mortality and Chronic Bronchitis), Exhibit 3.8 (Hospital Admissions) and Exhibit 3.9 (Daily Illness).

Exhibit 3.4 SO_2 Emissions for 41 Modeled Power Plants

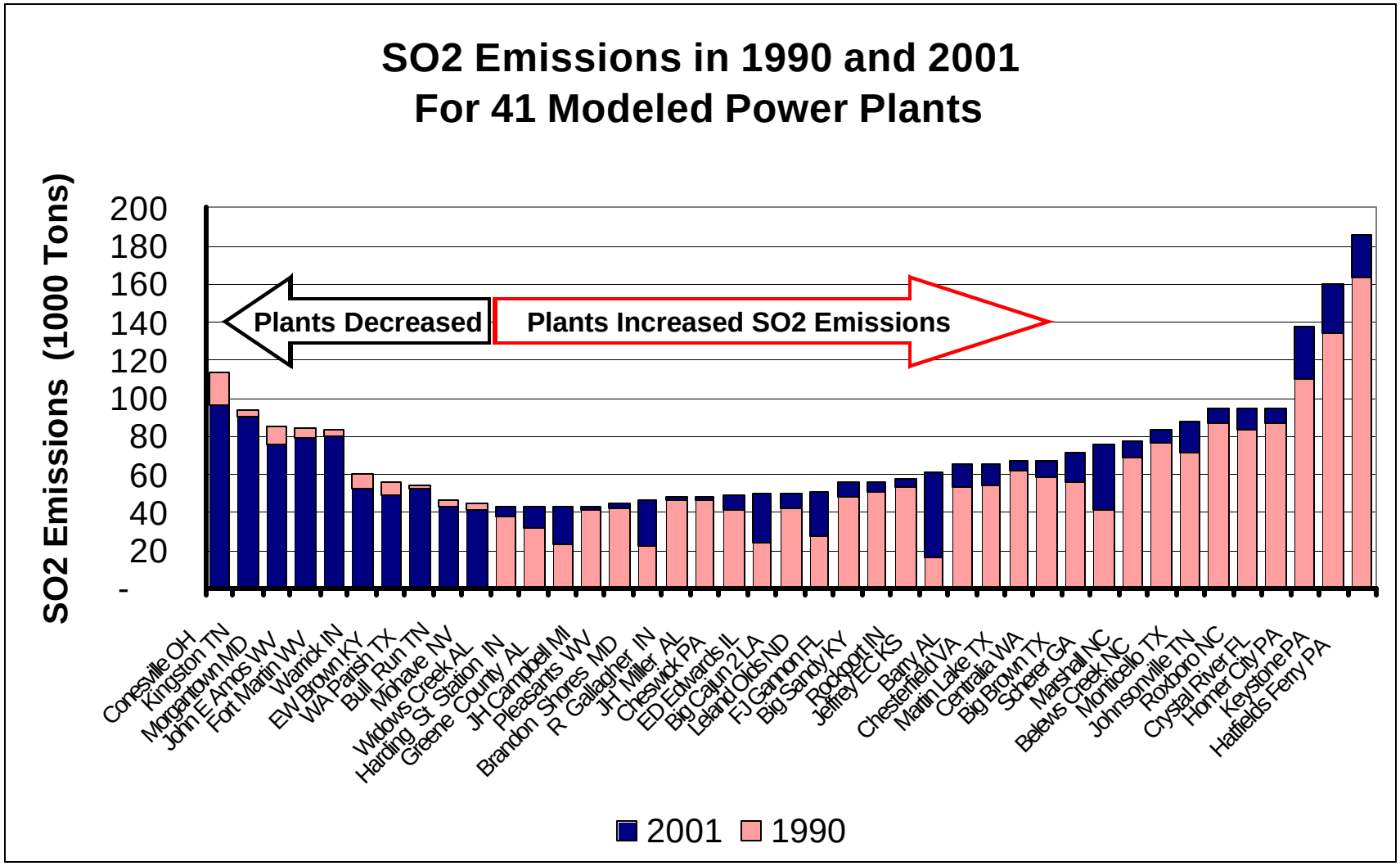

Exhibit 3.5 Percent Change in SO_2 Emissions for 41 Modeled Power Plants

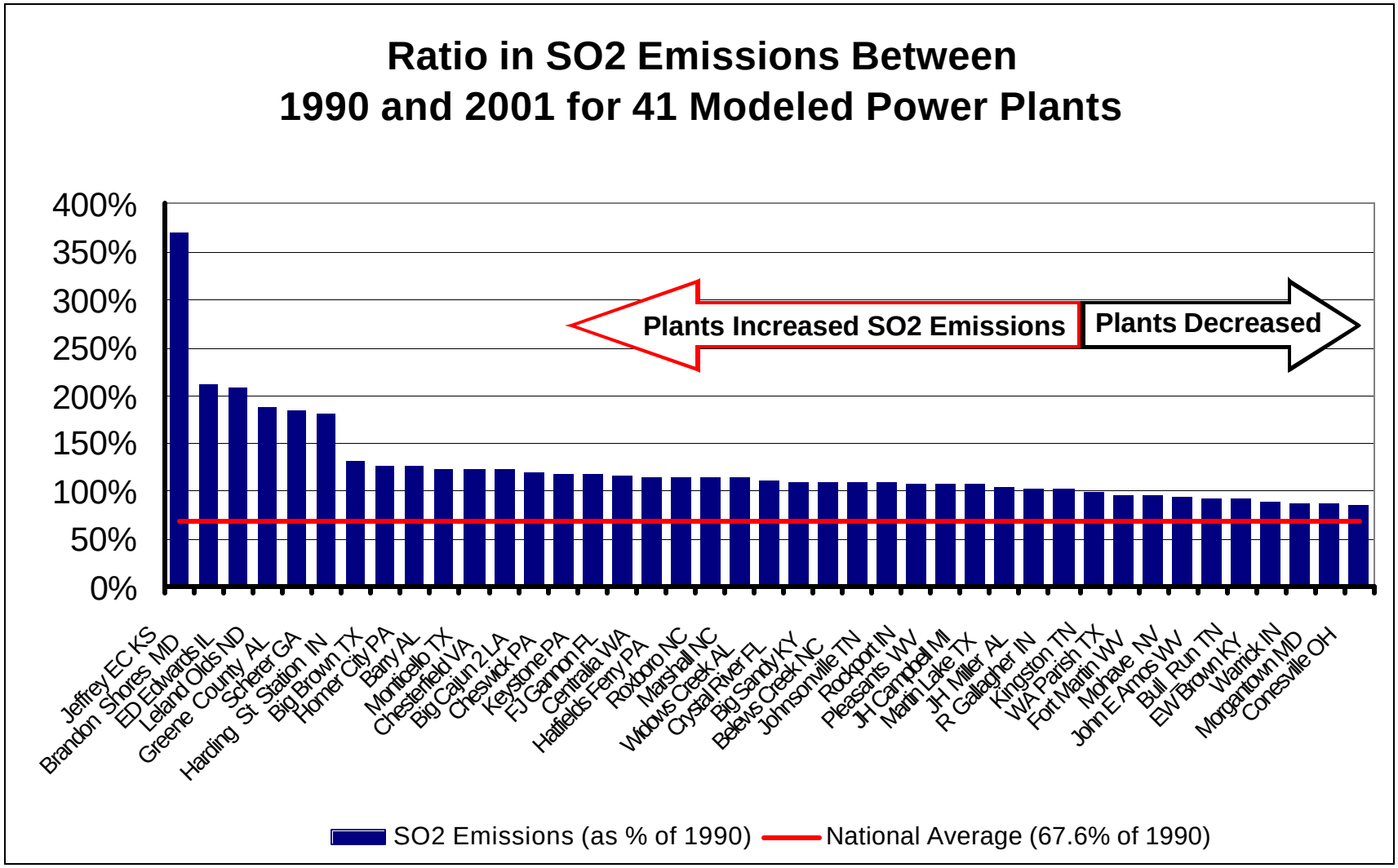

Exhibit 3.6 Emissions From 41 Modeled Facilities

	SO_2 Emissions (Tons)			NO_x Emissions (Tons)
	1990	2001	Ratio, 2001/1990	2001
Emissions from 41 Modeled Facilities				
Barry AL	53,378	65,902	123.5%	23,026
Belews Creek NC	76,251	83,077	109.0%	34,203
Big Brown TX	55,278	70,594	127.7%	12,520
Big Cajun 2 LA	41,930	50,217	119.8%	21,158
Big Sandy KY	51,157	55,846	109.2%	19,899
Brandon Shores MD	22,077	46,766	211.8%	13,746
Bull_Run TN	47,146	43,049	91.3%	17,319
Centralia WA	58,434	66,906	114.5%	18,244
Chesterfield VA	53,920	65,995	122.4%	16,418
Cheswick PA	41,279	49,002	118.7%	6,749
Conesville OH	113,635	96,738	85.1%	22,092
Crystal River FL	86,824	94,851	109.2%	34,712
ED Edwards IL	24,082	50,126	208.1%	9,612
EW Brown KY	55,104	49,106	89.1%	7,800
FJ Gannon FL	47,803	55,036	115.1%	25,332
Fort Martin WV	83,534	79,661	95.4%	12,334
Greene County AL	23,390	43,115	184.3%	11,536
Harding St Station IN	32,735	43,053	131.5%	6,724
Hatfields Ferry PA	163,432	185,496	113.5%	27,402
Homer City PA	109,449	137,573	125.7%	24,991
Jeffrey EC KS	16,528	60,924	368.6%	14,722
JH Miller AL	46,323	47,615	102.8%	29,944
JH Campbell MI	41,002	43,513	106.1%	30,887
John E Amos WV	84,943	78,851	92.8%	36,368
Johnsonville TN	86,688	94,199	108.7%	20,394
Keystone PA	134,848	159,725	118.4%	19,239
Kingston TN	92,821	90,291	97.3%	26,166
Leland Olds ND	27,364	51,456	188.0%	14,780
Marshall NC	68,628	77,291	112.6%	20,124
Martin Lake TX	62,392	66,134	106.0%	22,080
Mohave NV	44,372	41,299	93.1%	19,430
Monticello TX	71,056	87,263	122.8%	19,106
Morgantown MD	85,290	75,335	88.3%	16,760
Pleasants WV	42,066	44,815	106.5%	14,562
R Gallagher IN	46,458	47,511	102.3%	6,663
Rockport IN	53,755	57,365	106.7%	34,997
Roxboro NC	83,332	94,206	113.0%	25,448
Scherer GA	41,417	75,423	182.1%	31,274
WA Parish TX	54,528	52,534	96.3%	14,079
Warrick IN	59,697	52,777	88.4%	17,228
Widows Creek AL	38,291	42,788	111.7%	26,242
Total			113.2%	842,672

Exhibit 3.7 Premature Mortality and Chronic Bronchitis

	Mortality		Cause-Specific Mortality (Pope 02)		Chronic Disease
	Pope et al. '02	Krewski et. al. '00	Cancer	Cardio-Pulmonary	Chronic Bronchitis
Health Effects for 41 Individual Major Power Plants					
Barry AL	60	69	9	43	40
Belews Creek NC	149	171	21	106	107
Big Brown TX	71	82	10	51	52
Big Cajun 2 LA	43	50	6	31	29
Big Sandy KY	102	118	14	74	70
Brandon Shores MD	86	99	12	62	62
Bull Run TN	73	85	10	53	51
Centralia WA	29	34	4	20	26
Chesterfield VA	132	152	18	93	97
Cheswick PA	99	114	13	72	67
Conesville OH	201	232	27	145	138
Crystal River FL	107	123	16	78	69
ED Edwards IL	103	118	14	73	70
EW Brown KY	90	103	13	65	60
FJ Gannon FL	95	110	16	79	62
Fort Martin WV	155	179	21	112	106
Greene County AL	52	60	7	37	34
Harding St Station IN	101	116	14	72	70
Hatfields Ferry PA	360	415	48	259	246
Homer City PA	269	310	36	195	187
Jeffrey EC KS	41	47	5	29	28
JH Miller AL	68	79	10	49	48
JH Campbell MI	91	105	12	66	63
John E Amos WV	146	168	20	105	103
Johnsonville TN	155	179	22	112	110
Keystone PA	302	348	40	218	208
Kingston TN	149	171	21	107	127
Leland Olds ND	101	116	14	73	69
Marshall NC	139	161	19	100	118
Martin Lake TX	68	79	9	49	49
Mohave NV	41	47	5	29	35
Monticello TX	84	96	11	60	58
Morgantown MD	130	149	18	93	91
Pleasants WV	85	98	11	61	59
R Gallagher IN	101	116	14	73	68
Rockport IN	112	129	15	80	75
Roxboro NC	153	176	21	110	115
Scherer GA	107	123	15	76	73
WA Parish TX	56	65	8	40	42
Warrick IN	155	179	21	112	103
Widows Creek AL	79	91	11	57	56
Total	4,844	5,576	666	3,496	3,412

Exhibit 3.8 Hospital Admissions and Emergency Room Visits

	Hospital Admissions			ER Visits
	COPD	Pneumonia	Asthma	Asthma
Health Effects for 41 Individual Major Power Plants				
Barry AL	7	8	5	15
Belews Creek NC	19	23	14	40
Big Brown TX	9	11	7	21
Big Cajun 2 LA	5	6	4	11
Big Sandy KY	13	15	9	26
Brandon Shores MD	11	14	8	23
Bull Run TN	9	11	7	19
Centralia WA	4	5	3	10
Chesterfield VA	17	20	13	36
Cheswick PA	12	15	9	25
Conesville OH	25	31	18	51
Crystal River FL	14	17	8	24
ED Edwards IL	12	15	10	27
EW Brown KY	11	13	8	23
FJ Gannon FL	12	14	7	21
Fort Martin WV	19	24	14	39
Greene County AL	6	7	5	13
Harding St Station IN	12	15	9	27
Hatfields Ferry PA	45	55	33	90
Homer City PA	34	42	24	68
Jeffrey EC KS	5	6	4	11
JH Miller AL	8	10	6	18
JH Campbell MI	11	14	8	24
John E Amos WV	18	22	13	38
Johnsonville TN	19	24	14	42
Keystone PA	38	46	27	76
Kingston TN	22	27	14	48
Leland Olds ND	12	15	9	26
Marshall NC	21	25	13	44
Martin Lake TX	9	11	6	19
Mohave NV	6	7	5	14
Monticello TX	10	13	8	23
Morgantown MD	16	20	12	34
Pleasants WV	11	13	8	22
R Gallagher IN	12	15	9	26
Rockport IN	13	16	10	28
Roxboro NC	20	25	14	43
Scherer GA	13	15	10	28
WA Parish TX	7	8	6	18
Warrick IN	18	22	14	39
Widows Creek AL	10	12	7	21
Total		743		1,276

Exhibit 3.9 Daily Illness

	Acute Bronchitis	Lower Resp. Disease	Upper Resp. Disease	Minor Restricted Activity	Work Loss Days	Asthma Attack
Health Effects for Individual Major Power Plants						
Barry AL	137	1,532	1,518	58,783	11,066	1,340
Belews Creek NC	348	3,877	3,846	156,721	29,503	3,458
Big Brown TX	183	2,043	2,064	77,576	14,604	1,761
Big Cajun 2 LA	105	1,175	1,151	44,183	8,317	979
Big Sandy KY	229	2,555	2,509	103,735	19,528	2,260
Brandon Shores MD	201	2,243	2,244	89,737	16,893	2,001
Bull_Run TN	165	1,845	1,819	75,138	14,144	1,643
Centralia WA	81	906	968	36,808	6,930	855
Chesterfield VA	319	3,557	3,544	142,498	26,827	3,136
Cheswick PA	219	2,447	2,388	98,403	18,524	2,149
Conesville OH	452	5,039	4,953	201,150	37,868	4,424
Crystal River FL	208	2,317	2,291	92,956	17,500	2,282
ED Edwards IL	240	2,678	2,621	104,445	19,661	2,325
EW Brown KY	200	2,235	2,166	90,984	17,127	1,951
FJ Gannon FL	180	2,008	1,969	81,859	15,416	2,019
Fort Martin WV	350	3,900	3,783	157,013	29,558	3,395
Greene County AL	117	1,302	1,271	51,309	9,659	1,140
Harding St Station IN	233	2,600	2,587	102,686	19,331	2,292
Hatfields Ferry PA	811	9,053	8,758	364,371	68,601	7,856
Homer City PA	609	6,792	6,656	272,682	51,337	5,962
Jeffrey EC KS	98	1,098	1,078	41,798	7,868	931
JH Miller AL	156	1,741	1,771	69,042	12,997	1,581
JH Campbell MI	213	2,373	2,341	92,513	17,415	2,054
John E Amos WV	331	3,688	3,671	149,211	28,089	3,295
Johnsonville TN	351	3,913	4,049	155,725	29,315	3,613
Keystone PA	684	7,632	7,446	306,131	57,634	6,663
Kingston TN	337	3,757	4,568	152,599	28,727	4,128
Leland Olds ND	240	2,678	2,622	103,083	19,405	2,301
Marshall NC	325	3,626	4,266	147,318	27,734	3,852
Martin Lake TX	10	1,815	1,893	69,281	13,042	1,634
Mohave NV	123	1,376	1,502	49,214	9,264	1,214
Monticello TX	200	2,236	2,273	85,172	16,033	1,959
Morgantown MD	301	3,360	3,301	134,227	25,269	2,938
Pleasants WV	191	2,131	2,113	86,024	16,194	1,897
R Gallagher IN	230	2,563	2,492	101,814	19,166	2,222
Rockport IN	255	2,849	2,766	111,995	21,083	2,450
Roxboro NC	360	4,011	4,167	161,809	30,462	3,737
Scherer GA	252	2,809	2,750	111,153	20,924	2,414
WA Parish TX	164	1,830	1,782	67,905	12,783	1,474
Warrick IN	355	3,956	3,808	155,336	29,242	3,374
Widows Creek AL	179	1,992	2,023	80,335	15,123	1,818
Total	10,979	124,198	124,375	4,940,516	930,081	111,071

References

Abbey, D.E., F. Petersen, P.K. Mills and W.L. Beeson. 1993. Long-Term Ambient Concentrations of Total Suspended Particulates, Ozone, and Sulfur Dioxide and Respiratory Symptoms in a Nonsmoking Population. Archives of Environmental Health. Vol. 48(1): 33-46.

Abbey, D.E., B.L. Hwang, R.J. Burchette, T. Vancuren and P.K. Mills. 1995a. Estimated Long-Term Ambient Concentrations of Pm(10) and Development of Respiratory Symptoms in a Nonsmoking Population. Archives of Environmental Health. Vol. 50(2): 139-152.

Abbey, D.E., B.E. Ostro, F. Petersen and R.J. Burchette. 1995b. Chronic Respiratory Symptoms Associated with Estimated Long-Term Ambient Concentrations of Fine Particulates Less Than 2.5 Microns in Aerodynamic Diameter (PM2.5) and Other Air Pollutants. J Expo Anal Environ Epidemiol. Vol. 5(2): 137-159.

Abbey, D.E., R.J. Burchette, S.F. Knutsen, W.F. McDonnell, M.D. Lebowitz and P.L. Enright. 1998. Long-term particulate and other air pollutants and lung function in nonsmokers. American Journal of Respiratory and Critical Care Medicine. Vol. 158(1): 289-298.

Abt Associates Inc. 2000. The Particulate-Related Health Benefits of Reducing Power Plant Emissions. Prepared for Clean Air Task Force, Boston, MA. Bethesda, MD. October.

Ackermann-Liebrich, U., P. Leuenberger, J. Schwartz, C. Schindler, C. Monn, C. Bolognini, J.P. Bongard, O. Brandli, G. Domenighetti, S. Elsasser, L. Grize, W. Karrer, R. Keller, H. KellerWossidlo, N. Kunzli, B.W. Martin, T.C. Medici, A.P. Perruchoud, M.H. Schoni, J.M. Tschopp, B. Villiger, B. Wuthrich, J.P. Zellweger and E. Zemp. 1997. Lung function and long term exposure to air pollutants in Switzerland. Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) Team. Am J Respir Crit Care Med. Vol. 155(1): 122-129.

Adams, P.F. and M.A. Marano. 1995. Current Estimates from the National Health Interview Survey, 1994. National Center for Health Statistics. Hyattsville, MD. Vital Health Statistics, Series 10, No. 193. December.

Collins, J.G. 1997. Prevalence of Selected Chronic Conditions: United States 1990-1992. National Center for Health Statistics. Hyattsville, MD. Vital Health Statistics, Series 10, No. 194.

Detels, R., D.P. Tashkin, J.W. Sayre, S.N. Rokaw, F.J. Massey, A.H. Coulson and D.H. Wegman. 1991. The UCLA Population Studies of Cord .10. a Cohort Study of Changes in Respiratory Function Associated With Chronic Exposure to Sox, Nox, and Hydrocarbons. American Journal of Public Health. Vol. 81(3): 350-359.

Dockery, D.W., F.E. Speizer, D.O. Stram, J.H. Ware, J.D. Spengler and B.G. Ferris, Jr. 1989. Effects of Inhalable Particles on Respiratory Health of Children. Am Rev Respir Dis. Vol. 139: 587-594.

Dockery, D.W., J. Cunningham, A.I. Damokosh, L.M. Neas, J.D. Spengler, P. Koutrakis, J.H. Ware, M. Raizenne and F.E. Speizer. 1996. Health Effects of Acid Aerosols On North American Children - Respiratory Symptoms. Environmental Health Perspectives. Vol. 104(5): 500-505.

E.H. Pechan & Associates Inc. 1995. Updates to Fugitive Emission Components of the National Particulate Inventory. William Kuykendal, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Innovative Strategies and Economics Group. July 17.

E.H. Pechan & Associates Inc. 1997. Control Measure Analysis of Ozone and PM Alternatives -- Methodologies and Results, Final Technical Support Document. Prepared for U.S. EPA, Office of Air Quality Planning and Standards, Innovative Strategies and Economics Group. Research Triangle Park, NC. July 17.

E.H. Pechan & Associates Inc. 1996. Regional Particulate Control Strategies Phase II. Prepared for the U.S. Environmental Protection Agency, Office of Policy, Planning, and Evaluation. Washington, DC. Pechan Report No. 96.09.006/1776. September.

E.H. Pechan & Associates Inc. 1997a. Control Measure Analysis of Ozone and PM Alternatives -- Methodologies and Results. Prepared for U.S. EPA, Office of Air Quality Planning and Standards, Innovative Strategies and Economics Group. Research Triangle Park, NC. July 17.

E.H. Pechan & Associates Inc. 1997b. Ozone Transport Assessment Group (OTAG) Emissions Inventory Development Report, Volume III: Projections and Controls. Draft report prepared for U.S. EPA, Office of Air Quality Planning and Standards, Innovative Strategies and Economics Group. Research Triangle Park, NC. July 17.

E.H. Pechan & Associates Inc. 1999. Procedures for Developing Base Year and Future Year Mass and Modeling Inventories for the Tier 2 Final Rulemaking. Prepared for U.S. EPA, Office of Air Quality Planning and Standards. Springfield, VA. September.

E.H. Pechan & Associates Inc. 2000. Analysis of Air Quality Impacts of Limiting Emissions from Electricity Generating Units: Draft Report. Prepared for Abt Associates Inc. Springfield, VA. September.

Empire State Electric Energy Research Corporation (ESEERCO). 1994. New York State Environmental Externalities Cost Study. Report 2: Methodology. Prepared by RCG/Hagler, Bailly, Inc. November.

Graves, E.J. and B.S. Gillum. 1997. Detailed Diagnoses and Procedures, National Hospital Discharge Survey, 1994. National Center for Health Statistics. Hyattsville, MD. Vital Health Statistics, Series 13, No. 127. March.

Krewski, D., R. Burnett, M. Goldberg, K. Hoover, J. Siemiatycki, M. Jerrett, M. Abrahamowicz and M. White. 2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Health Effects Institute. Cambridge. July.

Krupnick, A.J. 1988. An Analysis of Selected Health Benefits from Reductions in Photochemical Oxidants in the Northeastern United States: Final Report. Prepared for U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Washington, DC. EPA Contract No. 68-02-4323. September.

Lipfert, F.W. 1993. A critical review of studies of the association between demands for hospital services and air pollution. Environ Health Perspect. Vol. 101 Suppl 2: 229-68.

McDonnell, W.F., D.E. Abbey, N. Nishino and M.D. Lebowitz. 1999. Long-term ambient ozone concentration and the incidence of asthma in nonsmoking adults: the AHSMOG study. Environ Res. Vol. 80(2 Pt 1): 110-21.

Medical Center Information Systems: Duke University Health Systems ICD-9-CM.; November 5: http://dumccss.mc.duke.edu/standards/termcode/icd9/index.html, 1999.

Mood, A.M., F.A. Graybill and D.C. Boes. 1974. Introduction to the Theory of Statistics. 3rd ed. McGraw Hill Book Company: New York.

National Heart, L., and Blood Institute. 1997. Guidelines for the Diagnosis and Management of Asthma: Expert Panel Report 2. National Institutes of Health. Bethesda, MD. NIH Publication No. 97-4051. July.

Neas, L.M., D.W. Dockery, J.H. Ware, J.D. Spengler, B.G. Ferris and F.E. Speizer. 1994. Concentration of Indoor Particulate Matter As a Determinant of Respiratory Health in Children. American Journal of Epidemiology. Vol. 139(11): 1088-1099.

Ostro, B.D. 1987. Air Pollution and Morbidity Revisited: A Specification Test. Journal of Environmental Economics and Management. Vol. 14: 87-98.

Ostro, B.D. and S. Rothschild. 1989. Air Pollution and Acute Respiratory Morbidity - an Observational Study of Multiple Pollutants. Environ Res. Vol. 50(2): 238-247.

Pope, C.A., D.W. Dockery, J.D. Spengler and M.E. Raizenne. 1991. Respiratory Health and Pm10 Pollution - a Daily Time Series Analysis. American Review of Respiratory Disease. Vol. 144(3): 668-674.

Pope, C.A., J. Schwartz and M.R. Ransom. 1992. Daily Mortality and PM10 Pollution in Utah Valley. Archives of Environmental Health. Vol. 47(3): 211-217.

Pope, C.A., M.J. Thun, M.M. Namboodiri, D.W. Dockery, J.S. Evans, F.E. Speizer and C.W. Heath. 1995. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Respir Crit Care Med. Vol. 151(3): 669-674.

Pope, C.A., 3rd, R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito and G.D. Thurston. 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. Jama. Vol. 287(9): 1132-41.

Richards, W., S.P. Azen, J. Weiss, S. Stocking and J. Church. 1981. Los Angeles air pollution and asthma in children. Ann Allergy. Vol. 47(5 Pt 1): 348-54.

Rossi, G., M.A. Vigotti, A. Zanobetti, F. Repetto, V. Gianelle and J. Schwartz. 1999. Air pollution and cause-specific mortality in Milan, Italy, 1980-1989. Arch Environ Health. Vol. 54(3): 158-64.

Samet, J., S. Zeger, F. Dominici, F. Curriero, I. Coursac, D. Dockery, J. Schwartz and A. Zanobetti. 2000. The National Morbidity, Mortality, and Air Pollution Study. Health Effects Institute. Cambridge, MA. Report No. 94. May.

Schwartz, J. 1993. Particulate Air Pollution and Chronic Respiratory Disease. Environ Res. Vol. 62: 7-13.

Schwartz, J., D. Slater, T.V. Larson, W.E. Pierson and J.Q. Koenig. 1993. Particulate air pollution and hospital emergency room visits for asthma in Seattle. Am Rev Respir Dis. Vol. 147(4): 826-31.

Schwartz, J. 1994a. Air Pollution and Hospital Admissions For the Elderly in Detroit, Michigan. American Journal of Respiratory and Critical Care Medicine. Vol. 150(3): 648-655.

Schwartz, J. 1994b. Air Pollution and Hospital Admissions For the Elderly in Birmingham, Alabama. American Journal of Epidemiology. Vol. 139(6): 589-598.

Schwartz, J. 1994c. What Are People Dying of On High Air Pollution Days. Environmental Research. Vol. 64(1): 26-35.

Schwartz, J., D.W. Dockery, L.M. Neas, D. Wypij, J.H. Ware, J.D. Spengler, P. Koutrakis, F.E. Speizer and B.G. Ferris. 1994. Acute Effects of Summer Air Pollution On Respiratory Symptom Reporting in Children. Am J Respir Crit Care Med. Vol. 150(5): 1234-1242.

Schwartz, J. 2000. Harvesting and long term exposure effects in the relation between air pollution and mortality [see comments]. Am J Epidemiol. Vol. 151(5): 440-8.

Schwartz, J. 2001. Is there harvesting in the association of airborne particles with daily deaths and hospital admissions? Epidemiology. Vol. 12(1): 55-61.

Sheppard, L., D. Levy, G. Norris, T.V. Larson and J.Q. Koenig. 1999. Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington, 1987-1994. Epidemiology. Vol. 10(1): 23-30.

Smith, D.H., D.C. Malone, K.A. Lawson, L.J. Okamoto, C. Battista and W.B. Saunders. 1997. A national estimate of the economic costs of asthma. Am J Respir Crit Care Med. Vol. 156(3 Pt 1): 787-93.

Spix, C., J. Heinrich, D. Dockery, J. Schwartz, G. Volksch, K. Schwinkowski, C. Collen and H.E. Wichmann. 1993. Air pollution and daily mortality in Erfurt, east Germany, 1980-1989. Environ Health Perspect. Vol. 101(6): 518-26.

Stella, G. 1999. U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Emission Factors and Inventory Group. Personal communication with Erica Laich, E.H. Pechan & Associates, Inc. Email providing information on monthly percentage profiles by state, prime mover, and fuel use in developing June and August daily heat input and emissions to EGU inventory. 21, J.

Turner, D.B. 1979. Workbook of Atmospheric Dispersion Estimates, AP-26. U.S. EPA, Office of Air Programs. Research Triangle Park, NC.

U.S. Bureau of the Census. 1997. Statistical Abstract of the United States: 1997. 117 ed. Washington, DC.

U.S. Bureau of the Census. 1998. Statistical Abstract of the United States: 1998. 118 ed. Washington, DC.

U.S. Centers for Disease Control. 1999. CDC Wonder.

U.S. EPA. 1986. Review of the National Ambient Air Quality Standards for Particulate Matter: Updated Assessment of Scientific and Technical Information Addendum to the 1982 OAQPS Staff Paper. U.S. EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC. EPA 450/05-86-012.

U.S. EPA. 1997. Regulatory Impact Analyses for the Particulate Matter and Ozone National Ambient Air Quality Standards and Proposed Regional Haze Rule. U.S. EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC. July.

U.S. EPA. 1998a. Regulatory Impact Analysis for the NOx SIP Call, FIP, and Section 126 Petitions. U.S. EPA, Office of Air and Radiation. Washington, DC. EPA-452/R-98-003. December.

U.S. EPA. 1998b. Analyzing Electric Power Generation Under the CAAA. U.S. EPA, Office of Air and Radiation. March.

U.S. EPA. 1999. An SAB Advisory: The Clean Air Act Section 812 Prospective Study Health and Ecological Initial Studies. Prepared by the Health and Ecological Effects SubCommittee (HEES) of the Advisory Council on the Clean Air Compliance Analysis, Science Advisory Board, U.S. Environmental Protection Agency. Washington, DC. EPA-SAB-Council-ADV-99-005. February.

Whittemore, A.S. and E.L. Korn. 1980. Asthma and Air Pollution in the Los Angeles Area. Am J Public Health. Vol. 70: 687-696.

Yamartino, R.J. 1985. Atmospheric Pollutant Deposition Modeling. In: Handbook of Applied Meteorology. Houghton, D.D., Ed. John Wiley & Sons: New York.

Zeger, S.L., F. Dominici and J. Samet. 1999. Harvesting-resistant estimates of air pollution effects on mortality. Epidemiology. Vol. 10(2): 171-5.

Appendix A: Particulate Matter C-R Functions

Appendix A describes the concentration-response functions that we use in this analysis. Note that for all of the concentration-response functions we define ΔPM as $PM_{baseline}$ - $PM_{control}$, and we define the change in incidence as: - ($incidence_{control}$ - $incidence_{baseline}$). Also note that we base all of our estimates on annual PM levels. When estimating effects with C-R functions originally designed for daily changes in PM, we use the annual data with the assumption that all days in the year have the same concentration. Because the C-R functions are reasonably linear, whether one uses the mean annual change or the daily changes occurring through out the year, the results are essentially the same.

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.

Mortality (Pope et al., 2002) Based on ACS Cohort: Mean $PM_{2.5}$

The Pope et al. (2002) analysis is a longitudinal cohort tracking study that uses the same American Cancer Society (ACS) cohort as the original Pope et al. (1995) study, and the Krewski et al. (2000) reanalysis. Pope et al. (2002) analyzed survival data for the cohort from 1982 through 1998, 9 years longer than the original Pope study. Pope et al. (2002) 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) followed Krewski et al. (2000) and Pope et al. (1995, Table 2) and reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9

codes: 401-440 and 460-519), and "all other" deaths.[6] Like the earlier studies, Pope et al. (2002) found that mean $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. In addition, Pope et al. (2002) found a significant relationship with lung cancer mortality, which was not found in the earlier studies. None of the three studies found a significant relationship with "all other" deaths.

Pope et al. (2002) 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.

The C-R function to estimate the change in long-term mortality is:

$$\Delta Nonaccidental\ Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = county-level annual non-accidental death rate per person
β = $PM_{2.5}$ coefficient = 0.0040182 (all cause mortality, '79-'83 PM data)
$\Delta PM_{2.5}$ = change in annual <u>mean</u> $PM_{2.5}$ concentration
pop = population of ages 30 and older
σ_β = standard error of β = 0.0024338 (all cause mortality, '79-'83 PM data)

Incidence Rate. To estimate county-specific baseline mortality incidence among individuals ages 30 and over, this analysis used the average annual all-cause county mortality rate from 1996 through 1998 (U.S. Centers for Disease Control, 1999).

Coefficient Estimate (β). The coefficient (β) for $PM_{2.5}$ all-cause mortality using '79-'83 PM data is estimated from the relative risk (1.041) associated with a change in mean exposure of 10.0 $\mu g/m^3$. Exhibit E-1 summarizes the calculations we made.

$$b = \frac{\ln(1.041)}{(10.0)} = 0.0040182\,.$$

Standard Error (σ_β). The standard error (σ_β) for all cause mortality, '79-'83, was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk. Exhibit E-1 summarizes the calculations we made.

[6] 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.

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.19)}{24.5} - \frac{\ln(1.12)}{24.5}\right)}{1.96} = 0.0012625$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.12)}{24.5} - \frac{\ln(1.06)}{24.5}\right)}{1.96} = 0.0011466$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.0012046$$

Exhibit C-1 Estimated Coefficients and Standards Errors for Mortality

			Pollutant Measure (years)		
Mortality	**Measure**	**Statistic**	***$PM_{2.5}$ (79-83)***	***$PM_{2.5}$ (99-00)***	***$PM_{2.5}$ (ave)***
All Cause	Relative Risk	Mean	1.041	1.059	1.062
		High (97.5%)	1.075	1.099	1.110
		Low (2.5%)	1.008	1.020	1.016
	Coefficient (β)		0.0040182	0.0057325	0.0060154
	Standard Error (σ_β)	Mean	0.0016416	0.0019030	0.0022573
		High (97.5%)	0.0016397	0.0018916	0.0022554
		Low (2.5%)	0.0016436	0.0019144	0.0022592
Cardiopulmonary	Relative Risk	Mean	1.059	1.079	1.093
(ICD-9 code 401-440, 460-519)		High (97.5%)	1.105	1.140	1.135
		Low (2.5%)	1.015	1.023	1.005
	Coefficient (β)		0.0057325	0.0076035	0.0088926
	Standard Error (σ_β)	Mean	0.0021673	0.0027625	0.0031032
		High (97.5%)	0.0021694	0.0028058	0.0019238
		Low (2.5%)	0.0021651	0.0027191	0.0042826
Lung Cancer	Relative Risk	Mean	1.082	1.127	1.135
(ICD-9 code 162)		High (97.5%)	1.158	1.219	1.234
		Low (2.5%)	1.011	1.041	1.044
	Coefficient (β)		0.0078811	0.0119559	0.0126633
	Standard Error (σ_β)	Mean	0.0034631	0.0040268	0.0042653
		High (97.5%)	0.0034634	0.0040037	0.0042667
		Low (2.5%)	0.0034628	0.0040499	0.0042639

Source: Pope et al. (2002, Table 2). 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.

Mortality (Krewski et al., 2000) Based on ACS Cohort: Mean $PM_{2.5}$

The C-R function to estimate the change in long-term mortality is:

$$\Delta Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = county-level all-cause annual death rate per person ages 30 and older
β = $PM_{2.5}$ coefficient = 0.0046257
$\Delta PM_{2.5}$ = change in annual mean $PM_{2.5}$ concentration
pop = population of ages 30 and older
σ_β = standard error of β = 0.0012046

Incidence Rate. To estimate county-specific baseline mortality incidence among individuals ages 30 and over, this analysis used the average annual all-cause county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999). Note that the Krewski et al. (2000) replication of Pope et al. (1995) used the same all-cause mortality when estimating the impact of PM.

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.12) associated with a change in mean exposure of 24.5 $\mu g/m^3$ (based on the range from the original ACS study) (Krewski et al., 2000, Part II - Table 31).

$$b = \frac{\ln(1.12)}{(24.5)} = 0.0046257.$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Krewski et al., 2000, Part II - Table 31).

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.19)}{24.5} - \frac{\ln(1.12)}{24.5}\right)}{1.96} = 0.0012625$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.12)}{24.5} - \frac{\ln(1.06)}{24.5}\right)}{1.96} = 0.0011466$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.0012046$$

Chronic Morbidity

Schwartz (1993) and Abbey et al. (1993; 1995b) 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) 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 (Detels et al., 1991; Ackermann-Liebrich et al., 1997; Abbey et al., 1998).[7]

We estimate the changes in the new cases of chronic bronchitis by pooling the estimates from the studies by Schwartz (1993). The Schwartz study is somewhat older and uses a cross-sectional design, however, it is based on a national sample, unlike the Abbey et al. study which is based on a sample of California residents.

Chronic Bronchitis (Schwartz, 1993)

Schwartz (1993) 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, 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[8], 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.

Schwartz (1993) 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 PM, then baseline incidence drops by 25 percent with

---

[7] 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.

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

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 C-R function to estimate the change in chronic bronchitis is:

$$\Delta Chronic\,Bronchitis = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot b} + y_0} - y_0\right]\cdot\left[\frac{z_0}{y_0}\right]\cdot pop\,,$$

where:

y_0 = national chronic bronchitis prevalence rate for individuals 18 and older (Adams and Marano, 1995, Table 62 and 78) = 0.0535

z_0 = annual bronchitis incidence rate per person (Abbey et al., 1993, Table 3) = 0.00378

β = estimated PM_{10} logistic regression coefficient = 0.0123

ΔPM_{10} = change in annual average PM_{10} concentration

pop = population of ages 30 and older without chronic bronchitis = 0.9465*population 30+

σ_β = standard error of β = 0.00434 .

Prevalence Rate. The national chronic bronchitis prevalence rate was not available for individuals 30 and older. Instead, we used the prevalence rate for individuals 18 and older (Adams and Marano, 1995, Table 62 and 78). The 1994 national figures are the latest available, and are suggested here.

Incidence Rate. The annual incidence rate is derived by taking the number of new cases (234), dividing by the number of individuals in the sample (3,310), as reported by Abbey et al.(1993, Table 3), dividing by the ten years covered in the sample, and then multiplying by one minus the reversal rate (the percentage of reversals is estimated to be 46.6% based on Abbey et al. (1995a, Table 1)). Using the same data base, Abbey et al. (1995a, Table 1) reported the incidences by three age groups (25-54, 55-74, and 75+) for "cough type" and "sputum type" bronchitis, but they did not report an overall incidence rate for bronchitis.

Coefficient Estimate (β). The estimated logistic coefficient (β) is based on the odds ratio (= 1.07) associated with 10 $\mu g/m^3$ change in TSP (Schwartz, 1993, p. 9). Assuming that PM_{10} is 55 percent of TSP[9] and that particulates greater than ten micrometers are harmless, the coefficient is calculated as follows:

$$b_{PM_{10}} = \frac{\ln(1.07)}{0.55\cdot 10} = 0.0123.$$

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

Standard Error (σ_β). The standard error for the coefficient (σ_β) is calculated from the reported lower and upper bounds of the odds ratio (1.02 to 1.12) (Schwartz, 1993, p. 9):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.12)}{0.55 \cdot 10} - \frac{\ln(1.07)}{0.55 \cdot 10}\right)}{1.96} = 0.00424$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.07)}{0.55 \cdot 10} - \frac{\ln(1.02)}{0.55 \cdot 10}\right)}{1.96} = 0.00444$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.00434.$$

Population. The study population in Schwartz (1993) includes 3,874 individuals over the age of 30, living in 57 urban areas in the United States. To what extent the study should be applied to individuals under the age of 30 is unclear, and no effect is assumed for these individuals.

Hospital Admissions

There is a wealth of epidemiological information on the relationship between air pollution and hospital admissions for various respiratory and cardiovascular diseases; in addition, some studies have examined the relationship between air pollution and emergency room (ER) visits. Because most emergency room visits do not result in an admission to the hospital -- the majority of people going to the ER are treated and return home -- we treat hospital admissions and ER visits separately, taking account of the fraction of ER visits that do get admitted to the hospital, as discussed below.

Hospital admissions require the patient to be examined by a physician, and on average may represent more serious incidents than ER visits (Lipfert, 1993, p. 230). The two main groups of hospital admissions estimated in this analysis are respiratory admissions and cardiovascular admissions. There is not much evidence linking air pollution with other types of hospital admissions. The only types of ER visits that have been linked to air pollution in the U.S. or Canada are asthma-related visits.

Hospital Admissions for COPD (Samet et al., 2000, 14 Cities)

The C-R function to estimate the change in hospital admissions for COPD[10] associated with daily changes in PM_{10} is:

$$\Delta COPD\,Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0	= daily hospital admission rate for COPD per person 65 and older = 3.12 E-5
β	= PM_{10} coefficient = 0.00288
ΔPM_{10}	= change in daily average PM_{10} concentration
pop	= population age 65 and older
σ_β	= standard error of β = 0.00139

Incidence Rate. COPD hospital admissions (ICD-9 codes: 490-492, 494-496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.378 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves and Gillum, 1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 2.88 percent increase in admissions due to a PM_{10} change of 10.0 $\mu g/m^3$ (Samet et al., 2000, Part II - Table 14)[11]. This translates to a relative risk of 1.029. The coefficient is calculated as follows:

$$b = \frac{\ln(1.029)}{10.0} = 0.00288.$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the percent increase (Samet et al., 2000, Part II - Table 14):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{0.0564}{10} - \frac{0.0288}{10}\right)}{1.96} = 0.00141$$

[10] ICD-9 codes 490-492 and 494-496.

[11] 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).

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{0.0288}{10} - \frac{0.0019}{10}\right)}{1.96} = 0.00137$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00139.$$

Hospital Admissions for Pneumonia (Samet et al., 2000, 14 Cities)

The C-R function to estimate the change in hospital admissions for pneumonia[12] associated with daily changes in PM_{10} is:

$$\Delta pneumonia\ admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0	= daily hospital admission rate for pneumonia per person 65 and older = 5.30 E-5
β	= PM_{10} coefficient = 0.00207
ΔPM_{10}	= change in daily average PM_{10} concentration
pop	= population age 65 and older
σ_β	= standard error of β = 0.00058

Incidence Rate. Congestive heart failure hospital admissions (ICD-9 codes: 480-487) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.642 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves and Gillum, 1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 2.07 percent increase in admissions due to a PM_{10} change of 10.0 $\mu g/m^3$ (Samet et al., 2000, Part II - Table 14)[13]. This translates to a relative risk of 1.021. The coefficient is calculated as follows:

$$b = \frac{\ln(1.021)}{10.0} = 0.00207.$$

[12] ICD-9 codes 480-487.

[13] 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).

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the percent increase (Samet et al., 2000, Part II - Table 14):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{0.0322}{10} - \frac{0.0207}{10}\right)}{1.96} = 0.00059$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{0.0207}{10} - \frac{0.0094}{10}\right)}{1.96} = 0.00058$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00058.$$

Hospital Admissions for Asthma (Sheppard et al., 1999, Seattle)

Sheppard et al. (1999) studied the relation between air pollution in Seattle and nonelderly hospital admissions for asthma from 1987 to 1994. They used air quality data for PM_{10}, $PM_{2.5}$, coarse $PM_{2.5-10}$, SO_2, ozone, and CO in a Poisson regression model with control for time trends, seasonal variations, and temperature-related weather effects. They found asthma hospital admissions associated with PM_{10}, $PM_{2.5}$, coarse $PM_{2.5-10}$, 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 model was found using ozone. However, ozone data was only available April through October, so they did not consider ozone further. The C-R function in this analysis is based on a two-pollutant model with CO and $PM_{2.5}$.

The C-R function to estimate the change in hospital admissions for asthma associated with daily changes in $PM_{2.5}$ is:

$$\Delta Asthma\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:
y_0 = daily hospital admission rate for asthma per person = 4.52 E-6
β = $PM_{2.5}$ coefficient = 0.00227
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration
pop = population of ages less than 65

σ_β = standard error of $\beta = 0.000948$

Incidence Rate. Hospital admissions for asthma (ICD-9 code: 493) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.375 million) divided by the 1994 population (227.210 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with CO, the daily average coefficient (β) is estimated from the relative risk (1.03) associated with a change in $PM_{2.5}$ exposure over the interquartile range of 8 to 21 $\mu g/m^3$ (Sheppard et al., 1999, Table 3 and p. 28):

$$b = \frac{\ln(1.03)}{13} = 0.00227 .$$

Standard Error (σ_β). The standard error (σ_β) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Sheppard et al., 1999, p. 28):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.06)}{13} - \frac{\ln(1.03)}{13}\right)}{1.96} = 0.00113$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.03)}{13} - \frac{\ln(1.01)}{13}\right)}{1.96} = 0.000770$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000948 .$$

Emergency Room Visits

There is a wealth of epidemiological information on the relationship between air pollution and hospital admissions for various respiratory and cardiovascular diseases; in addition, some studies have examined the relationship between air pollution and ER visits. Because most ER visits do not result in an admission to the hospital -- the majority of people going to the ER are treated and return home -- we treat hospital admissions and ER visits separately, taking account of the fraction of ER visits that do get admitted to the hospital, as discussed below.

The only types of ER visit that have been explicitly linked to ozone in U.S. and Canadian epidemiological studies are asthma visits. However, it seems likely that ozone may be linked to other types of respiratory-related ER visits.

Emergency Room Visits for Asthma (Schwartz et al., 1993, Seattle)

Schwartz et al. (1993) examined the relationship between air quality and emergency room visits for asthma 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.

The C-R function to estimate the change in daily emergency room visits for asthma associated with daily changes in PM_{10} is:

$$\Delta\ Asthma\ ERvisits = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily ER visits for asthma per person under 65 years old = 7.69 E-6
β = PM_{10} coefficient (Schwartz et al., 1993, p. 829) = 0.00367
ΔPM_{10} = change in daily average PM_{10} concentration
pop = population of ages 0-64
σ_β = standard error of β (Schwartz et al., 1993, p. 829) = 0.00126

Incidence Rate. Smith et al. (1997, p. 789) reported that in 1987 there were 445,000 asthma admissions and 1.2 million asthma ER visits. Assuming that all asthma hospital admissions pass through the ER room, then 37% of ER visits end up as hospital admissions. As described below, the 1994 asthma admission rate for people less than 65 is 4.522 E-6. So one might assume, ER visits = (1/0.37)*asthma admission rate = 2.7*asthma admission rate = 1.22 E-5. Now, ER visits (subtracting out those visits that end up as admissions)= 1.7*asthma admission rate = 7.69 E-6.

Asthma hospital admissions (ICD-9 code: 493) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.375 million) divided by the 1994 population of individuals under 65 years old (227.21 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves and Gillum, 1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Acute Morbidity

In addition to chronic illnesses and hospital admissions, there is a considerable body of scientific research that has estimated significant relationships between elevated air pollution levels and other morbidity health effects. Chamber study research has established relationships between specific air pollution chemicals and symptoms such as coughing, pain on deep inspiration, wheezing, eye irritation and headaches. In addition, epidemiological research has found air pollution relationships with acute infectious diseases (e.g., bronchitis, sinusitis) and a variety of "symptom-day" categories. Some "symptom-day" studies examine excess incidences of days with identified symptoms such as wheezing,

coughing, or other specific upper or lower respiratory symptoms. Other studies estimate relationships for days with a more general description of days with adverse health impacts, such as "respiratory restricted activity days" or work loss days.

A challenge in preparing an analysis of the minor morbidity effects is identifying a set of effect estimates that reflects the full range of identified adverse health effects but avoids double counting. From the definitions of the specific health effects examined in each research project, it is possible to identify a set of effects that are non-overlapping, and can be ultimately treated as additive in a benefits analysis.

Acute Bronchitis C-R Function (Dockery et al., 1996)

Dockery et al. (1996) examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a study of 13,369 children ages 8-12 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.[14] They also found nitrates were linked to asthma, and sulfates linked to chronic phlegm. It is important to note that the study examined annual pollution exposures, and the authors did not rule out that acute (daily) exposures could be related to asthma attacks and other acute episodes.

Earlier work, by Dockery et al. (1989), 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) study is the better study to develop a C-R function linking $PM_{2.5}$ with bronchitis. The C-R function to estimate the change in acute bronchitis is:

$$\Delta Acute\ Bronchitis = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{2.5}\cdot b} + y_0} - y_0\right]\cdot pop\,,$$

where:

y_0 = annual bronchitis incidence rate per person = 0.044
β = estimated $PM_{2.5}$ logistic regression coefficient = 0.0272
$\Delta PM_{2.5}$ = change in annual average $PM_{2.5}$ concentration
pop = population of ages 8-12
σ_β = standard error of β = 0.0171

Incidence Rate. Bronchitis was counted in the study only if there were "reports of symptoms in the past 12 months" (Dockery et al., 1996, p. 501). It is unclear, however, if the cases of bronchitis are

[14] 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 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.

In 1994, 2,115,000 children ages 5-17 experienced acute conditions (Adams and Marano, 1995, Table 6) out of population of 48.110 million children ages 5-17 (U.S. Bureau of the Census, 1998, Table 14), or 4.4 percent of this population. This figure is somewhat lower than the 5.34 percent of children under the age of 18 reported to have chronic bronchitis in 1990-1992 (Collins, 1997, Table 8). Dockery et al. (1996, p. 503) reported that in the 24 study cities the bronchitis rate varied from three to ten percent. Finally a weighted average of the incidence rates in the six cities in the Dockery et al. (1989) study is 6.34 percent , where the sample size from each city is used to weight the respective incidence rate (Dockery et al., 1989, Tables 1 and 4).[15] This analysis assumes a 4.4 percent prevalence rate is the most representative of the national population. Note that this measure reflects the fraction of children that have a chest ailment diagnosed as bronchitis in the past year, not the number of days that children are adversely affected by acute bronchitis.[16]

Coefficient Estimate (β). The estimated logistic coefficient (β) is based on the odds ratio (= 1.50) 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 et al., 1996, 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.

$$b_{PM_{2.5}} = \frac{\ln(1.50)}{(20.7 - 5.8)} = 0.0272 .$$

Standard Error (σ_β). The standard error of the coefficient (σ_β) is calculated from the reported lower and upper bounds of the odds ratio (Dockery et al., 1996, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(2.47)}{14.9} - \frac{\ln(1.50)}{14.9}\right)}{1.96} = 0.0171$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.50)}{14.9} - \frac{\ln(0.91)}{14.9}\right)}{1.96} = 0.0171$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.0171 .$$

[15]The unweighted average of the six city rates is 0.0647.

[16]In 1994, there were 13,707,000 restricted activity days associated with acute bronchitis, and 2,115,000 children (ages 5-17) experienced acute conditions (Adams and Marano, 1995, Tables 6 and 21). On average, then, each child with acute bronchitis suffered 6.48 days.

Lower Respiratory Symptoms (Schwartz et al., 1994)

Schwartz et al. (1994) used logistic regression to link lower respiratory symptoms in children with SO_2, NO_2, ozone, PM_{10}, $PM_{2.5}$, sulfate and H^+ (hydrogen ion). Children were selected for the study if they were exposed to indoor sources of air pollution: gas stoves and parental smoking. The study enrolled 1,844 children into a year-long study that was conducted in different years (1984 to 1988) in six cities. The students were in grades two through five at the time of enrollment in 1984. By the completion of the final study, the cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

In single pollutant models SO_2, NO_2, $PM_{2.5}$, and PM_{10} were significantly linked to cough. In two-pollutant models, PM_{10} had the most consistent relationship with cough; ozone was marginally significant, controlling for PM_{10}. In models for upper respiratory symptoms, they reported a marginally significant association for PM_{10}. In models for lower respiratory symptoms, they reported significant single-pollutant models, using SO_2, O_3, $PM_{2.5}$, PM_{10}, SO_4, and H^+.

The C-R function used to estimate the change in lower respiratory symptoms is:

$$\Delta Lower\ \mathrm{Re}spiratory\ Symptoms = -\left[\frac{y_0}{\left(1 - y_0\right)\cdot e^{\Delta PM_{2.5}\cdot b} + y_0} - y_0\right]\cdot pop\,.$$

where:

y_0 = daily lower respiratory symptom incidence rate per person = 0.0012
β = estimated $PM_{2.5}$ logistic regression coefficient = 0.01823
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration
pop = population of ages 7-14
σ_β = standard error of β = 0.00586

Incidence Rate. The proposed incidence rate, 0.12 percent, is based on the percentiles in Schwartz et al. (Schwartz et al., 1994, Table 2). They did not report the mean incidence rate, but rather reported various percentiles from the incidence rate distribution. The percentiles and associated values are 10^{th} = 0 percent, 25^{th} = 0 percent, 50^{th} = 0 percent, 75^{th} = 0.29 percent, and 90^{th} = 0.34 percent. The most conservative estimate consistent with the data are to assume the incidence is zero up to the 75^{th} percentile, a constant 0.29 percent between the 75^{th} and 90^{th} percentiles, and a constant 0.34 percent between the 90^{th} and 100^{th} percentiles. Alternatively, assuming a linear slope between the 50^{th} and 75^{th}, 75^{th} and 90^{th}, and 90^{th} to 100^{th} percentiles, the estimated mean incidence rate is 0.12 percent,[17] which is used in this analysis.

[17]For example, the 62.5^{th} percentile would have an estimated incidence rate of 0.145 percent.

Coefficient Estimate (β). The coefficient β is calculated from the reported odds ratio (= 1.44) in a single-pollutant model associated with a 20 $\mu g/m^3$ change in $PM_{2.5}$ (Schwartz et al., 1994, Table 5):

$$b = \frac{\ln(1.44)}{20} = 0.01823.$$

Standard Error (σ_β). The standard error for the coefficient (σ_β) is calculated from the reported lower and upper bounds of the odds ratio (Schwartz et al., 1994, Table 5):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.82)}{20} - \frac{\ln(1.44)}{20}\right)}{1.96} = 0.00597$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.44)}{20} - \frac{\ln(1.15)}{20}\right)}{1.96} = 0.00574$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.00586.$$

Population. Schwartz et al. (1994, Table 5 and p. 1235) enrolled 1,844 children into a year-long study that was conducted in different years in different cities; the students were in grades two through five and lived in six U.S. cities. All study participants were enrolled in September 1984; the actual study was conducted in Watertown, MA in 1984/85; Kingston-Harriman, TN, and St. Louis, MO in 1985/86; Steubenville, OH, and Portage, WI in 1986/87; and Topeka, KS in 1987/88. The study does not publish the age range of the children when they participated. As a result, the study is somewhat unclear about the appropriate age range for the resulting C-R function. If all the children were in second grade in 1984 (ages 7-8) then the Topeka cohort would be in fifth grade (ages 10-11) when they participated in the study. It appears from the published description, however, that the students were in grades two through five in 1984.[18] By the completion of the study, some students in the Topeka cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

Upper Respiratory Symptoms (Pope et al., 1991)

Using logistic regression, Pope et al. (1991) 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

[18]Neas et al. (1994, p. 1091) used the same data set; their description suggests that grades two to five were represented initially.

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 et al., 1991, p. 669)." The patient-based subjects (ranging in age from 8 to 72) were receiving treatment for asthma and were referred by local physicians. Regression results for the school-based sample (Pope et al., 1991, Table 5) show 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.

The C-R function used to estimate the change in upper respiratory symptoms is:

$$\Delta Upper\ \mathrm{Re}spiratory\ Symptoms = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot b} + y_0} - y_0\right]\cdot pop,$$

where:

y_0 = daily upper respiratory symptom incidence rate per person = 0.3419
β = estimated PM_{10} logistic regression coefficient (Pope et al., 1991, Table 5) = 0.0036
ΔPM_{10} = change in daily average PM_{10} concentration
pop = asthmatic population[19] ages 9 to 11 = 6.91% of population ages 9 to 11
σ_β = standard error of β (Pope et al., 1991, Table 5) = 0.0015

Incidence Rate. The incidence rate is published in Pope et al. (Pope et al., 1991, Table 2). Taking a sample-size-weighted average, one gets an incidence rate of 0.3419.

Minor Restricted Activity Days (Ostro and Rothschild, 1989)

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for $PM_{2.5}$, two-week average O_3 has highly variable association with RRADs and MRADs. Controlling for O_3, two-week average $PM_{2.5}$ was significantly linked to both health endpoints in most years.

---

[19]Adams (1995, Table 57) reported that in 1994, 6.91% of individuals under the age of 18 have asthma.

The study is based on a "convenience" sample of individuals ages 18-65. 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 65 and younger. The elderly appear more likely to die due to PM exposure than other age groups (e.g., Schwartz, 1994c, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994a; Schwartz, 1994b).

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 used in this analysis is a weighted average of the coefficients (Ostro, 1987, Table IV) using the inverse of the variance as the weight. The C-R function to estimate the change in the number of minor restricted activity days (MRAD) is:

$$\Delta MRAD = \Delta y \cdot pop = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = daily MRAD daily incidence rate per person = 0.02137
β = inverse-variance weighted $PM_{2.5}$ coeffcient = 0.00741
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration[20]
pop = adult population ages 18 to 65
σ_β = standard error of β = 0.0007

Incidence Rate. The annual incidence rate (7.8) provided by Ostro and Rothschild (1989, p. 243) was divided by 365 to get a daily rate of 0.02137.

Coefficient Estimate (β). The coefficient is a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4) using the inverse of the variance as the weight:

$$b = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = 0.00741.$$

[20]The study used a two-week average pollution concentration; the daily rate used here is assumed to be a reasonable approximation.

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

$$s_b^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}}\right) = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{g}\right) = \sum_{i=1976}^{1981} \text{var}\left(\frac{b_i}{s_{b_i}^2 \cdot g}\right).$$

This reduces down to:

$$s_b^2 = \frac{1}{g} \Rightarrow s_b = \sqrt{\frac{1}{g}} = 0.00070.$$

Work Loss Days (Ostro, 1987)

Ostro (1987) estimated the impact of $PM_{2.5}$ on the incidence of work-loss days (WLDs), restricted activity days (RADs), and respiratory-related RADs (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average $PM_{2.5}$ levels were significantly linked to work-loss days, RADs, and RRADs, however there was some year-to-year variability in the results. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function used here is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight.

The study is based on a "convenience" sample of individuals ages 18-65. 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 65 and younger. The elderly appear more likely to die due to PM exposure than other age groups (e.g., Schwartz, 1994c, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994a; Schwartz, 1994b). On the other hand, the number of workers over the age of 65 is relatively small; it was under 3% of the total workforce in 1996 (U.S. Bureau of the Census, 1997, Table 633).

The C-R function to estimate the change in the number of work-loss days is:

$$\Delta WLD = \Delta y \cdot pop = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = daily work-loss-day incidence rate per person = 0.00648
β = inverse-variance weighted $PM_{2.5}$ coefficient = 0.0046

$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration[21]
pop = population of ages 18 to 65
σ_β = standard error of $\beta = 0.00036$

Incidence Rate. The estimated 1994 annual incidence rate is the annual number (376,844,000) of WLD per person in the age 18-64 population divided by the number of people in 18-64 population (159,361,000). The 1994 daily incidence rate is calculated as the annual rate divided by 365.[22] Data are from U.S. Bureau of the Census (1997, Table 14) and Adams (1995, Table 41).

Coefficient Estimate (β). The coefficient used in the C-R function is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight:

$$b = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = 0.0046 .$$

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

$$s_b^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{g} \right) = \sum_{i=1976}^{1981} \text{var}\left(\frac{b_i}{s_{b_i}^2 \cdot g} \right) .$$

This eventually reduces down to:

$$s_b^2 = \frac{1}{g} \Rightarrow s_b = \sqrt{\frac{1}{g}} = 0.00036 .$$

Asthma Attacks: Whittemore and Korn (1980)

Whittemore and Korn (1980) 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

---

[21]The study used a two-week average pollution concentration; the daily rate used here is assumed to be a reasonable approximation.

[22]Ostro (1987) analyzed a sample aged 18 to 65. It is assumed that the age 18-64 rate is a reasonably good approximation to the rate for individuals 18-65. Data are from U.S. Bureau of the Census (1997, Table 14) and Adams (1995, Table 41).

34-week periods in 1972-1975. The analysis focused on TSP and ozone. 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 O_x were significantly related to reported asthma attacks.

The C-R function to estimate the change in the number of asthma attacks is:

$$\Delta \, asthma \, attacks = -\left[\frac{y_0}{(1 - y_0) \cdot e^{\Delta PM_{10} \cdot b} + y_0} - y_0 \right] \cdot pop,$$

where:

y_0 = daily incidence of asthma attacks = 0.027 (Krupnick, 1988, p. 4-6)

β = PM_{10} coefficient = 0.00144

ΔPM_{10} = change in daily PM_{10} concentration

pop = population of asthmatics of all ages = 5.61% of the population of all ages (Adams and Marano, 1995 Table 57).

σ_β = standard error of β = 0.000556

Incidence Rate. The annual rate of 9.9 asthma attacks per asthmatic is divided by 365 to get a daily rate. A figure of 9.9 is roughly consistent with the recent statement that "People with asthma have more than 100 million days of restricted activity" each year (National Heart, 1997, p. 1). This 100 million incidence figure coupled with the 1996 population of 265,557,000 (U.S. Bureau of the Census, 1997, Table 2) and the latest asthmatic prevalence rate of 5.61% (Adams and Marano, 1995, Table 57), suggest an annual asthma attach rate per asthmatic of 6.7.

Coefficient Estimate (β). Based on a model with ozone, the coefficient is based on a TSP coefficient (0.00079) (Whittemore and Korn, 1980, Table 5). Assuming that PM_{10} is 55 percent of TSP[23] and that particulates greater than ten micrometers are harmless, the coefficient is calculated as follows:

$$b = \frac{0.00079}{0.55} = 0.00144.$$

Standard Error (σ_β). The standard error (σ_β) is calculated from the two-tailed p-value (<0.01) reported by Whittemore and Korn (1980, Table 5), which implies a t-value of at least 2.576 (assuming a large number of degrees of freedom).

$$s_b = \frac{b}{t} = \frac{0.144}{2.576} = 0.000556.$$

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